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Perspective: Replication does not reliably measure scientific productivity

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Abstract

Replication surveys are becoming a standard tool for assessing the knowledge production of scientific disciplines. In psychology, economics, and preclinical cancer biology, replication rates near 50% have been advanced as evidence that these disciplines have failed to reliably produce knowledge, are rife with questionable research practices, and warrant reform. Concerns over failed replications are sometimes leveraged to erode faith in science, even claiming that the majority of published research is false. Even when quantitatively grounded, the assumptions underlying such claims are highly restrictive; for example, the effect sizes are fixed across empirical contexts, and null hypotheses of exactly zero effect are assumed to have a high probability of being true. Here we derive a theoretical model of the publication process that relaxes these assumptions. Accounting for variation in observed effect sizes across empirical settings and acknowledging that most treatments have some effect—even if small and idiosyncratic—we find that aggregate measures of replication rates provide little insight into whether a scientific discipline is productive. Applying our model to data from large-scale replication surveys, suggest that concerns over the reliability of scientific research may be overstated. We highlight how proposed reforms may be ineffective at improving replicability and worse yet, detrimental to broader measures of scientific productivity.

Keywords: Metascience, Replication, Reproducibility, Statistics, Psychology.

Surveys across multiple disciplines have demonstrated that a large portion of statistically significant findings fail to achieve significance upon replication [1, 2, 3]. Such findings are often taken as evidence that something is rotten in the state of science: either the vast majority of attempted research generates negative results that go unpublished, or researchers often engage in questionable research practices (QRPs) such as outcome switching, harking, p-hacking, or uncorrected multiple comparisons to achieve significant results [4, 5]. Some researchers have argued that many or even most published scientific findings are false positives [6].

The past decade's focus on replication rates has propelled a much-needed conversation around questionable research practices and how to avoid them. It has also spurred an ongoing discussion about best practices with respect to assessing and reporting statistical significance. Moreover, it has profoundly impacted the process and perception of science, not always for the better. In attempting to correct the record, substantial resources have been routed towards conducting replications in lieu of pursuing novel research agendas. Failed replications of established findings can make a splash, reducing the public's faith in those results in particular and in science more generally [7]. Within academia, low rates of replication have been argued to indicate that some disciplines have "failed beyond repair", jeopardizing future funding and research [8]. Individual authors of research that fails to replicate can face personal or professional consequences ranging from disparagement to harassment to irreparable career damage [9, 10]. Promising evidence-based interventions may be shelved or delayed following a failed replication.

How can we reconcile a preponderance of statistically significant findings in the published literature with the low rates of success reported in replication studies? One possible explanation is that the vast majority of attempted research goes unpublished because the findings are non-significant. This is the so-called file drawer effect [11]. The magnitude of the file drawer effect depends on the nature of the hypotheses that researchers choose to test—the less likely they are to be correct *a priori*, the larger the file drawer. In psychology, replication rates have lead researchers to infer that researchers are testing hypotheses that are unlikely em a priori—with prior probabilities as low as 10% [4, 12]. We find this explanation implausible. On average, newly-hired assistant professor in psychology has 16 publications, many or all of which contain multiple experiments or hypothesis tests [13]. Yet negative results compose only a small fraction of the published literature across disciplines and in social psychology in particular [14]. If only one experiment in ten proved successful, amassing this quantity of positive results would require an impractical expenditure of effort and resources in a very short period of time, and an exceptionally large file drawer. Furthermore, evidence from registered reports is inconsistent with the 10% prior probability scenario: approximately 40% of registered reports achieve significance—suggesting a file-drawer size on the order of 1 to 1.5 times the size of the published scientific record[15, 16].

An alternative explanation is that false positives arise from researchers intentionally or inadvertently adopting QRPs that lead to inappropriate rejection of the null hypothesis [17]. A QRP-based interpretation of the replication crisis aligns with the strong incentives to compile a competitive CV. However, in such a world, honest researchers with basic statistical training would be a rarity, filtered out by a job market where paper tallies matter.

Whatever the explanation for failed replications, the past decade has seen a movement toward scientific reforms seek to improve transparency and publish null results, reducing incentives to engage in QRPs and thereby improve replication [18]. These range from preregistration and registered reports, to improving theory prior to experimentation or strengthening thresholds for significance [12]. While many of these proposed reforms have the potential to convey considerable benefits, they are unlikely to come without costs—particularly if imposed indiscriminately. Preregistration, for example, may incentivize researchers to stick with previously-specified models, regardless of whether or not more appropriate models become clear once the data are acquired. Registered reports may limit exploratory research and discourage novel or high-risk approaches [19]. Overall, these reforms risk redefining quality science in a manner that prioritizes some forms of quantitative inquiry over others.

The core concern in the so-called replication crisis is that low replication rates in a field indicate that the field is likely to be publishing a large number of incorrect findings. But such arguments tend to rely on two assumptions: (1) that effect sizes of interest are fixed across contexts and (2) that point-null hypotheses (e.g., that the actual effect of a manipulation is exactly zero) have a meaningful probability of being true [6]. Critically, this implies that effects vary solely due to measurement error and are not mediated or biased by context or statistical modeling decisions. Such arguments simply ignore the well-established fact that effects vary across experimental contexts beyond what would be expected by measurement error alone [20, 21, 22, 23, 24, 25]. Similarly, the notion that true effect sizes can be precisely zero is not grounded in reality. Rather, it is a mathematical convenience that facilitates the calculation of sampling distributions—a relic of a pre-digital era. Given the centrality of replication to our appraisal of scientific progress and reform, we would do well to consider the data around replication in light of the fact that these two assumptions are often unrealistic.

Results

Motivation

The assumptions we have just described have been inherited from the null hypothesis significance testing framework. Together, they have had an instrumental role in launching and framing conceptions of scientific productivity and reform. Interest in the replication crisis has largely centered around a formal model designed to estimate the probability a result is true, conditioned on significance [6, 26]. According to this approach, p-values inherently evaluate the plausibility of the data given some null model, M, often with an effect size d equal to precisely zero (e.g., $Pr(\text{data} \mid M, d = 0)$). Informally though, scientists rely on (or misinterpret) p-values as evidence that the null model can be rejected in favor of the alternate hypothesis H_a that $d \neq 0$. These are not the same, as the probability of significance (+) given the null hypothesis H_0 is not the probability of the null hypothesis, given significance, i.e, $P(H_0 \mid +) \neq P(+ \mid H_0)$. Bayes' rule makes it possible to estimate the probability that an alternate hypothesis is True, given significance was observed [26, 6]:

$$Pr(H_a \mid +) = \frac{Pr(+|H_a)Pr(H_a)}{Pr(+|H_a)Pr(H_a) + Pr(H_0)Pr(+|H_0)} \tag{1}$$

Here, $Pr(+|H_a)$ is the power of an experiment, and $Pr(+|H_0)$ is the threshold for significance, typically 0.05. Moreover, this calculation requires an additional piece of information: the prior probability of the hypothesis being True, $Pr(H_a)$. Intuitively, a highly improbably hypothesis is likely be a false positive even when significance is achieved. Using this model, an adequately powered study with 80% power that is significant at $p < .05$ with a 10% *a priori* chance of being True would nonetheless have a 36% chance of being a False claim. Similar calculations can be used to evaluate evidence subsequent to a replication effort, using the posterior probability from the first study as the prior probability for the replication. A successful high-powered (95%) replication at $p < .001$ for the study described above would yield a 99.99% chance the study is True, while a failed replication would render an 2.7% chance. This is the ostensible power of replications—the ability to forge an uncertain finding

into reliable knowledge. On the strength of this claim we've been sold the obverse, a very different claim: that failures to replicate a study suggest that its findings are false. From there, the argument goes, the fact that a large portion of attempted replications fail must mean that much of the published literature is false. Under this model, replication truly makes sense as a cornerstone of scientific inquiry, and we are in crisis.

Further, this framing implies that arbitrary study with a similar prior probability of being true, power, and significance threshold would only observe significance $\approx 12\%$ of the time. The overabundance of significant findings in the literature—coupled with the rates at which successful scholars are able to publish—has thus been used to conclude that QRPs are widespread, fraud is not infrequent, and wasted effort abounds as negative results accumulate in file drawers. Under this model, the "crisis" has a clear cause leading to obvious remedies. This model and verbal or mathematical extensions have guided scientific reform, from calls to redefine statistical significance to the need for registered reports and increased transparency [12, 17, 4].

Yet, this model is based on a particularly common and fraught assumption. In almost any context we would investigate in practice the null hypothesis that $d = 0$ has nearly zero probability of being precisely true. Even if there is no true causal relationship, the context we measure an effect or the analyses we perform will mediate the observed effect—if ever so slightly—away from zero [24]. These mediators may be consistent and therefore identifiable, or ephemeral and hard to pin down. As Andrew Gelman has noted, under the strictest interpretation, all findings rejecting a point null hypothesis with a two-tailed test are correct, if not usefully so [27].

If we cannot discretize effects into true ($d \neq 0$) and false ($d = 0$), we may instead consider them as continuous quantities. This view is motivated by the notion that scientists are often interested in effects that are common—though by no means identical—across contexts or a population [27, 28]. A given quantitative investigation into an effect can be viewed as sampling from a distribution of hypothetical replications spanning some broader population and range of contexts. On one extreme, these imagined replications may be conceptual, considering diverse implementations and contexts such that observed effects vary widely. On the other, these replications may be thought of as "close replications", explicitly designed to minimize variation, as in a "many labs" context [22, 23]. Under this framework, science could be considered to be producing knowledge if published effects reliably convey information about the broader effect of interest [27]. For example, one could ask whether a significant effect chosen arbitrarily from the published literature will be consistent in direction and magnitude with the average of the imagined replications. This view of science echoes the notion that results should be consistent across contexts, yet replaces a restrictive binary truth with continuous calibration.

This calibration-minded approach is quite natural for Bayesian statisticians, yet the bulk of research produced relies on null hypothesis significance testing. This raises a question of whether published effect sizes—heavily selected for significance—can nonetheless be calibrated to broader effects of interest. Under what conditions does this occur? Moreover, does the rate at which papers replicate in a binary sense provide a reasonable metric regarding whether a discipline or area of study is reliably producing knowledge? Do replications truly distinguish fact from fiction? More generally, does relaxing assumptions of binary Truth provide a qualitatively different perspective on the "replication crisis" and proposed scientific reform?

To address these questions, we derive a model of publication and replication that incorporates variation in effect sizes across contexts. Our model builds on, combines, and extends several previous models or schools of thought on varying (or heterogenous) effects and effect-size calibration [28, 27, 20, 29]. We leverage this model to examine the impact of varying effects on publication and replication rates. We examine whether low rates of replication provide information about the tendency for published results to reflect the true direction and magnitude of average underlying effects. We use Bayesian methods to apply our model to data from replication surveys. Finally, we simulate a body of published literature to examine the validity of common concerns over low rates of replication and the likely consequences of proposed interventions.

Theory

For simplicity, our model (Fig. 1) assumes that researchers conduct one-sample t-tests on idealized data and evaluate significance at $\alpha = .05$. Their hypotheses correspond to average effect sizes, d, that are normally distributed about zero, and with a characteristic scale of variation, τ. Hypothesis tests that are statistically significant are published; those that fail to achieve significance are not.

$$d \sim \text{Normal}(0, \tau) \tag{2}$$

The average effect size studied in a given field (i.e. Cohen's d, $E[|d|]$)), will be equal to $\tau \times \sqrt{2/\pi}$. However, when a given effect is measured in practice, features unique to that context may mediate the average effect by adding additional mediator variance σ such that for a given hypothesis j with replication-averaged effect size d_j, study-specific effect sizes, d'_j will be distributed such that:

$$d'_j \sim \text{Normal}(d_j, \sigma) \tag{3}$$

while across hypotheses and contexts:

$$d' \sim \text{Normal}(0, \sqrt{\tau^2 + \sigma^2}) \tag{4}$$

Here σ captures the magnitude of bias in an observed effect size resulting from mediators specific to a given empirical context. This can occur for numerous reasons, ranging from a poorly chosen statistical model to imperfect randomization, differing sample populations, environmental conditions, or flexibility in experimental design. Even well designed and documented procedures in highly controlled contexts can vary in implementation such that $\sigma > 0$. Notably, this is distinct from unbiased measurement error, ϵ, because it is independent across experiments rather than individuals and therefore cannot be reduced by increasing sample size within any individual experiment.

In addition to context-mediated effects, incorporating measurement error into our model gives us the observed effect size for an individual measurement, d_{obs}.

$$d_{obs} \sim \text{Normal}(0, \sqrt{\tau^2 + \sigma^2 + \epsilon^2}) \tag{5}$$

Since the values of τ and σ are shared across all observations within a single experiment, the mean effect size observed in an experiment is reduced by a factor of $\sqrt{n}$ only in the component of variance that is independent between observations, ϵ:

$$\bar{d}_{obs} \sim \text{Normal}(0, \sqrt{\tau^2 + \sigma^2 + \epsilon^2/\sqrt{n}}) \tag{6}$$

Note that even if the hypothesized effect size is truly zero, then as the sample size $n \to \infty$ the expected magnitude of the observed effect size will be $|\bar{d}_{obs}| = \sigma \times \sqrt{2/\pi}$.

From here, we can use a power analysis to estimate the probability that an arbitrary novel hypothesis, examined in an experiment with sample size n, achieves statistical significance at some threshold α. Where $\Phi(\cdot)$ is the standard normal cumulative distribution function and t_c is the critical value of the test statistic for statistical significance at a given α, this probability is given by

$$Pr(p < \alpha) = 2 \times \Phi\left(-\frac{(\epsilon/\sqrt{n})t_c}{\sqrt{\epsilon^2/n + \tau^2 + \sigma^2}}\right). \tag{7}$$

For simplicity throughout, we standardize effect sizes relative to measurement error such that $\epsilon = 1$. We assume that experimental observations are genuinely normally-distributed (as per the model above), but we do not necessarily assume that the statistical analyst makes this assumption (i.e. t_c may depend on n as the critical threshold in a t-test). We note this aspect of the model is an extension of common techniques for estimating statistical power for varying effects [28, 30**?**].

Applying our model to a fixed sample size of $n = 100$, we find that the majority of attempted hypotheses will obtain significance provided study-specific effect sizes ($\sqrt{\tau^2 + \sigma^2}$) are sufficiently large (Fig. 2A). This implies that high rates of publication across a field by themselves can be consistent either with typically large hypothesized effect sizes (τ), the presence of large mediation effects (σ), or some combination of the two.

We can further use our model to estimate the probability that a measured effect will replicate in the same direction. It is useful here to define ρ as the proportion of variance due to the hypothesized effect size, which also defines the correlation between the outcomes of two experiments of the same sample size, exposed to differing mediation effects:

$$\rho = \tau^2/\sqrt{\epsilon^2/n + \tau^2 + \sigma^2} \tag{8}$$

Using this definition, we can express the replication probability as the probability that a second experiment will record an observed effect size $\bar{d}_{rep} > t_c$, conditioned on the first doing so:

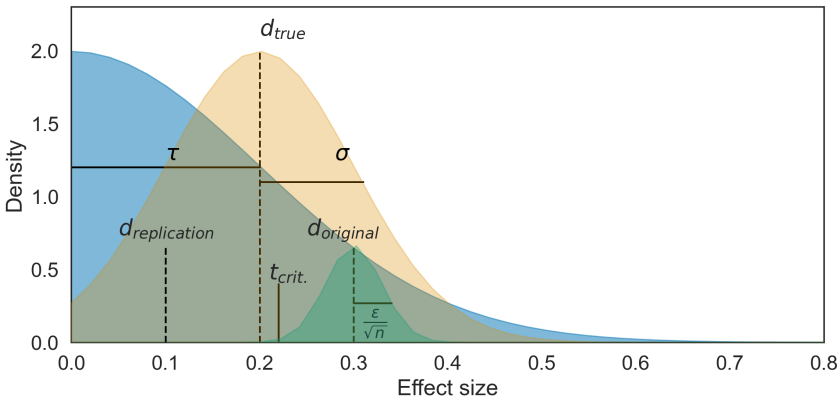

Figure 1: Overview of the theoretical model. Within a field, researchers propose hypotheses with average effect sizes characterized by a normal distribution with standard deviation τ. For a given hypothesis, the hypothesized effect size, d_j is *mediated* across contexts such that the observed effect sizes vary from one experiment to the next. The mediated effect size is represented by a normal distribution with mean d_j and variance σ. The variance due to mediation, σ, differs from measurement error ϵ, in that it is insensitive to sample size, n. Research will observe and publish a significant effect size provided that: $d_{orig} > t_c$.

$$Pr(\text{rep}) = Pr(\bar{d}_{rep} > t_c \mid \bar{d}_{orig} > t_c) \tag{9}$$

$$= \frac{1}{\Phi(-t_c)} \int_{t_c}^{\infty} \phi(x)\Phi\left(\frac{\rho x - t_c}{\sqrt{1-\rho^2}}\right) dx \tag{10}$$

where $\phi(\cdot)$ is the standard normal probability density function.

Visualising this expression shows that replication rates are fundamentally constrained by the extent to which effects vary (σ), the distribution of hypothesized effects (τ) and the sample size (n). Publication rates will exceed what is expected were a strict null hypothesis to be plausibly true, and will increase rapidly with N, σ, and τ. (Fig. 2A). However, these published effects will only reliably replicate if $\sigma << \tau$ (Fig. 2B). That is, replication rates will be low unless the typical scale of hypothesized effect sizes is sufficiently large to overwhelm the variation caused by mediator effects. This uncoupling of replication from publication is a qualitative difference between varying and fixed effects models [28].

Under-powered studies are often cited as a reason for low-rates of replication, suggesting that we can increase rates of replication by increasing sample sizes [31]. However, our analysis suggests that increasing sample sizes cannot universally improve low replication rates. Figure 2C–D demonstrates this by exploring the impact of sample size n and variable effect size σ for a fixed value of $\tau = 0.2$. Specifically, we find that large samples improve publication rates yet only meaningfully increase replication when $\sigma < \tau$ (Fig. 2C–D). Above that threshold, replication rates of $\approx 50\%$ will be observed even for arbitrarily large sample sizes. The implications are striking: a field with access to large datasets spanning a wide range of contexts will appear quite productive in terms of obtaining significant results—but replication rates may remain low and thus the field will be inefficient at producing transferable knowledge. As with publication rates, a varying-effects reduces the coupling between sample size and replication.

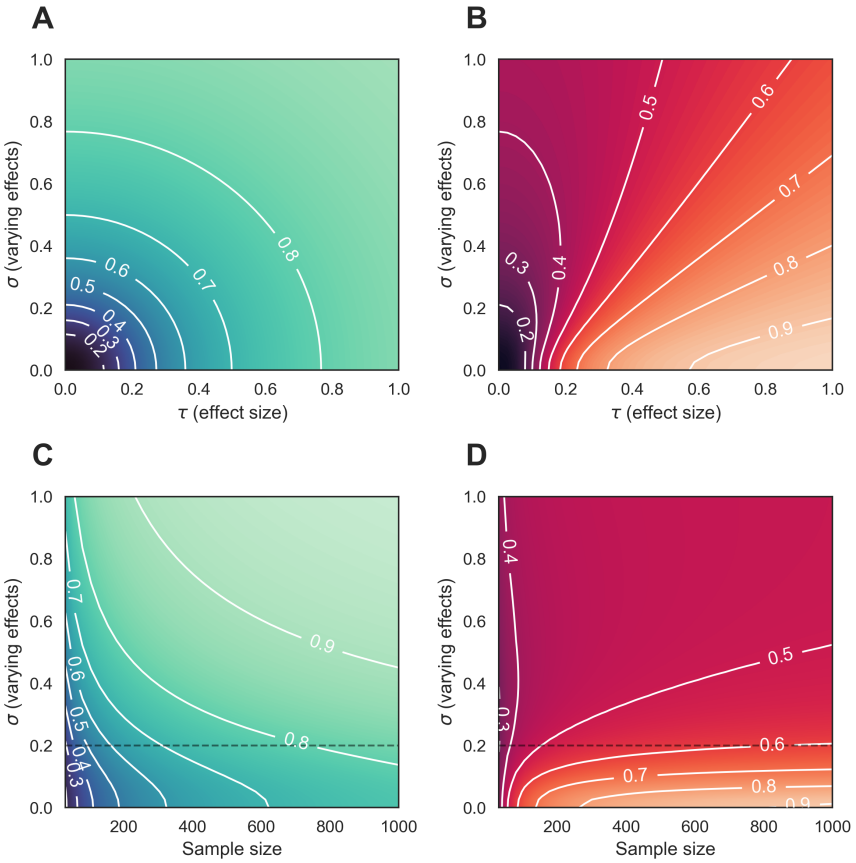

Figure 2: A) Contour lines indicate publication rates with sample size $n = 100$. The probability of publication increases with both the magnitude of hypothesized effects, τ and their variability across contexts, σ. B) Contour lines indicate probability of successful replication, again with $n = 100$. This probability increases with increasing effect size τ and usually but not always decreases with increasing varying effects σ. C) Publication probability for fixed $\tau = .2$ increases with sample size (horizontal axis) and varying effect size (vertical axis) D) Replication probability for fixed $\tau = .2$ increases with of sample size and usually decreases with varying effect size.

Replication is often presented as a binary affair: either a study replicates, or it doesn't. This obscures important complexities in the nature of failed replications and erroneous findings. Sometimes a replication will simply fail to yield a significant result. Other times, a replication will actually find a significant result in the opposite direction, suggesting that even the direction of the effect may have been wrongly identified. We call this a Type-S error, and such errors are of particular concern as effects can be qualitatively challenging to reconcile with existing research and may lead to incorrect decisions in applied contexts. We can extend our model to examine the proportion of significant effects that indicate the incorrect direction relative to their replication-averaged effects:

$$
\begin{aligned}
Pr(\text{Type-S}) &= Pr(d' > 0 \mid \bar{d}_{obs} < -t_c) && (11)\\
&= \frac{Pr(d' > 0) \times Pr(\bar{d}_{obs} < -t_c \mid d' > 0)}{Pr(d_{obs} < -t_c)} && (12)\\
&= \frac{\Phi(0)}{\Phi(\frac{-t_c}{\sqrt{\tau^2+\sigma^2+\epsilon^2/n}})} \\
&\times \int_0^\infty 2 \times \Phi(\frac{-t_c - x}{\sqrt{\sigma^2+\epsilon^2/n}})\phi(\frac{x}{\tau})dx && (13)
\end{aligned}
$$

Here, ϕ is the standard normal probability density function and Φ is the standard normal cumulative density function. The expression above gives the probability that the observed effect size is below $-t_c$, for all hypothesized effect sizes $d > 0$. This model is derived from earlier work on Type-S error, but explicitly incorporating the sample size and associated error [32]. Evaluating this expression over a range of values of τ and σ reveals that most research will indicate the correct direction of an effect, even in contexts where replication rates are low (Fig. 3A). This results from the presence of a signal (even if small) favoring outcomes in the direction of the underlying effect. This theoretical finding is consistent with generally low rates of significant reversals in replication surveys [33, 2, 3] We further evaluate Type-S error as a function of sample size and variation in effect sizes for fixed $\tau = .2$. Across sample sizes, Type-S error increases with σ. However, for sample sizes below $n \approx 200$, this effect is less pronounced as low power requires that mediator effects and signal are aligned in direction to achieve significance (Fig. 3C). For small sample sizes and small effects, artificially increasing σ could paradoxically improve detection of weak effects through a phenomenon akin to stochastic resonance S1. This could occur by intentionally adding noise to effect sizes or through some actions typically associated with QRPs, provided they're direction-agnostic.

Beyond Type-S error, published effects may be exaggerated in magnitude from the underlying effect. This is commonly referred to as either Type-M error or the exaggeration ratio: the ratio of the reported effect to the replication-averaged effect [27]. According to our model, Type-M errors arise because experiments where the hypothesized effect size is small are more likely to nonetheless return significant results and thus be published when the mediator effects or sample variance are large and in the same direction, producing spuriously strong observed effect sizes. We can estimate type-M error in published (here, significant) studies by calculating the expected value of published effects and dividing by the average effect size: $\tau\sqrt{2/\pi}$.

$$
\begin{aligned}
\text{Type-M} &= E(|\bar{d_{\text{obs}}}| \mid |\bar{d_{\text{obs}}}| > t_c)/E(|d|) \\
&= \frac{\sqrt{\tau^2+\sigma^2+\epsilon^2/n}}{\tau\sqrt{2/\pi}} \frac{\phi(t_c/\sqrt{\tau^2+\sigma^2+\epsilon^2/n})}{1-\Phi(t_c/\sqrt{\tau^2+\sigma^2+\epsilon^2/n})} && (14)
\end{aligned}
$$

We find that type-M error among significant findings will be low, provided τ is sufficiently large. When τ is small, context-specific mediation in the same direction as the underlying effect is necessary to achieve significance, artificially inflating observed effects. However, when τ is large, observed effects can achieve significance regardless of the mediation specific to a given context. Across sample sizes with fixed τ, type-M error increases with σ. In contrast

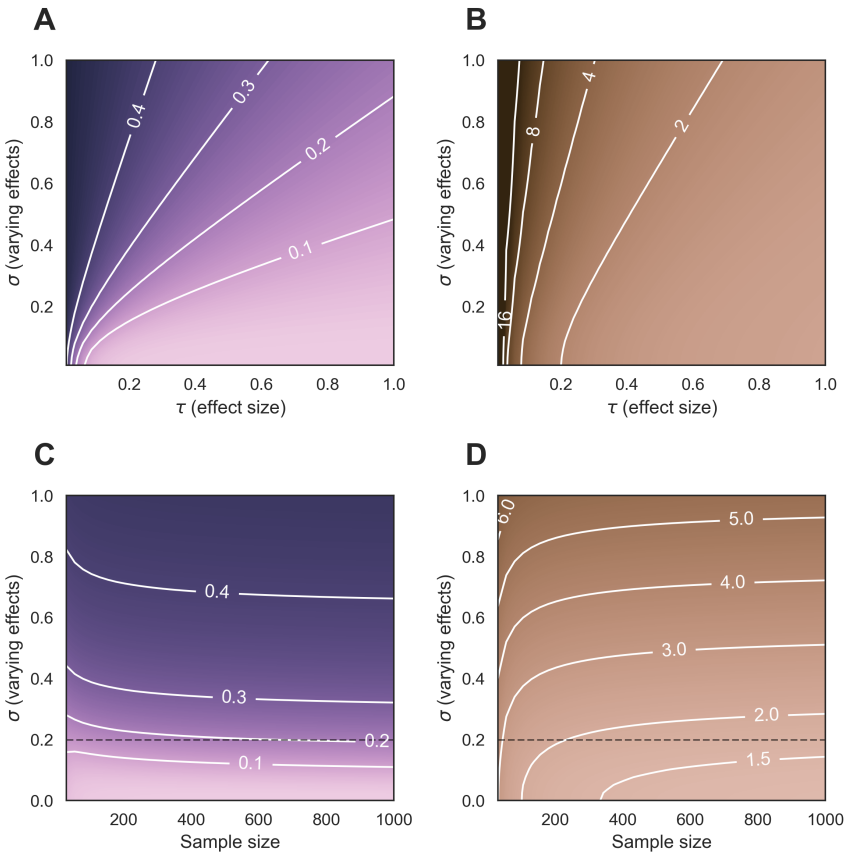

Figure 3: A) Contour lines indicate the probability that a published study determines the wrong direction for an effect (Type-S error). This probability decreases with the magnitude of hypothesized effects τ and increases with the variability across contexts σ. B) As in A, depicting the proportion of studies that replicate C) Type-S error for fixed $\tau = .2$ decreases slightly with sample size (horizontal axis) and increases strongly with the magnitude of varying effects (vertical axis). D) Type-M error for fixed $\tau = .2$ decreases with sample size (vertical axis) and increases with the magnitude of varying effects (vertical axis)..

to type-S error, type-M error is more pronounced at small sample sizes. This arises, however, from the same mechanism—directional alignment between mediation and the hypothesized effect.

Finally, we note that our theoretical approach is extensible to contexts where a proportion of hypotheses have near-zero effect sizes while others tend to have non-zero effects. This may occur, for instance, in testing of potential pharmaceuticals, where some are biologically inert and others exhibit biological activity. We can estimate this by considering the published literature as a mixture of two classes of papers, those with $\tau_1 \gtrsim 0$ and $\tau_2 > 0$. Doing so with 90% of studies evaluating arbitrarily small true effects nonetheless yields plausible file drawer sizes owing to the influence of σ. However, an overabundance of "true" null hypotheses in the literature tends to reduce rates of replication. Under these conditions Type-S error is large as σ drives significance contrary to the directions of the trivially small effects. Type-M error is difficult to interpret in this context as true effects near zero render type-M error arbitrarily large (Fig S2, S3).

This example highlights how it may be necessary to adapt the theory outlined above to

contexts where there are mechanistic reasons to believe the hypothesized effects (or their variation across contexts) are not normally distributed or differ from our model in qualitative ways. For example, research into life-saving treatments may have more capacity for effects in the direction of prolonging life than shortening it. Any specific set of distributional assumptions will necessarily alter the relationship between replication and type-S and type-M error. Taking this into account, replication as a metric is unlikely to be reliably coupled to publication, sample sizes, or type-M/S error. For this reason, comparison of replication rates across disciplines is likely to be particularly fraught.

Statistical and computational model

Our analytical results reveal that a given rate of replication is consistent with wide range of typical effect sizes, variation in observed effects, and sample sizes relative to measurement error. Moreover, replication does not reliably correspond to type-S and type-M error. Replication rates near 50% (for instance) could arise from either a field with large sample sizes and replication-averaged effects that are small relative to variable effects, or inadequate sample sizes and replication-averaged effects that are larger relative to variable effects. This stands in contrast to the notion that replication rates directly measure the abundance of false (i.e. $d = 0$) findings in the literature.

To distinguish between these possibilities, it is necessary to constrain the parameters for our model to values corresponding to replication-averaged effect sizes, mediation, and sample sizes (i.e., τ, σ, and n) typical of a given discipline. Here, we estimate these parameters from the Reproducibility Project: Psychology (RPP) dataset, a large-scale survey of replication in the field of psychology [2]. In this study, researchers attempted to replicate 97 significant findings from the psychology literature. They obtained significance for $\approx 40\%$ of replication attempts, noting effect sizes were smaller on average for replications. We estimate parameters for our model, assuming that true effects are normally distributed about zero (i.e, $d' \sim \text{Normal}(0, \tau)$) with average bias in a given context σ. We further assume that significant original effects are censored by t_c (See Methods). We note that these assumptions are made for comparison with our purely theoretical model, but more sophisticated modeling choices could explicitly model variation as a function of effect size or incorporate mixtures of effect size distributions.

Our model fitting procedure produced an estimate for τ of 0.80, (Cohen's d, 94% C.I.[.65 0.97]), larger than variation attributable to context ($\sigma = 0.61$, 89% C.I.[0.49, 0.71], Fig. 4A, Table S1). Using the joint posterior distribution of parameters, we simulated a body of literature consisting of attempted experiments in which significance was obtained through an independent samples t-test. Effect sizes for each "experiment" were drawn from a normal distribution such that $d \sim \phi(0, \tau)$ and $d_{orig} \sim \phi(d_j, \sigma)$. For simplicity, we consider those with significant results to be "published" proportionate to the observed selection for significance (at $p < .05$) in the original dataset. The published studies are then replicated by conducting an identical statistical test with the replication effect size distributed such that $d_{rep} \sim \phi(d_j, \sigma)$.

At the median sample size from the original experiments $N \approx 50$, our simulations reveal that approximately half of attempted experiments will achieve significance (Fig. 4B). In contrast to previous estimated file drawer ratios of 10 : 1, our simulations suggest that initial experiments will be significant at rates consistent with those observed in registered reports [4, 15]. These results further suggest that increasing sample size above ≈ 200 can ensure the majority of attempted research achieves significance (Fig. 4B). This can also occur if

researchers relax thresholds for significance in any number of ways: by accepting $p < 0.1$ as *marginally significant*, by deviating from pre-registered plans, by publishing exploratory analyses or by engaging in QRPs. By contrast, strengthening thresholds for significance—which has been proposed a solution for the replication crisis[12]—dramatically reduces the number of significant findings . Given preferential publishing and citation of significant findings, researchers choosing to adopt stricter significance thresholds may do so at a cost to their perceived productivity and quantifiable scientific impact (e.g., citations, H-index). Similarly, journals adopting these standards may receive fewer manuscripts.

Our model generates similar observed effect sizes, that are particularly inflated at the sample sizes typical of studies in the replication survey (4C). At these sample sizes, we observe replication rates between 30 and 60%, sharply increasing with sample size (4D). At the median sample size $N \approx 50$, our simulations exhibit higher rates of replication than were observed in the RPP (39%). This is likely due to the idealized nature of our simulations, wherein all "researchers" conduct the same statistical test on data that perfectly meet its assumptions, with identical sample sizes that are independent of the effect size.

Our model does suggest that stricter criteria for significance can improve replication rates, particularly for the smaller sample sizes typical of studies included in the RPP (Fig. 4D, [12]). Yet reducing thresholds for significance has unintended consequences on observed effect sizes among significant findings. For small sample sizes, smaller choices for α inflate estimates of effect sizes because significance is more likely to be achieved when there is directional alignment between mediation and the effect (Fig. 4C). Should significant research continue to be preferentially published or cited, stricter criteria for significance may increase systematic errors in estimating the magnitude of effects (i.e., Type-M error).

If the goal of some reform is simply to improve rates of replication, increasing average sample sizes may be particularly effective (Fig. 4D). However, this effect begins to saturate at $\approx 65\%$ of research replicating for sample sizes greater than ≈ 200. Yet our model further reveals that low rates of replication (and indeed failed replication) may not be particularly indicative of whether psychology is producing results that are correct in direction. Even for regions of parameter space where rates of replication are low, most ($> 80\%$) of studies will identify the correct direction of the effect (i.e., avoid Type-S error, Fig. 4E). This is a natural consequence of variation in observed effect sizes being lower than the average hypothesized effect: it is unlikely that context-specific effects can overcome the true effect enough to obtain significance in the opposing direction.

Similarly, significant replication reversals should be rare ($<\approx 10\%$) in the absence of QRPs, confounded models, or large values for α (Fig. 4F). Somewhat counter-intuitively, small sample sizes may protect against type-S error and reversals by requiring alignment between mediation and directional effects—increasing type-M error. For some disciplines, choice of sample size may act as a lever to manage trade-offs between type-S and type-M error. Further, the low simulated rates of type-S error highlight the possibility that the vast majority of published psychological research is "true" (i.e. consistent in direction), albeit with effect sizes biased by significance as a filter for publication. The low rates of replication observed in the RPP (39% compared to economics 62%) may have been closer to 70% had the original papers and replication survey used arbitrarily large sample sizes.

We note that these results should not be interpreted as the true state of psychology as a field. In reality, the specific tests used and their appropriateness to the data will impact publication,

replication, type-S, and type-M error. Further, there may exist relationships between σ, N, and hypothesized effects. Small effects may vary less than large ones, or researchers may choose sample sizes based on intuition about likely values of d and σ. For these reasons, the above results are better interpreted as reflecting an idealized field with similar observed effect sizes that vary in a similar manner to psychological research. Yet, even in such an idealized environment, high rates of replication may not be possible even absent QRPs or large file-drawers. Moreover, our model highlights how efforts to improve replication can come at a cost to both productivity or Type-S/M error.

Discussion

It is difficult to overstate the importance of ensuring science, as an institution, is reliably producing knowledge. Eroded faith in science has undermined our ability to effectively manage a pandemic, and convince the world that action is needed to address climate change[34, 35]. In a world where point null hypotheses can be true and varying effects matter little, widespread replication failures force us to accept that science is wrought with unethical behavior, full of falsehoods, and wasting substantial resources on investigations that never see the light of day. Those skeptical of scientific inquiry would have cause. Whole disciplines will need to be rebuilt from scratch and textbooks must be rewritten. We would need immediate and drastic reform of scientific institutions and processes far exceeding what has currently been proposed.

However, if we acknowledge the role of varying effects, scientific inquiry can be productive, largely ethical, and generally devoid of fraud and wasted effort. Replication is no longer an arbiter of truth, with successes and failures being minimally informative. Currently proposed scientific reforms in this world would have differing, often unintended consequences. Lowering thresholds for significance may reduce productivity without producing literature that is substantially more calibrated to the broader effects of interest (Fig 4). Increasing sample size may inadvertently make type-S error worse (Fig. 3C). If QRPs do not pose an existential threat to scientific productivity, benefits of increased transparency will need to be re-calibrated against concerns that some reforms may impose disproportionate costs to early career researchers, particularly those whose identities are underrepresented in science [10]. Scientists, scrutinized by their peers and accused of unethical behavior due to failed replications are owed an apology.

Our model is not presented to make claims about the true state of science as a whole—in many ways, over-reliance on a single model is what got us here in the first place [6]. Rather it serves as a tool for viewing replication and its relationship to scientific productivity in a new light. Distinguishing between these two dramatically differing perspectives is essential. Empirical evidence, from meta-analyses and "many labs" studies will be helpful yet need to be grounded in formal theory and methodology [36]. Extensions of our model or others should be compared with observations and adjusted, both within fields and across science.

Of course, we are not the first to point out that varying effects (or heterogeneity) can impact replication [20, 21, 28, 24]. Empirical evidence from meta-analyses in psychology has suggested substantial heterogeneity, argued as sufficient to explain the replication crisis [24]. Yet power analyses conducted on average heterogeneity observed in "many labs" studies were used to argue the opposite [23]. Absent formal methodology to bridge these disparate observations, disagreement over the impact of heterogeneity remains, with camps on either side [21].

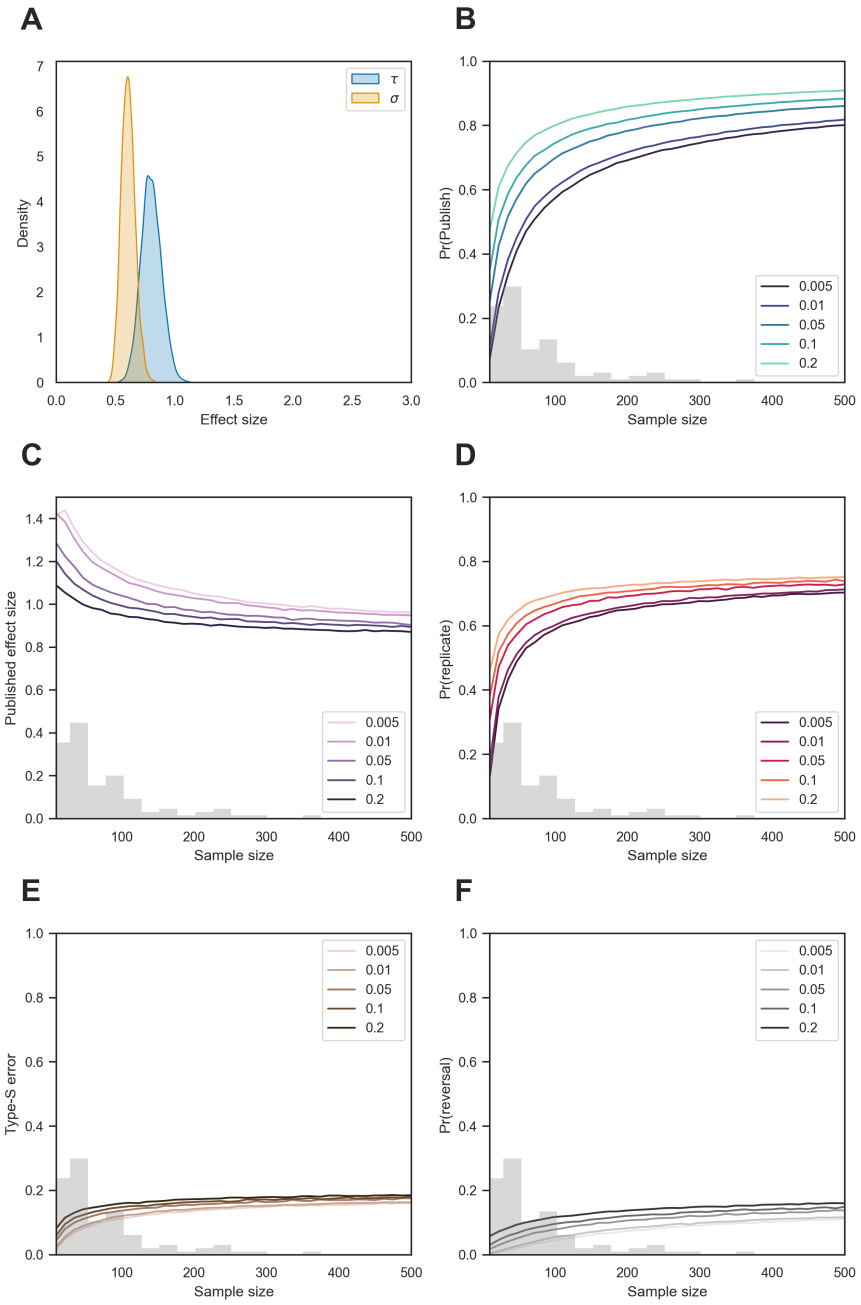

Figure 4: Simulation of publication and replication in psychology based on the RPP dataset. A) Posterior distributions of parameter estimates for hypothesized effect sizes τ and mediation σ B) Rates of publication as a function of sample size for varying levels of α. C) Average published effect sizes as a function of sample size and varying α. D) Rates of replication as a function of sample size and α. E) Type-S error as a function of sample size and α F) Replication significance in the opposing direction, reversals, as a function of sample size and α. For all plots, the grey histogram indicates the distribution of sample sizes of original experiments in the RPP

These conflicting views can be reconciled from the perspective of our model. Within a many labs context, careful protocols define a new distribution of hypothetical replications with a unique $d_{j,ML}$ and smaller $\sigma_{j,ML}$. One would expect to see high replication rates for the subset of replicated studies where this unique $d_{j,ML}$ is large enough to overwhelm $\sigma_{j,ML}$. Indeed this is often the case, although it is typically interpreted as a distinction between "robust" and "fragile" effects [37]. When surveyed across the broader literature, the imagined distribution of replications differs (larger σ, new d_j), altering one-off replication rates. Debates such as the one above highlight a broader trend of relying on verbal argumentation—absent formal theory—to reconcile empirical results from the social sciences into broader conclusions about science as a whole [36]. Formal theory will be essential for making sense of conflicting observations and understanding when and whether they generalize.

The simplicity that enabled our analysis leads to several limitations. Questionable research practices certainly do occur. However, their consequences on inference may differ substantially, in a manner that is dependent on the parameters unique to a given discipline or area of study (Supplementary Fig S1). Moreover, our model assumes that researchers have a well-specified model, appropriate to their data and question. Poorly specified models could increase σ, or lead to apparently reliable findings that are merely the result of a particularly robust confound or violated assumption [36]. More generally, even correct statistical inference provides no guarantee of correct interpretation or decision-making.

We strongly caution against interpreting our model, in the exact form described above, as something that can be applied across the breadth of scientific inquiry without adaptation or adjustment. Fields vary widely in terms of their average effect sizes, sample sizes, variability, and the distributions of each. In some contexts the parameters of our model may covary, for instance with larger effects being more variable [23]. If warrented, fragility of effects could be incorporated by assuming a distribution of σ rather than a fixed value. For some contexts, point nulls may be argued to apply. Indeed these could be recovered from our model by considering a distribution of effect sizes that is a mixture of a Dirac delta function centered on zero and some other distribution of "true" effects, perhaps with $\sigma \gtrsim 0$ (Supplementary Fig. S2, S3). More generally, implementations and extensions of our model restore (or erode) the coupling of replication and other measures of productivity. It is precisely this lack of reliable coupling that makes replication a poor general measure of scientific productivity.

A corollary of this variation across disciplines is that conflict between our model and observations in some discipline is both to be expected and cause for further investigation. Indeed it is unreasonable that a single model—ours or any other—could provide universal insight into scientific best practices that incorporate every disciplines' unique properties, constraints, costs, and benefits. After all, how could best practices derived in psychological studies on Amazon Mechanical Turk be expected to apply to studies of elusive snow leopards, or the petabytes of data gathered by particle accelerators.

Yet at this point in time, we are barreling forward with whole-of-science scientific reforms, from journal policies to norms of preregistration and sample size expectations. These reforms have placed replication front and center, as a cornerstone of scientific inquiry. Doing so has eroded public trust in science [7] and our own trust in fellow scientists' abilities and motivations. Here we show that relaxing the transparently fraught assumptions of traditional models raises doubts about whether replication can be an arbiter of truth for specific studies, or a meaningful measure of knowledge production. A varying-effects framing yields a view of scientific productivity that is more nuanced and adapatable with far less baggage—less

wasted effort and no need for widespread QRPs or a literature rife with falsehood. Given this possibility, placing replication as a cornerstone of scientific productivity and reform warrants reflection.

Methods

Theoretical Analysis

Our analyses was conducted in using Python 3.9.10. We analyzed our purely theoretical model as described in text using standard functions in Numpy and Scipy. For each figure, we constructed a mesh grid of parameters and numerically evaluated our model for each parameter combination.

Parameter Estimation from Replication Surveys

To estimate parameters from replication survey datasets, we adapted our theory to a generative Bayesian model coded in PyMC3:

$$
\begin{aligned}
\sigma &\sim \text{Exponential}(1) \\
\tau &\sim \text{Exponential}(1) \\
d &\sim \text{Normal}(0, \tau) \\
s &= \sqrt{\sigma^2 + \frac{1}{\sqrt{n}}} \\
d_{o,i} &\sim \text{TruncatedNormal}(d_i, s) \\
d_{r,i} &\sim \text{Normal}(d_i, s)
\end{aligned}
$$

Effect sizes from the original dataset were converted into Cohen's d. As effect sizes in the dataset were presented as absolute values, effects assigned a direction, s_i, at random ($s = \{-1, 1\}$). For each of i studies, this model assumes the original and replication effect sizes ($d_{o,i}$ and $d_{r,i}$, Cohen's d) as normally distributed with mean mu_o and mu_r and standard deviation defined by σ and measurement error. To accommodate censoring from publication filters, d_o was estimated using normal distribution truncated by the minimum effect size that would have achieved significance for the sample size. Values for d are partially pooled using shared hyperparameters for τ (the average effect size). Prior predictive simulations were used to ensure the model and priors produced reasonable ranges of effect sizes. Posterior predictive checks were used to evaluate model fit.

Simulations

We simulated a body of published literature (Fig 4) using 500 draws from the joint posterior distribution from our parameter estimation. For each draw, we generated 1000 true effect sizes corresponding to hypothesized research and distributed such that $d_{true} \sim \text{Normal}(0, \tau)$. For

each of the true effect sizes, an initial "experiment" was conducted by generating an observed effect size such that $d_{orig} \sim \text{Normal}(d_{true}, \sigma)$.

For each effect size, we calculated the power of a two-sample, two-tailed t-test, $1-\beta$. Studies were considered "published" with probability $\theta \times (1-\beta) + \beta \times (1-\theta)$, where θ was the observed proportion of significant findings in the literature. We then drew a second effect size, d_{rep}, for each published effect using the same procedure for obtaining d_{orig}. One-tailed power analyses were used to calculate the probability of replication and reversal. Similarly, one-tailed power analyses were used on d_{orig} of published studies to calculate the rate of type-s error. This processes was repeated across varying values for α and N and shown in the Figure 4.

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Author Contributions

All authors were involved in the conceptualization of the study. J.B-C and R.P.M. devised the initial theoretical model. J.B-C analyzed the data. All authors were involved in writing up the results.

Competing Interests

The Authors Declare No Competing Interests

1. Code Availability

All code used to generate the analysis are available on GitHub (https://github.com/josephbb/ReplicationSurv

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