

CAUSAL EXAGGERATION: UNCONFOUNDED BUT INFLATED CAUSAL ESTIMATES

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ABSTRACT

The credibility revolution in economics has made causal inference methods ubiquitous. Simultaneously, an increasing amount of evidence highlights that the literature strongly favors statistically significant results. I show that these two phenomena interact in a way that can substantially worsen the reliability of published estimates: while causal identification strategies alleviate bias caused by confounders, they reduce statistical power and can create another type of bias—exaggeration—when combined with selection on significance. This is consequential as estimates are routinely turned into decision-making parameters for policy makers conducting cost-benefit analyses. I characterize this confounding-exaggeration trade-off theoretically and using realistic Monte Carlo simulations replicating prevailing identification strategies and document its prevalence in the literature. I then discuss potential avenues to address this issue.

[Link to the most recent version of the paper](#)

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I INTRODUCTION

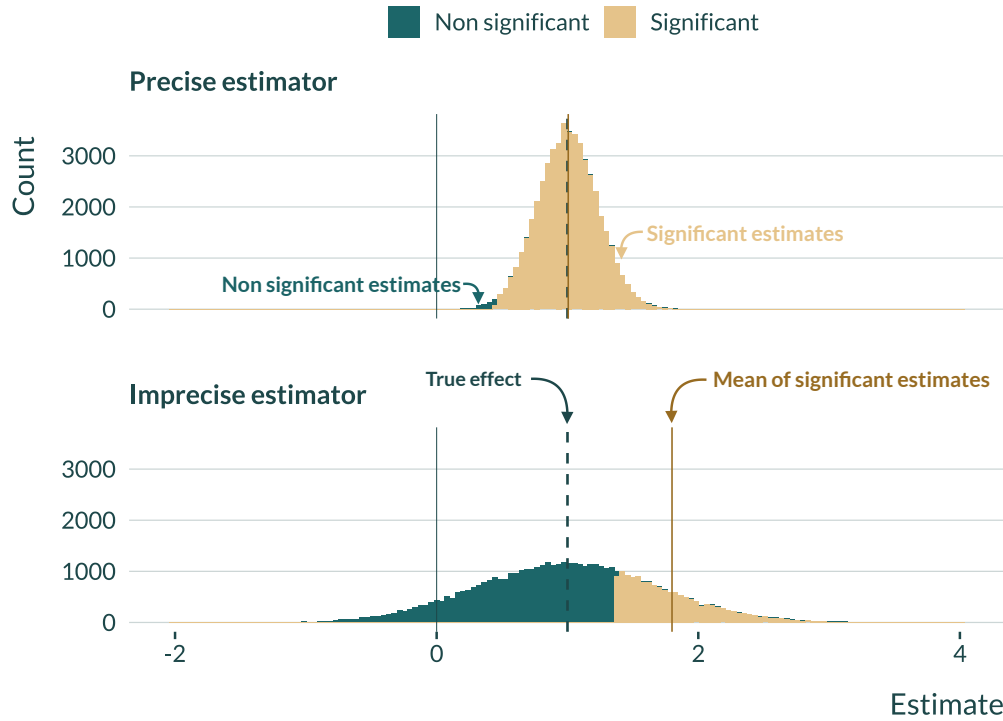
One of the main challenges of empirical economics is identifying causal effects. Identification strategies such as Regression Discontinuity (RD), Instrumental Variables (IV), Difference-in-Differences (DiD) and event studies help us achieve this goal. To do so, these strategies only use a portion of the variation in the data. They exploit the exogenous part of the variation in the treatment or decrease the sample size by only considering observations for which the as-if random assignment assumption is credible. This reduction in the variation used can decrease precision and thus statistical power—the probability of rejecting the null hypothesis when it is false, or put simply, the probability of obtaining a statistically significant estimate. There is, therefore, a tension between reducing confounding and statistical power.

When statistical power is low, not only is the estimator imprecise but statistically significant estimates exaggerate the true effect size (Gelman and Tuerlinckx 2000, Ioannidis 2008, Gelman and Carlin 2014). Only estimates at least 1.96 standard errors away from zero are statistically significant at the 5% level. In under-powered studies, these estimates make up a selected subsample of all estimates, located in the tails of the distribution of all possible estimates. The average of these statistically significant estimates differs from the true effect, located at the center of the distribution if the estimator is unbiased. They exaggerate the true effect and the less precise the estimator, the larger exaggeration is. Figure 1 illustrates the inflation of significant estimates caused by imprecision. When power is low, obtaining a statistically significant estimate from an unbiased estimator does not guarantee that it will be close to the true effect. An estimator $\hat{\beta}$ of the true effect β might be unbiased in the traditional sense of $\mathbb{E}[\hat{\beta}] = \beta$ but conditionally biased in the sense that $\mathbb{E}[\hat{\beta} | \text{Significant}] \neq \beta$. For statistically significant estimates, the tension between statistical power and reducing confounding is thus a tension between reducing confounding and exaggerating the true effect size.

Yet, exaggeration only arises under two conditions: 1) a publication bias favors statistically significant results and 2) statistical power is low. A large body of literature underlines that the economics literature selects results based on statistical significance (Rosenthal 1979, Brodeur et al. 2016, Andrews and Kasy 2019, Abadie 2020, Brodeur et al. 2020, for instance). Additional studies have highlighted its frequent and substantial lack of statistical power and resulting exaggeration (Ioannidis et al. 2017, Ferraro and Shukla 2020). Even in experimental economics, with a high level of control and an arguable absence of confounders, studies from top economics journals failed to replicate, the original estimates being on average inflated by a factor of at least 1.5 (Camerer et al. 2016). In the non-experimental economics literature, where statistical power is rarely a central consideration under current practices, several meta-analyses provide evidence of consequential exaggeration. Ioannidis et al. (2017) finds that the median statistical power in a wide

Figure 1 – Significance and distribution of two unbiased estimators with different variances

Imprecise estimators can cause exaggeration



Notes: 100,000 draws from two normal distributions $\mathcal{N}(1, 0.05)$ and $\mathcal{N}(1, 0.5)$. Significance level: 5%

range of areas of economics is no more than 18%. Despite the widespread use of convincing causal identification strategies and usually large sample sizes, they show that nearly 80% of estimates are likely exaggerated by a factor of two. In environmental economics, using a more conservative approach, [Ferraro and Shukla \(2020\)](#) finds that 56% of estimates are exaggerated by a factor of two or more. I provide evidence of exaggeration in a subfield of this literature, that on the acute health effects of air pollution in a companion paper ([Bagilet 2023](#)) and further expand this analysis in the present paper. The magnitude of exaggeration can be considerable and in some situations could be on par with that of a bias caused by confounders, as illustrated in section II. It is therefore crucial to take exaggeration into account and to understand its drivers.

Accurate point estimates are instrumental as they often inform policy decisions through Cost-Benefit Analyses (CBA). For instance, environmental economics estimates enter the computation of the Social Cost of Carbon or routinely help policy makers decide of the implementation of regulations. Yet, the underlying effects can be relatively small and thus difficult to capture, making the studies subject to exaggeration. Estimates of the impact of environmental regulations on job losses constitute a good example ([Gray et al. 2023](#)). For instance, [Walker \(2011\)](#) documents

the impact of the Clean Air Act amendments of 1990 on employment and finds an effect of -14.2% (s.e. 4.3). For similar policies, other studies find smaller effects, of the order of magnitude of -3% (Greenstone 2002, Gray et al. 2023). The design of Walker (2011) would not be precise enough to retrieve an effect size of this magnitude. If the true effect was in fact of this magnitude, the statistical power of the study would be 11% and significant estimates would exaggerate the true effect by a factor of 3.5 on average. In this example, bias caused by exaggeration would be substantial and could have detrimental policy implications.¹

In the present paper, I argue that the use of causal identification strategies contributes to exaggeration. Reviewing a literature, using a mathematical derivation and Monte Carlo simulations, I show that design choices in quasi-experimental studies can be seen as a trade-off between avoiding confounding and overestimating true effect sizes due to a resulting loss in power. To limit the threat of confounding, causal inference methods discard variation and therefore reduce statistical power. When combined with a statistical significance filter, this results in exaggeration bias. While causal identification strategies are essential to describe causal relationships, this paper emphasizes that a perfectly convincing identification does not guaranty an absence of “bias” and that improving identification can actually pull us away from the true effect. The same strategies which remove the bias caused by confounding factors actually introduce another type of bias.

All causal identification strategies discard variation in order to identify causal effects but the confounding-exaggeration trade-off is mediated through a distinctive channel for each of them. RD designs discard part of the variation by only considering observations within the bandwidth, decreasing the effective sample size and thus precision, even if the initial sample size was large. An IV strategy only uses the subset of the variation in the treatment that is explained by the instrument. In studies leveraging exogenous shocks, the variation used to identify an effect sometimes only comes from a limited number of changes in treatment status. Approaches that do not actually leverage natural experiments but aim to identify a causal effect by controlling for confounders also limit the variation used. Matching prunes units that cannot be matched and thus reduces the effective sample size. Adding controls or fixed effects can increase the variance of the estimator and exaggeration if they absorb more of the variation in the treatment than in the outcome variable.

Since causal identification strategies can be interpreted as ways of controlling for confounders, this last point actually ties all the strategy-specific arguments together. Fixed Effects (FEs) based identification strategies such as DiD control for the invariant, unobserved, and arguably endogenous part of the variation in the outcome. An IV approach essentially partials out the variation in x unexplained by the instruments. Fuzzy-RD and propensity score matching can be

1. Of course, in the particular setting of Walker (2011), the true effect might be larger than 3%. I am not claiming that the study is flawed but instead that its level of precision would produce inflated significant estimates if the true effect was of the order of magnitude of 3%.

thought of as control function approaches, of the forcing variable and propensity score respectively. In addition, excluding observations that are outside the bandwidth or unmatched is equivalent to controlling for observation-level fixed effects for these observations. When these identification strategies absorb more of the variation in the treatment than in the outcome, they increase the variance and can cause exaggeration. Considering a simple linear homoskedastic model gives the intuition for this trade-off between exaggeration and omitted variable bias (OVB) for control approaches. Let $y_i = \alpha + \beta x_i + \delta w_i + u_i$, $\forall i \in \{1, \dots, n\}$, with x the variable of interest, w a potentially unobserved variable correlated with x and u an error term. Under usual assumptions and using the Frisch-Waugh-Lovell theorem, we get that $\sigma_{\hat{\beta}_{\text{OVB}}}^2$ and $\sigma_{\hat{\beta}_{\text{CTRL}}}^2$, the variance of the estimators for β when omitting w (short regression) and controlling for it (long regression) are respectively:

$$\sigma_{\hat{\beta}_{\text{OVB}}}^2 = \frac{\sigma_{u_{\text{OVB}}}^2}{n \sigma_x^2} = \frac{\sigma_{y^{\perp x}}^2}{n \sigma_x^2} \quad \text{and} \quad \sigma_{\hat{\beta}_{\text{CTRL}}}^2 = \frac{\sigma_{u_{\text{CTRL}}}^2}{n \sigma_{x^{\perp w}}^2} = \frac{\sigma_{y^{\perp x, w}}^2}{n \sigma_{x^{\perp w}}^2}$$

where $\sigma_{u_{\text{OVB}}}^2$ and $\sigma_{u_{\text{CTRL}}}^2$ are the variances of the residuals in the regression of y on x and of y on x and w respectively, $\sigma_{y^{\perp x}}^2$ and $\sigma_{y^{\perp x, w}}^2$ are the variances of the parts of y that are orthogonal to x and to x and w respectively, σ_x^2 is the variance of x and $\sigma_{x^{\perp w}}^2$ is the variance of the part of x orthogonal to w . Thus,

$$\sigma_{\hat{\beta}_{\text{ovb}}}^2 < \sigma_{\hat{\beta}_{\text{ctrl}}}^2 \Leftrightarrow \frac{\sigma_{y^{\perp x}}^2}{n \sigma_x^2} < \frac{\sigma_{y^{\perp x, w}}^2}{n \sigma_{x^{\perp w}}^2} \Leftrightarrow \frac{\sigma_{x^{\perp w}}^2}{\sigma_x^2} < \frac{\sigma_{y^{\perp x, w}}^2}{\sigma_{y^{\perp x}}^2}$$

Controlling for w will increase the variance of the estimator if the fraction of the variance unexplained by w is greater for $y^{\perp x}$ than for x . Put differently, if controlling absorbs more of the variation in x than in the residual part of y ($y^{\perp x}$), it will increase the variance of the estimator. Since exaggeration increases with the variance of the estimator, controlling for a confounder can increase exaggeration. This has direct implications when using fixed effects: an exaggeration bias can arise from the use of fixed effects if they absorb more of the variation in x than in $y^{\perp x}$. Under some circumstances discussed in sections III and IV, controlling can even produce an exaggeration bias larger than the OVB that would result from an absence of controls.

In the remainder of the paper, I first document the magnitude of the trade-off. To do so, I build on existing literature reviews to discuss evidence of exaggeration bias in a large set of causal economic studies (Brodeur et al. 2020, Young 2022, Bagilet 2023). I then focus on IV designs as they allow for a within-study comparison between causal and non-causal estimates, the Two-Stage Least-Squares (2SLS) and the corresponding “naive” Ordinary Least Squares (OLS) respectively. For many IV studies published in top journals, the IV both reduces precision, limits statistical power to the point that 2SLS estimates reject the OLS point estimate and are not able to provide strong evidence supporting that the OLS is biased (Young 2022). By reducing precision, IVs

and the causal identification strategies at large, likely induce exaggeration and produce biased significant estimates. In the literature on the short term health effects of air pollutions, only 2% of the IV designs would retrieve an effect size equal to that of the “naive” OLS—at the conventional 80% power threshold. The median exaggeration factor would be of 4.5.² Finally, for an example study, I further compare confounding and exaggeration biases. I show that in this study the bias of the IV could be 3.1 times larger than that of the OLS and that exaggeration likely explains this difference.

Next, I derive a formal proof of the existence of the trade-off for prevailing causal identification strategies. Specifically, I show that the bias caused by exaggeration can be larger than the one caused by confounders. I also analyze the drivers of exaggeration and demonstrate that it increases as the strength of the instruments decreases, the number of exogenous shocks decreases or when controlling for a confounder absorbs more of the variation in the treatment than in the outcome.

Then, I show that this “causal exaggeration” arises for realistic parameter values by further exploring its drivers in realistic settings. The exaggeration of statistically significant estimates can be defined as the absolute value of the ratio of the estimated effect over the true effect, a quantity which is never known in a real world setting. In order to be able to compute this quantity, I turn to simulations. Monte-Carlo simulations also allow to vary the value of the parameter of interest *ceteris paribus*, something that would not be possible otherwise. To make these simulations concrete, I calibrate the simulations to emulate typical low-powered studies from environmental, education, labor, health and political economics. All other simulation assumptions are chosen to facilitate the recovery of the effect of interest: I consider simple linear models with constant and homogenous treatment effects, *i.i.d.* observations and homoskedastic errors. All the models are correctly specified and accurately represent the data generating process, except for the omitted variable. I find that causal exaggeration can be substantial in realistic settings where the remaining variation available for identification is limited.

Finally, I discuss concrete avenues to address this causal exaggeration when carrying out a non-experimental study³. First, a series of tools can be used to evaluate the potential magnitude of confounding and exaggeration issues separately. Sensitivity analyses help with the former while power calculations help with the latter. For instance, the sensitivity analysis tools developed in [Cinelli and Hazlett \(2020\)](#) enable to assess how strong confounders would have to be to change the estimate of the treatment effect beyond a given level we are interested in. Then, considering the attention given to bias avoidance in the economics literature, I underline that making power central

2. I will replace this analysis with an analysis of the broader IV literature studied in [Young \(2022\)](#). However, the data I need to do so are not publicly available and I will only have access to them in a few weeks.

3. In experimental studies, there are essentially no confoundings. A solution to increase power and reduce exaggeration is generally to increase sample size, reduce noise by improving measurement or improving balance or to focus on larger potential effects.

to non-experimental analyses, even after an effect has been found, would help limit bias caused by exaggeration. Prospective power simulations help identify the design parameters affecting power and exaggeration by approximating the data generating process (Gelman 2020, Black et al. 2021). Retrospective power calculations allow to evaluate whether a study would have enough power to confidently estimate a range of smaller but credible effect sizes (Gelman and Carlin 2014, ?). Focusing more specifically on the trade-off and its drivers, I present tools to visualize the variation actually used for identification when using causal identification strategies. The [companion website](#) describes in details how such analyses can be implemented. Finally, I briefly discuss potential solutions to mitigate this trade-off.

This paper contributes to three strands of the applied economics literature. First, the idea that causal identification estimators, while unbiased, may be imprecise is not new; this is part of the well-known bias-variance trade-off (Imbens and Kalyanaraman 2012, Deaton and Cartwright 2018, Hernán and Robins 2020, Ravallion 2020). I approach this literature from a different angle: through the prism of statistical power and publication bias. Not only the limited precision resulting from the use of causal identification methods could make it difficult to draw clear conclusions regarding the exact magnitude of the effect but I argue that it might also inherently lead to inflated published effect sizes, creating another “bias”. The bias-variance trade-off can in fact be a bias-bias trade-off.

Second, studies discussing the exaggeration of statistically significant estimates due to a lack of power usually do not investigate its determinants or focus on specific causal identification methods separately (Ioannidis et al. 2017, Schell et al. 2018, Ferraro and Shukla 2020, Black et al. 2022, ?, Arel-Bundock et al. 2022). In a companion paper, I highlight tangible design parameters that can cause exaggeration for a wide range of empirical designs (Bagilet 2023). In the present paper, I take a step back and propose an overarching mechanism, inherent to causal identification strategies as a whole, and that can explain these issues: although each strategy does so through different means, in essence they discard part of the variation, thereby increasing the risks of exaggeration.

Third, this study contributes to the literature on replicability in economics (Camerer et al. 2016, Ioannidis et al. 2017, Christensen and Miguel 2018, Kasy 2021). The trade-off presented in this paper suggests that the widespread use of convincing causal identification methods in economics may not shield the field from potential replication threats.

In the following section, I document evidence of causal exaggeration in the causal economics literature. In section III, I study the drivers of exaggeration and formally show in a simple setting that the use of causal identification strategies can exacerbate it. In section IV, I implement realistic Monte-Carlo simulations to illustrate the existence of the confounding-exaggeration trade-off. I discuss potential solutions to navigate this trade-off in section V and conclude in section VI.

II CAUSAL EXAGGERATION IN THE LITERATURE

The trade-off presented in this paper only has concrete implications if the use of causal identification strategies yields substantial exaggeration, especially as compared to the amount of confounding bias these methods allow avoiding.

The literature on the acute health effects of air pollution, that I explore in a companion paper, provides a good illustration of potential exaggeration of causal methods (Bagilet 2023). The historical literature mostly relies on associations (Dominici and Zigler 2017, Bind 2019). A more recent literature based on causal identification strategies confirms the adverse effects of air pollution in the short term (Schwartz et al. 2015; 2018, Deryugina et al. 2019). Yet, causal estimates are substantially larger than what would have been predicted by the standard epidemiology literature, some estimates being more than 10 times larger. Even within a given setting, my literature review shows that the median of the ratio of the obtained 2SLS to their corresponding “naive” OLS estimates is 3.8. Similarly, the data in Young (2022) shows that in a set of 30 instrumental variables papers published in journals from the American Economic Association, the median ratio of headline IV estimates over the corresponding OLS is 2.3 and more than 5.4 for 25% of the estimates. What can explain that causal methods yield such large effects sizes, as compared to non-causal methods? Causal strategies could arguably remove omitted variable bias, reduce attenuation bias caused by classical measurement error in air pollution exposure or target a different causal estimand. But as I argue in this section, exaggeration and imprecision could also explain part of this difference. In the example case of studies on the short-term health effects of air pollution, causal studies often display a low relative precision, not only because effects are typically small and the data relatively coarse—at the city-day level—but also because they sometimes leverage rare exogenous shocks such as public transportation strikes, take advantage of air pollution alerts to build RDD that limit sample size or use instruments that do not strongly predict air pollution levels.

II. 1 QUANTIFYING EXAGGERATION

As discussed in the introduction, lack of power and exaggeration are widespread in the economics literature; Ioannidis et al. (2017) shows that nearly 80% of estimates published in a wide array of empirical economic literatures are exaggerated, typically by a factor of two and one-third by a factor of four or more. To focus more specifically on the causal inference literature, I first leverage data from Brodeur et al. (2020). This paper reviews of the universe of papers published in the top-25 economics journals in 2015 and 2018, to examine the extent of selection on significance in these articles, comparing across methods. It shows that IV and to a lesser extent DID are particularly subject to publication bias—one of the two ingredients of exaggeration—while RDD and RCT seem to be less prone to this issue.

The exaggeration of an estimator corresponds to the expected value of the absolute value of significant estimates. As such, it depends on the true magnitude of the estimand of interest; this true effect determines the value the estimator is centred on. Exaggeration can therefore only be calculated by hypothesizing true effect sizes. To circumvent this limitation, I first evaluate the proportion of studies in [Brodeur et al. \(2020\)](#) that would have a design reliable enough to retrieve an effect size equal to half of the obtained estimate. This is done by drawing a large number of times from a normal distribution centred on this hypothetical effect size and with a standard deviation equal to the standard error of the estimate in the study and computing the average of the draws that are 1.96 standard errors away from 0. Under this assumption, the median power would be 37% and the median exaggeration ratio of significant estimates would be 1.6. Only 11% of studies would have a power greater than 80%. One quarter of the studies would, on average, exaggerate the true effect sizes by a factor greater than 2.1. These results are relatively similar across methods (IV, DID, RDD and RCT).

This hypothesis on the true effect size, despite enabling to get an overview of this wide literature, suffers from an important limitation. There is no *a priori* reason to believe that the magnitude of the true effect of a specific estimation would be equal to half its point estimate. However, since [Ioannidis et al. \(2017\)](#) finds a typical exaggeration of two in the economics literature, we may expect that this assumption is reasonable, on average. Regardless, I am not claiming that it would be the true effect but rather wonder what would be the power and exaggeration under this justifiable assumption. This approach is also to some extent conservative: hypothesized effect sizes based on exaggerated estimates will be too large and will thus minimize exaggeration.

To reduce the burden of assumptions, I focus on IV designs as they allow making a less arbitrary intra-study comparison, comparing the 2SLS estimates to the “naive” OLS one. In the literature on the short-term health effects of air pollution, assuming the true effect is close to the naive estimate, the median power of the IVs would only be 8.4% and median exaggeration would reach 4.5. Only 2.0% of the IV designs would have a power of 80% to detect an effect size of the magnitude of the OLS estimate. Significant 2SLS estimates would be greatly biased. This can be explained by the fact that the IV drastically reduces precision; the median standard error of the 2SLS estimators is 3.8 times larger than the one of the corresponding OLS. [Young \(2022\)](#) has documented this reduction in precision more broadly, showing that published IVs papers have little power. This lack of precision makes most of the 2SLS estimates statistically indistinguishable from the corresponding OLS estimate, despite the fact that 2SLS point estimates substantially differ in magnitude from their OLS counterpart, even regularly being of a different sign.

II. 2 ILLUSTRATION OF THE TRADE-OFF

In order to limit hypotheses and to investigate causal exaggeration further, I focus on an example study, [He et al. \(2020\)](#), investigating the impact of PM2.5 air pollution on mortality. I compare the bias of the “naive” OLS to that of the IV by computing their distance to an estimate of the “true effect” they target. I define this “true effect” in two ways. First, I use the results of a meta-analysis of epidemiological studies ([Shah et al. 2015](#)). By pooling a number of studies carried out in various contexts, this meta-estimate might represent the average effect one may expect from such a study. However, the studies in this meta-analysis do not rely on canonical causal identification strategies and may be thought of as suffering from confounding. I thus consider the result of [Deryugina et al. \(2019\)](#)—a precise causal study that may be less exposed to exaggeration—as an alternative estimate of the true effect. This estimate may be context specific and the “true effect” in [He et al. \(2020\)](#) may deviate from these. The present discussion is conditional on this true underlying effect being close to these hypothesized true effect sizes.

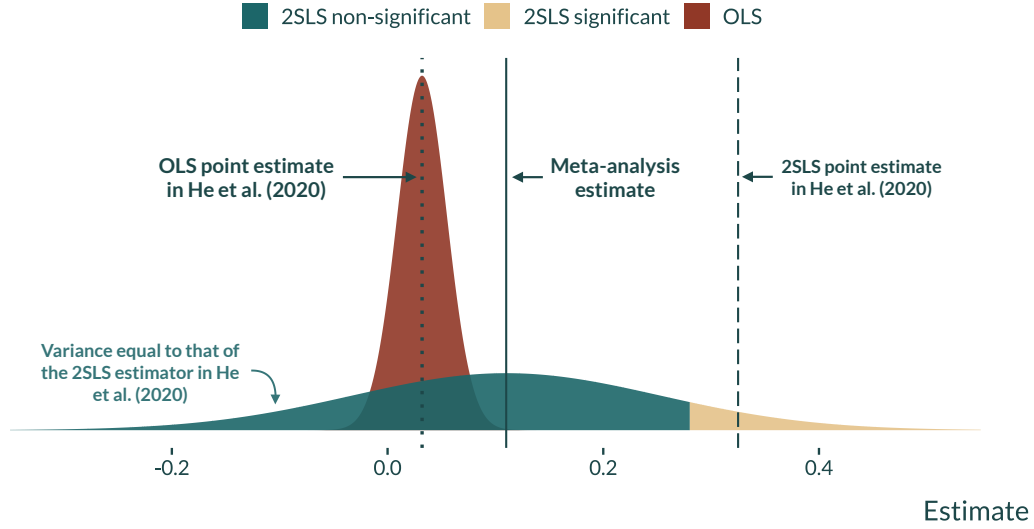
[He et al. \(2020\)](#) finds that a “ $10\mu g.m^{-3}$ increase in PM2.5 increases mortality by 3.25%” (s.e. 1.43%). Their corresponding OLS results suggest a 0.32% increase (s.e. 0.23%). For a similar increment in air pollution, [Shah et al. \(2015\)](#) and [Deryugina et al. \(2019\)](#) document a 1.1% and 1.8% increase in mortality respectively. The OLS estimate in [He et al. \(2020\)](#) is closer to the “true effect” based on [Shah et al. \(2015\)](#) than their 2SLS estimate. Provided that the three estimands are comparable, the bias of the IV is larger than that of the OLS. If the true effect was in fact closer to the one found by [Deryugina et al. \(2019\)](#), both biases would be roughly equal and the bias of the IV still substantial.

Exaggeration could explain this difference. Even if the 2SLS estimator effectively removes all conventional biases, the design in [He et al. \(2020\)](#) would still yield exaggerated statistical significant estimates. Figure 2 illustrates this point.

The distribution of the 2SLS estimator represented in figure 2 assumes that the estimator is unbiased and thus centered on the meta-estimate found in [Shah et al. \(2015\)](#). The variance of this distribution corresponds to the variance of the 2SLS estimator found in [He et al. \(2020\)](#). Due to the lack of precision of this design, the statistically significant estimates are located in the tail of the distribution and substantially exaggerate the true effect, by a factor 3.2 on average. The 2SLS estimate found in [He et al. \(2020\)](#) could be one of these estimates. The distribution of the OLS estimator is the one obtained by the authors. I ignore exaggeration of the OLS for clarity but since the OLS is biased downward, inflating it would yield an estimate closer to the true effect.

This example illustrates that in a published study where a causal identification strategy substantially reduces the precision of the estimator, the resulting statistically significant estimates

Figure 2 – Illustration of the Confounding-Exaggeration Trade-off in He et al. (2020)



Notes: The distribution for the 2SLS estimator is centered on the true effect, represented by the solid line and defined as the meta-estimate found in Shah et al. (2015). Its variance is equal to the one of the 2SLS estimator in He et al. (2020). The distribution for the OLS estimator is centered on the OLS estimate found in He et al. (2020) and its variance equal to that of this same estimator. The dashed and dotted lines represent the 2SLS and OLS estimates found in He et al. (2020) respectively.

may be further away from the true effect than the “naive” OLS estimate, even if the estimator is unbiased. Note that a comparable result holds if the true effect is equal to the one found in Deryugina et al. (2019). With this design, the average exaggeration would be 3.1 times larger than the OVB (or 1.3 if the true effect is closer to the one found in Deryugina et al. (2019)).

III MATHEMATICAL DERIVATION

In this section, I formally prove the existence of the confounding-exaggeration trade-off and describe its drivers in a simple setting. To do so, I first define an exaggeration ratio and show that it increases with the variance of normally distributed biased estimators. This leads me to computing the asymptotic distributions of a series of estimators in order to prove their normality and study drivers of their variances, and ultimately of their exaggeration ratios. Finally, I show that, for any magnitude of OVB, exaggeration can be greater when using a causal inference method than the overall bias combining exaggeration and OVB in the naive regression.

III. 1 PROPERTIES OF THE EXAGGERATION RATIO

Following [Gelman and Carlin \(2014\)](#), we can define the exaggeration ratio E , as the expectation of the absolute value of significant estimates over the absolute value of the true effect. For an estimator $\hat{\beta}$ of a true effect β , with standard deviation σ and a two-sided hypothesis test of size α with threshold value z_α , let

$$E(\hat{\beta}, \sigma, \beta, z_\alpha) = \frac{\mathbb{E} \left[|\hat{\beta}| \mid \beta, \sigma, |\hat{\beta}| > z_\alpha \sigma \right]}{|\beta|} \quad (1)$$

[Lu et al. \(2019\)](#) and [Zwet and Cator \(2021\)](#) showed that, for given test and true effect sizes, the exaggeration ratio increases with the variance of an unbiased normally distributed estimator. We can extend this proof to biased estimators and get that: ⁴

Lemma 1. *For an estimator $\hat{\beta}_b \sim \mathcal{N}(\beta + b, \sigma^2)$ of a true effect of magnitude β and a fixed bias b of the same sign as and independent from the true effect,*

- *E is a decreasing function of the Signal-to-Noise Ratio (SNR), $\frac{\beta}{\sigma}$, and only depends on σ through this SNR.*
- $\lim_{\sigma \rightarrow \infty} E(\hat{\beta}_b, \sigma, \beta, z_\alpha) = +\infty$.

Figure 1 provides a clear intuition for these results in the unbiased case. Note that here, we focus on cases in which the bias is in the same direction as the true effect so that exaggeration from causal inference methods and OVB do not cancel each other.

Based on lemma III. 1, to study how exaggeration evolves with the IV strength in an IV setting, the number of exogenous shocks in a reduced form and the correlation between the explanatory variable of interest and the omitted variable of interest, we can show asymptotic normality and study how the variances of these estimators evolve with these parameters. This relies on the assumption that the sample size is large enough so that the sample distribution of the estimator is well approximated by their asymptotic distribution.

III. 2 SETTING AND DATA GENERATING PROCESS

Consider a usual linear homoskedastic regression model with an omitted variable. For any individual $i \in \{1, \dots, n\}$, we write:

$$y_i = \beta_0 + \beta_1 x_i + \delta w_i + u_i \quad (2)$$

4. All the proofs of the lemma and theorems are in appendix A.

where y is the outcome, x the explanatory variable, w an unobserved omitted variable, u an unobserved error term. $(\beta_0, \beta_1, \delta) \in \mathbb{R}^3$ are unknown parameters. β_1 is the parameter of interest.

Assume homogeneous treatment effects and homoskedasticity, along with the usual OLS assumptions (*i.i.d.* observations, finite second moments, positive-definiteness of $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i']$ —with $\mathbf{x}_i = (1, x_i)'$ — and u_i conditional mean-zero and uncorrelated with x_i and w_i). Assume that w_i is unobserved, correlated with x_i and that $\delta \neq 0$. To simplify the derivations, I further assume that the unobserved variable is centered, *i.e.* $\mathbb{E}[w_i] = 0$. I also assume that the variance of the component of w_i that is orthogonal to x_i (denoted $w_i^{\perp x}$) does not vary with x_i , *i.e.*, $\text{Var}(w_i^{\perp x} | x_i) = \text{Var}(w_i^{\perp x})$. Consider the following data generating process for x_i :

$$x_i = \mu_x + \gamma w_i + \epsilon_i \quad (3)$$

where $\gamma \in \mathbb{R}^*$ since x and w are correlated. Set $\rho_{xw} = \text{corr}(x, w) = \frac{\gamma \sigma_w}{\sigma_x}$. In the IV and reduced form sections, I further assume that there exists a valid instrumental variable z_i for x_i , *i.e.* that $\mu_x + \epsilon_i = \pi_0 + \pi_1 z_i + e_i$ where $(\pi_0, \pi_1) \in \mathbb{R}^2$ are unknown parameters. The existence or not of this valid instrument does not affect the results in the controlled and OVB cases. Since the instrument is valid, it satisfies exogeneity, *ie* $\mathbb{E}[z_i u_i] = 0$ and $\mathbb{E}[z_i w_i] = 0$, relevance, *ie* $\text{rank}(\mathbb{E}[z_i \mathbf{x}_i']) = 2$, and positive-definiteness of $\mathbb{E}[z_i z_i']$. The data generating process for x_i becomes:

$$x_i = \pi_0 + \pi_1 z_i + \gamma w_i + e_i \quad (4)$$

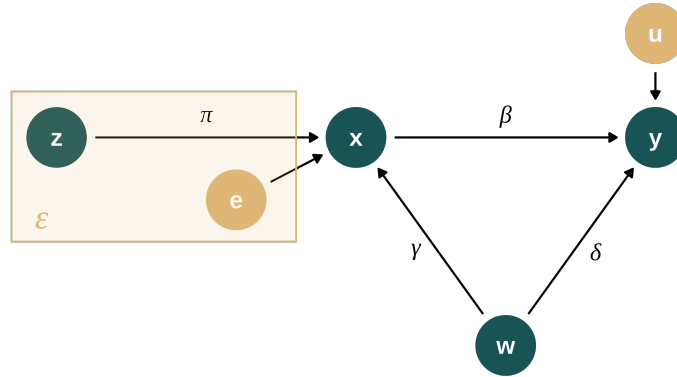
I assume that e_i is uncorrelated with z_i and w_i , *ie* $\mathbb{E}[z_i e_i] = 0$ and $\mathbb{E}[w_i e_i] = 0$. I also assume homoskedasticity for this term, such that $\mathbb{E}[e_i^2 | z_i, w_i] = \sigma_e^2$ is constant.

Overall, this DGP is close to the usual textbook one but with an additional omitted variable. The Directed Acyclic Graph (DAG) in figure 3 represents the data generating process.

III. 3 ASYMPTOTIC DISTRIBUTIONS OF THE ESTIMATORS

I now derive the asymptotic distributions of the various estimators. For each model, the goal is to show asymptotic normality and to study the evolution of the sampling distribution variances with the value of the parameter of interest, *i.e.*, a measure of the correlation between x and w (γ) in the controlled case, of the IV strength (π_1) in the IV case and of the number of exogenous shocks (σ_z^2 when z is a dummy) in the reduced form case. I assume that the sampling distributions are well approximated by the asymptotic distributions. In order for the variation of one factor not to impact other factors of interest, I consider the variances of the variables ($\sigma_y^2, \sigma_x^2, \sigma_w^2$ and σ_z^2) as

Figure 3 – DAG of the data generating process



Notes: for clarity the error terms are represented in this graph, in beige. Model parameters are noted as edge labels.

fixed but adjust for the variances of the error terms (σ_u^2 and σ_ϵ^2) when varying the values of one of the parameters (γ, δ and π_1). This corresponds to thinking in terms of shares of the variance of x and y explained by “defined” variables (*i.e.*, observed variables and w) *versus* by residuals. Finally note that comparison between cases with and without OVB for different parameter values is only relevant if varying the parameter of interest does not affect the OVB. I thus make comparative statics analyses at bias fixed, *i.e.*, as shown below, for $\gamma\delta = \kappa = cst$.

III. 3.1 NAIVE REGRESSION (OVB)

First, let us study the benchmark against which we are going to compare our causal approaches. Consider the “naive” regression of y on x (with w omitted).

Lemma 2. *Based on the data generating process described in section III. 2, for $\hat{\beta}_{\text{OVB}}$ the OLS estimate of β_1 in the regression of y on x , $\hat{\beta}_{\text{OVB}} \xrightarrow{d} \mathcal{N}(\beta_1 + b_{\text{OVB}}, \sigma_{\text{OVB}}^2)$, with*

$$b_{\text{OVB}} = \frac{\delta\gamma\sigma_w^2}{\sigma_x^2} \quad \text{and} \quad \sigma_{\text{OVB}}^2 = \frac{\sigma_u^2 + \delta^2\sigma_w^2(1 - \rho_{xw}^2)}{n \sigma_x^2}$$

The intuition for the formula of the asymptotic variance has been discussed in the introduction: $\sigma_u^2 + \delta^2\sigma_w^2(1 - \rho_{xw}^2)$ is the part of the variance in y that is not explained by x ($\sigma_{y \perp x}^2$).

Note that, varying the parameter of interest, ρ_{xw} , will change the bias and σ_{OVB}^2 . Since σ_x^2 and σ_w^2 are fixed, reasoning at $b_{\text{OVB}} = cst$ is equivalent to considering that $\gamma\delta = \kappa = const$. Then, noting that $\forall i, u_i = y_i - \beta_0 - \beta_1 x_i - \delta w_i$ and computing its variance, we can rewrite the variance of the

estimator as a function of fixed variances and one or less varying parameter:

$$\sigma_{\text{OVB}}^2 = \frac{\sigma_y^2 - \beta_1^2 \sigma_x^2 - 2\beta_1 \kappa \sigma_w^2 - \kappa^2 \frac{\sigma_w^4}{\sigma_x^2}}{n \sigma_x^2}$$

This expression underlines that, for a given bias, σ_{OVB}^2 does not vary with γ , or equivalently δ , the parameters of interest. Applying lemma III. 1 proves that E_{OVB} does not either.

III. 3.2 CONTROLLED REGRESSION

Next, let us turn to the “ideal” case in which no variable is omitted, *i.e.* we control for the omitted variable w and thus partial out confounders. The model considered accurately represents the DGP. This corresponds to the usual OLS setting with a constant and two regressors that are uncorrelated with the error: y regressed on x and w .

Lemma 3. *Based on the data generating process mentioned previously, for $\hat{\beta}_{\text{CTRL}}$ the OLS estimator of β_1 in the regression of y on x and w , $\hat{\beta}_{\text{CTRL}} \xrightarrow{d} \mathcal{N}(\beta_1, \sigma_{\text{CTRL}}^2)$, with*

$$\sigma_{\text{CTRL}}^2 = \frac{\sigma_u^2}{n \sigma_x^2 (1 - \rho_{xw}^2)}$$

Note that $\sigma_x^2(1 - \rho_{xw}^2)$ is the part of the variance of x that is not explained by w ($\sigma_{x \perp w}^2$) and σ_u^2 the part of the variance of y that is not explained by x nor w ($\sigma_{y \perp x, w}^2$); here too we retrieved a result described in introduction. For a given bias, we then rewrite σ_{CTRL}^2 as a function of fixed variances and one varying parameter, γ :

$$\sigma_{\text{CTRL}}^2 = \frac{\sigma_y^2 - \beta_1^2 \sigma_x^2 - \frac{\kappa^2}{\gamma^2} \sigma_w^2 - 2\beta_1 \kappa \sigma_w^2}{n (\sigma_x^2 - \gamma^2 \sigma_w^2)}$$

Since the numerator and denominator respectively increase and decrease with $|\gamma|$, σ_{CTRL}^2 increases with $|\gamma|$. For a given bias, the more w is correlated with x (and thus roughly the less it is with y since $\delta\gamma = \text{const}$), the larger the variance of the estimator. In addition, we can note that, for a given bias, the variance of the estimator can be arbitrarily large since $\lim_{\gamma^2 \rightarrow \frac{\sigma_x^2}{\sigma_w^2}} \sigma_{\text{CTRL}}^2 = +\infty$.

III. 3.3 INSTRUMENTAL VARIABLES

In the previous section, we considered a case in which we removed variation that included unwanted endogenous variation. We now turn to the IV, a converse situation where we select variation we want, exogenous variation. We estimate the IV model in which we regress y on $x_i = (1, x_i)'$ instrumented by $z_i = (1, z_i)'$. We are thus in a just-identified case and $\hat{\beta}_{\text{2SLS}} = \hat{\beta}_{\text{IV}}$.

Lemma 4. *Based on the data generating process mentioned above, for $\hat{\beta}_{IV}$ the IV estimator of β_1 in the regression of y on x instrumented by z , $\hat{\beta}_{IV} \xrightarrow{d} \mathcal{N}(\beta_1, \sigma_{IV}^2)$, with*

$$\sigma_{IV}^2 = \frac{\sigma_u^2 + \delta^2 \sigma_w^2}{n \sigma_x^2 \rho_{xz}^2}$$

Note that the numerator is $\sigma_{y \perp \hat{x}}^2$, the part of the variance in y that is not explained by \hat{x} , the predicted value of x in the first stage and the denominator is σ_x^2 . For a given bias, noting that $\rho_{xz} = \text{corr}(x, z) = \pi_1 \frac{\sigma_z}{\sigma_x}$ and replacing σ_u^2 , we can rewrite σ_{IV}^2 as a function of fixed variances and one varying parameter, π_1 :

$$\sigma_{IV}^2 = \frac{\sigma_y^2 - \beta_1^2 \sigma_x^2 - 2\beta_1 \kappa \sigma_w^2}{n \pi_1^2 \sigma_z^2}$$

Clearly, the smaller π_1 , the larger σ_{IV}^2 . In addition, $\lim_{\pi_1 \rightarrow 0} \sigma_{IV}^2 = +\infty$.

III. 3.4 REDUCED FORM

Let us now assume that we want to directly estimate the effect of the instrument on the outcome of interest. Plugging equation 4 into equation 2 yields:

$$y_i = (\beta_0 + \beta_1 \pi_0) + (\beta_1 \pi_1) z_i + ((\delta + \beta_1 \gamma) w_i + u_i + \beta_1 e_i)$$

Note that if we directly regress the outcome on the instrument, the resulting estimand will be different from that of the other models. To make them comparable, we could set π_1 to 1 so that an increase of 1 in the instrument causes an increase of β_1 in y . Regardless of whether we make this assumption or not, regressing y on z corresponds to the usual univariate, unbiased case and directly gives the following result:

Lemma 5. *Based on the data generating process mentioned previously, for $\hat{\beta}_{RED}$, the OLS estimator of the reduced form regression of y on z , $\hat{\beta}_{RED} \xrightarrow{d} \mathcal{N}(\beta_1, \sigma_{RED}^2)$, with*

$$\sigma_{RED}^2 = \frac{\sigma_y^2 - \beta_1^2 \pi_1^2 \sigma_z^2}{n \sigma_z^2}$$

Note that the numerator is the part of the variance of y that is not explained by z ($\sigma_{y \perp z}^2$). In addition, it is clear that the smaller σ_z^2 , the larger σ_{RED}^2 . In addition, $\lim_{\sigma_z \rightarrow 0} \sigma_{RED}^2 = +\infty$.

In the binary case, $\sigma_z^2 = p_1(1 - p_1)$ with p_1 the proportion of treated observations, *i.e.*, the proportion of 1 in z . When most observations have the same treatment status, *i.e.*, p_1 close to 0 or 1, σ_z^2 tends to zero and σ_{RED}^2 shoots up. There is not enough variation in the treatment status to precisely identify the effect of interest.

III. 4 EXAGGERATION RATIOS

Combining the results from lemma 2 through 5 regarding the asymptotic distribution of the various estimators with lemma III. 1 stating that exaggeration increases with the variance of a normally distributed estimator yields:

Theorem 1. *For the data generating process described in section III. 2, the exaggeration ratio of the controlled, IV and reduced form estimators, respectively E_{CTRL} , E_{IV} and E_{RED} , are such that:*

- E_{CTRL} increases with the correlation between the omitted variable and the explanatory variable of interest (i.e. $|\gamma|$ or $|\rho_{xw}|$), for a given bias,
- E_{IV} decreases with the strength of the IV (i.e. with $|\pi_1|$ or $|\rho_{xz}|$),
- E_{RED} increases when the number of exogenous shocks decreases in the binary case

Also using the same lemma and the limit properties of the variances described in section III. 2, and since, at fixed bias, E_{OVB} does not vary with the parameters of interest, we get:

Theorem 2. *For the data generating process described in section III. 2, $\forall b_{OVB}$,*

- $\exists \gamma$ s.t. $E_{CTRL} > E_{OVB}$
- $\exists \pi_1$ s.t. $E_{IV} > E_{OVB}$
- $\exists \sigma_z$ s.t. $E_{RED} > E_{OVB}$

For some parameter values, statistically significant estimates can be larger on average when using a convincing causal identification strategy that eliminates the omitted variable bias than when embracing the bias and running a naive biased regression.

IV SIMULATIONS

To study the drivers of exaggeration in concrete settings, I build simulations that reproduce real-world examples from economics of education for RDD, political economy for IV, health economics for exogenous shocks and environmental economics for control and fixed effects approaches. I split the simulations by identification strategy. Real-world settings enable clearly grasping the relationships between the different variables and to set realistic parameter values, based on existing studies. I do not reproduce a specific study but instead calibrate my simulations to emulate an archetypical study from each literature to underline that causal exaggeration is not bound to specific studies. It shows that exaggeration can arise for parameter values consistent with existing studies. To check the representativity of my simulations, I compare the Signal-to-Noise Ratios (SNR) obtained in my simulations to that of studies from the corresponding literature. As underlined by lemma 1, the SNR is a sufficient statistic for the exaggeration ratio; a SNR

consistent with the literature will ensure the representativity of the simulations with respect to the feature of interest, exaggeration. To limit estimation challenges and focus on power aspects, the treatment effects are linear, homogeneous, the models are correctly specified and accurately represent the data generating process, except for the omitted variable.

For each identification strategy, I start by laying out how the method enables retrieving a causal effect. It naturally points to the key parameter through which the confounding/exaggeration trade-off is mediated. I then briefly describe the example setting considered and the simulation assumptions. I finally display the simulation outputs and discuss the implications of the trade-off that are specific to the identification strategy considered. Extensively documented code for each simulation procedure is available on the [project's website](#).

IV. 1 REGRESSION DISCONTINUITY DESIGN

Intuition. A RDD relies on the assumption that for values close to the threshold, treatment assignment is quasi-random. It focuses on observations within a certain bandwidth around this threshold and discards observations further away. The effective sample used for causal identification is thus smaller than the total sample. A smaller bandwidth and effective sample size reduce precision and can create exaggeration. Here, the confounding-exaggeration trade-off is mediated by the size of the bandwidth.

Case-study and simulation procedure. To illustrate this trade-off, I consider a standard application of the sharp RD design from economics of education in which students are assigned to additional lessons based on the score they obtained on a standardized test. [Thistlethwaite and Campbell \(1960\)](#) introduced the concept of RDD using a similar type of quasi-experiment. Students with test scores below a given threshold receive the treatment while those above do not. Since students far above and far below the threshold may differ along unobserved characteristics such as ability, a RDD estimates the effect of the treatment by comparing outcomes of students whose initial test scores are immediately below and above this threshold.

The simulation framework is as follows. If a student i has an initial scores $Qual_i$ below a cutoff C , they must take additional lessons, making the allocation of the treatment T sharp: $T_i = \mathbb{I}[Qual_i < C]$. Both qualification and final test scores are affected by students' unobserved ability w in a non-linear (cubic) way. A high or low ability has a strong positive impact on test scores while an average one does not strongly impact test scores. The qualifying test score of student i is thus: $Qual_i = \mu_q + \gamma f(w_i) + \epsilon_i$, where f a non-linear function (here cubic) and $\epsilon_i \sim \mathcal{N}(0, \sigma_\epsilon^2)$ random noise. Their final test score is: $Final_i = \beta_0 + \beta_1 T_i + \eta Qual_i + \delta f(w_i) + u_i$, where β_1 is the causal parameter of interest. For ability to impact qualifying and final scores

similarly, I set $\delta = \gamma(1 - \eta)$.

These simulations are built and calibrated to emulate a typical study from this literature. I derive parameters of the distribution of grades from statistics from the Department of Education, treatment effect size is based on a meta-analysis of RCTs in economics of education (Kraft 2020). Sample and bandwidth sizes are consistent with an existing study leveraging an RDD to explore a similar question (Jacob and Lefgren 2004). The effect of ability is built to create a large and limited bias in estimates of the treatment effect for large and small bandwidths respectively. Further details on calibration are available on the [project’s website](#). Given these parameters values, I generate 1000 datasets with 60,000 observations. For each dataset, I estimate the treatment effect by regressing the final score on the treatment status and the qualifying score for different bandwidth sizes. The SNRs obtained in the simulations are aligned with SNRs observed in the literature, suggesting a realistic calibration.

Results. Figure 4 displays the results of these simulations. For large bandwidth sizes, the distribution of estimates is far away from the true effect; there is omitted variable bias. As the bandwidth decreases, the identification strategy gets rid of the OVB and the distribution of the estimates becomes centred on the true effect. At the same time, as the bandwidth and the effective sample size decrease, the distribution widens and significant estimates represent a smaller and smaller subset of the distribution, located in the tails of the distribution, creating exaggeration. While the average of all estimates gets close to the true effect as bandwidth size and thus OVB decrease, in this setting the average of statistically significant estimates never gets close to the true effect. For large bandwidths, the omitted variable biases the effect while for small bandwidths, the small effective sample size creates exaggeration issues. The optimal bandwidth literature describes a similar trade-off but with different consequences (Imbens and Kalyanaraman 2012). They consider a bias-precision trade-off, I consider an omitted variable bias-exaggeration bias trade-off. Here, the parameter mediating the trade-off can directly be adjusted in a continuous way by the researchers and the more we reduce one of these two biases, the more we increase the other.

IV. 2 CONTROLLING FOR CONFOUNDERS

Intuition. To identify a causal effect and avoid the risk of confounders, an “ideal” approach would be to partial them out by directly controlling for them. However, as discussed in the introduction and section III, controlling for an additional variable may increase the variance of the estimator if it absorbs more variation in the explanatory variable of interest than in the outcome variable. The same reasoning applies to a cornerstone of causal identification strategies, Fixed-Effects (FEs). If

Figure 4 – Evolution of the Bias with Bandwidth Size in Regression Discontinuity Design, conditional on significance.



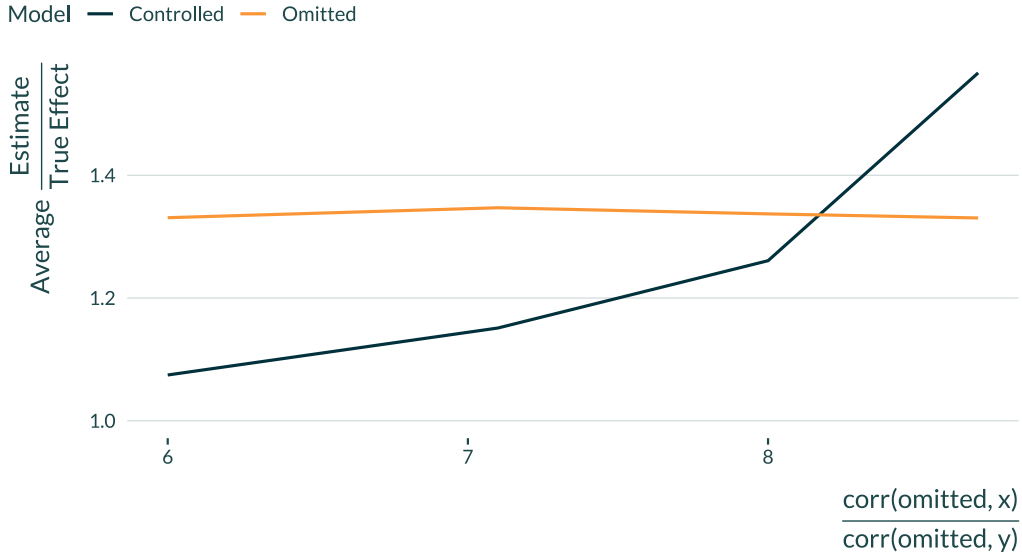
Notes: 1000 simulations. Significativity level: 5%, $N = 60,000$. The brown lines represent the average of significant estimates. The bandwidth size is expressed as the proportion of the total number of observations in the entire sample. Details on the simulation are available at this [link](#).

including FE partials out more of the variation in x than in y , it will increase the variance of the estimator and create exaggeration.

Case-study and simulation procedure. To highlight this trade-off, I consider the extreme case in which we either perfectly control for confounders or do not control for them. I consider a simple setting, with one outcome variable y , one explanatory variable x and an omitted variable w . The data generating process is the same as described in equations 2 and 3. As in section III, I reason at bias fixed and variances of the defined variables fixed, *i.e.* varying the share of the variance of x and y that is explained by w .

Results. Figure 5 displays the results of these simulations. The more the unobserved variable is linked to the explanatory variable of interest as compared to the outcome variable, *i.e.*, the larger the γ/δ ratio, the larger the exaggeration. When this ratio is large, controlling can cause exaggeration to become larger than the OVB plus exaggeration when the variable is omitted.

Figure 5 – Evolution of the Bias with the Correlation of the omitted variable with x and y , conditional on significance.



Notes: The blue line indicates the average bias for estimates from the control model that are statistically significant at the 5%. The orange line represents the bias of statistically significant estimates from the model with the omitted variable. In this simulation, $N = 2,000$. For now, the simulations is calibrated with arbitrary numbers but I will modify this in a later version of the project. Details on the simulation and calibration are available at this [link](#).

IV. 3 INSTRUMENTAL VARIABLES STRATEGY

Intuition. Instrumental variables strategies overcome the issue of unobserved confounding by only considering the exogenous variation in the treatment, *i.e.* the variation that is explained by the instrument. Even when this exogenous fraction of the variation is limited, the instrument can successfully eliminate confounding on average. However, in such cases, the IV estimator will be imprecise and statistical power low. In the case of the IV, the confounding-exaggeration trade-off is mediated by the strength of the instrument considered. The weaker the instrument, the more inflated statistically significant estimates will be.

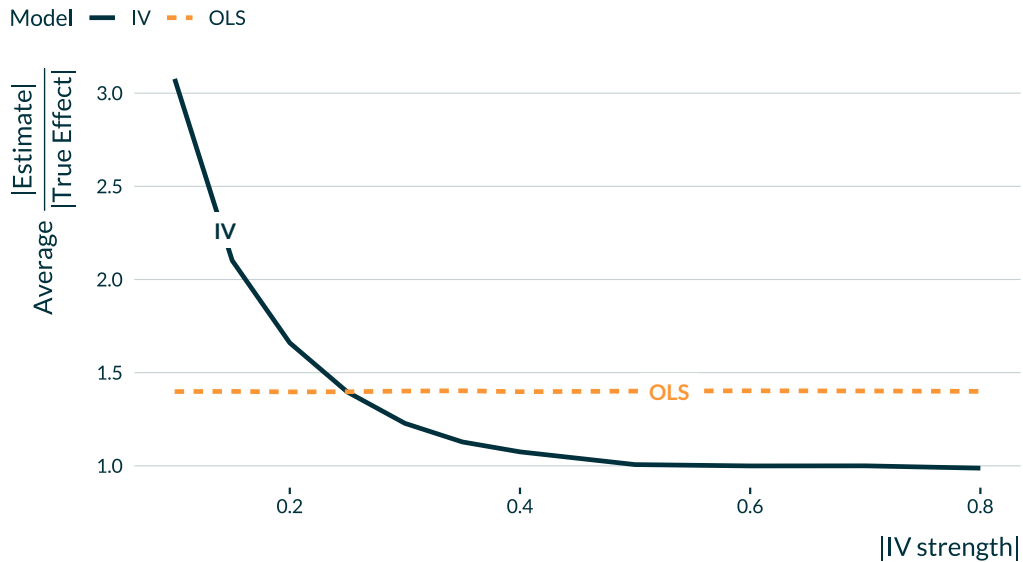
Case-study and simulation procedure. To illustrate this trade-off and its drivers, I consider the example of the impact of voter turnout on election results. To avoid the threat of confounding in this setting, existing studies take advantage of exogenous factors such as rainfall that affect voter turnout⁵. I reproduce such setting and assume that the true data generating process for the republican vote share is such that in location i , $Share_i = \beta_0 + \beta_1 Turnout_i + \delta w_i + u_i$, where w is an unobserved variable and $u \sim \mathcal{N}(0, \sigma_u^2)$ some random noise. The causal parameter of interest is β_1 . In addition, turnout is affected by the amount of rain: $Turnout_i = \pi_0 + \pi_1 Rain_i + \gamma w_i + e_i$,

5. I abstract from potential exclusion restriction violations of this instrument and simulate it as exogenous.

where $Rain_i$ is the amount of rain in location i on the day of the election and e some random noise drawn from $\mathcal{N}(0, \sigma_e^2)$. I refer to π_1 as the strength of the instrumental variable.

To make the simulations realistic, I calibrate them on existing studies. I derive sample size, distribution parameters and effect sizes from a set of studies using similar variables (Gomez et al. 2007, Fujiwara et al. 2016, Cooperman 2017). Details on the calibration choices are available on the [project's website](#). For each value of IV strength considered, I create 1000 datasets of 30,000 observations. I run both a naive OLS and a 2SLS model to estimate the impact of voter turnout on republican vote share. SNR obtained are aligned with SNR observed in this literature.

Figure 6 – Evolution of the Bias of Statistically Significant Estimates Against Strength of the Instrument in the IV Case.



Notes: The blue line indicates the average bias for IV estimates that are statistically significant at the 5%. The orange line represents the bias of statistically significant OLS estimates at the 5% level. The strength of the instrumental variable is expressed as the value of the linear parameter linking rainfall to turnout. In these simulations, $N = 30,000$. Details on the simulation are available at this [link](#).

Results. Figure 6 displays, for different IV strengths, the average of statistically significant estimates scaled by the true effect size for both the IV and the naive regression model. When the instrument is strong, the IV will recover the true effect, contrarily to the naive regression model. Yet, when the IV strength decreases, the exaggeration of statistically significant estimates skyrockets. Even if the intensity of the omitted variable bias is large, for limited IV strengths, the exaggeration ratio can become larger than the omitted variable bias. When the only available instrument is weak, using the naive regression model would, on average, produce statistically significant estimates that are closer to the true effect size than the IV. Of interest for applied

research, a large F -statistic does not necessarily attenuate this problem. This result is aligned with limitations around the use of first-stage F -statistics underlined in [Young \(2022\)](#).

IV. 4 EXOGENOUS SHOCKS

Intuition. Taking advantage of exogenous variation in the treatment status caused by exogenous shocks or events can also enable avoiding confounding. In many settings, while the number of observations may be large, the number of events, their duration or the proportion of individuals affected might be limited. As a consequence, the number of (un)treated observations can be small and the variation available to identify the treatment limited. As extensively discussed in the randomized controlled trial literature, statistical power is maximized when the proportion of treated observations is equal to the proportion of untreated ones and drops when one of these proportions gets close to 0. In studies using discrete exogenous shocks, a confounding-exaggeration trade-off is thus mediated by the number of treated observations.

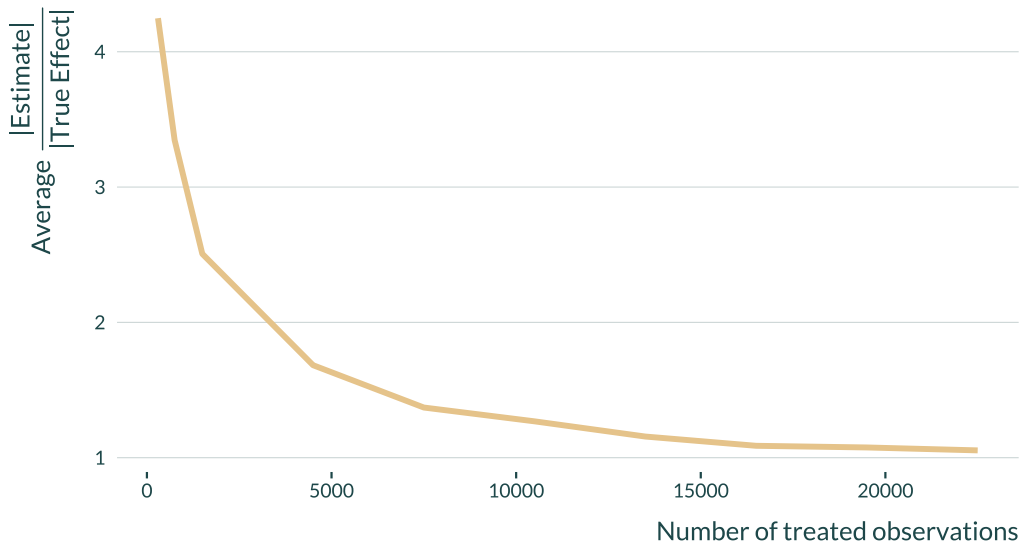
Case-study and simulation procedure. To illustrate this trade-off, I simulate a study of the impact of air pollution reduction on newborn weight of babies. To avoid confounding, one can exploit exogenous shocks to air pollution such as plant closures, creation of a low emission zone or of an urban toll ([Currie et al. 2015](#), [Neidell 2017](#)). I simulate such an analysis, at the zip code and monthly levels and focus on the example of toxic plant closures. I consider that the average birth weight in zip code z at time period t , bw_{zt} , depends on a zip code fixed effect ζ_z , a time fixed effect τ_t , and the treatment status T_{zt} , equal to one if a plant is closed in this period and 0 otherwise. The average birth weight in zip code z at time t is defined as follows: $bw_{zt} = \beta_0 + \beta_1 T_{zt} + \zeta_z + \tau_t + u_{zt}$. To further simplify the identification of the effect, I assume a non-staggered treatment allocation and constant and homogenous effects. I only vary the proportion of zip codes affected by toxic plant closings, keeping the length of the closures fixed.

I derive parameters values from studies from the literature ([Currie et al. 2015](#), [Neidell 2017](#)). The simulations mimic the design of such studies, considering a similar number of observations (150,000), distributions of variables, treatment allocation procedure and treatment effect sizes. I generate datasets with an increasing number of treated observations, varying the proportion of treated units and estimate the correct two-way fixed effects model. The simulation procedure is described in details on the [project's website](#).

Results. Figure 7 displays the results of these simulations. Even though the actual sample size is extremely large in this example, if the number of treated observations is small, as it can be the case in this literature, exaggeration can be substantial. A very large number of observations does

not necessarily shield us from exaggeration.

Figure 7 – Evolution of Bias With the Number of Treated Observations, for Statistically Significant Estimates, in the Exogenous Shocks Case



Notes: Significance level: 5%. In this simulation, $N = 150,000$. 1000 iterations for each number of treated observations considered. Details on the simulation are available at this [link](#).

V NAVIGATING THE TRADE-OFF

In the previous sections, I argued that that using causal identification strategies induces a trade-off between avoiding confounding and exaggerating true effects. How can we, as applied researchers using observational data, arbitrate it? Since key pieces of information such as the true effect and the effect of omitted variables are inherently unknown, we cannot directly compute the biases caused by confounders and exaggeration. In this section, I examine how we can, however, get a sense of threats from both sides of the trade-off and probe its main driver, the variation used for identification. I then discuss how changing attitudes towards statistical significance and replicating studies could limit the exaggeration issue.

V. 1 GAUGING OMITTED VARIABLE BIAS

On one side of the trade-off lies the widely discussed bias caused by confounders. Although it is in essence impossible to measure, tools such as sensitivity analyses are available to gauge its magnitude (Rosenbaum 2002, Middleton et al. 2016, Oster 2019, Cinelli and Hazlett 2020). For instance, the method developed in Cinelli and Hazlett (2020) enables assessing how strong

confounders would have to be to change the estimate of the treatment effect beyond a given level we are interested in. It offers bounds for the strength of the association between the treatment and potential omitted variables by weighting it against the measured association between the treatment and observed covariates. A typical conclusion from such an analysis would be: “omitted variables would have to explain as much residual variance of the outcome and the treatment as the observed covariate x (age for instance) to bring down the estimate to a value of β_l ”. In addition, the authors implement graphical tools to facilitate this comparison. I suggest to use such quantitative bias analyses to evaluate the restrictiveness of the causal approach required to limit the threat of unobserved confounding to acceptable levels. In settings where bias caused by confounders is likely to be low, the

V. 2 EVALUATING RISKS OF EXAGGERATION

On the other side of the trade-off lies the exaggeration emerging when statistical power is low. As OVB, exaggeration and statistical power are in essence impossible to measure as their computation depends on the true effect which is always unknown. Yet, power calculations can help assess them by making hypothesizes on the magnitude of the true effect. In randomized controlled trials, such computations are not only an established practice but a requirement (Duflo et al. 2007, ?, Athey and Imbens 2016). They are, however, rarely reported in non-experimental studies. Yet, taking publication bias and the threat of exaggeration into account highlights the necessity of running power calculations in non-experimental studies as well. A low power or a relatively large variance not only makes it more difficult to detect an effect or to draw clear conclusions about its magnitude when detected but it can also create a bias. To avoid this bias, I advocate to make power more central to non-experimental analyses. Currently, in causal inference textbooks, very few pages are devoted to statistical power in non-experimental studies (Angrist and Pischke 2009; 2014, Imbens and Rubin 2015, Cunningham 2021). To the best of my knowledge, only two textbooks discuss the matter in depth (Shadish et al. 2002, Huntington-Klein 2021). Results from power and exaggeration calculations would not only be highly informative but could also be reported very concisely in the robustness section of articles.

V. 2.1 PROSPECTIVE POWER CALCULATIONS

To evaluate the statistical power of a study, the risk of exaggeration and identifying the factors driving it, one can first simulate the design of the study (Hill 2011, Gelman 2020, Black et al. 2021). Simulating a data generating process from scratch requires thinking about the distribution of the variables, about their relationships and can also help underline the variation used for identification. I implemented such Monte Carlo simulations in Section IV. The replication material and R code

I provide can be used as an example to implement simulations for most causal identification strategies, based on data generated from scratch. In situations where the relationships among covariates are too complex to emulate, one can also start from an existing dataset and add a known treatment effect. I implemented real-data simulations in a companion paper and describe their implementation in its [replication material](#) (Bagilet 2023).

V. 2.2 RETROSPECTIVE POWER CALCULATIONS

Running post-analysis power calculations can also help getting a sense of the statistical power associated with a research design. Such *retrospective* calculations allow evaluating whether the design of the study would produce accurate and uninflated statistically significant estimates if the true effect was in fact smaller than the observed estimate (Gelman and Carlin 2014, Ioannidis et al. 2017, ?).

I illustrate how a retrospective analysis works by taking the example of Card (1993) on the relationship between human capital and income. He finds that an additional year of education, instrumented by the distance of growing up near a four-year college, causes a 13.2% average increase in wage. The associated standard error is 5.5%. Is there a risk of exaggeration with this design? Since, as noted by the author himself, the estimate is very imprecise we could expect so. If the existing literature suggests that such effects are likely to be close close to a 10% increase in wage, we may wonder if the design in Card (1993) would allow detecting such an effect. We can thus compute the statistical power of the study under the hypothesis of a true effect size of 10% and for a precision of 5.5% equal to that obtained in Card (1993).⁶ Statistically significant estimates (at the 5% level) would on average be roughly equal to 15%, therefore overestimating the true effect by a factor of 1.5. Statistical power, the proportion of estimates that are significant, would only be 44%. Conditional on a 10% true effect size being a reasonable assumption, this study would be under-powered and exaggeration substantial.

The usefulness of any retrospective power analysis lies on the assumption made regarding the true effect size. To identify a range of plausible effect sizes one can rely on results from meta-analyses or from existing studies that have a credible design (*e.g.*, a large randomized controlled trial).⁷ When such information is not available, power calculations can be ran for a range of smaller but credible effect sizes. Such credible effect sizes can be derived from theoretical findings. It is also possible to evaluate whether the design of our study would be sufficient to detect smaller effects than the point estimate obtained.

6. Timm et al. (2019) and Linden (2019) offer R and Stata packages that enable easily running these calculations through an extremely short command: `retrodesign(10, 5.5)`.

7. Note that when such meta-analyses are available, one can use a Bayesian procedure to shrink statistically significant estimates based on the corpus of estimates from prior studies Zwet and Gelman (2021), Zwet and Cator (2021), Zwet et al. (2021) .

V. 2.3 IMPRECISION MATTERS AFTER OBTAINING A SIGNIFICANT ESTIMATE

In non-experimental studies, estimator variance is often important to the extent that a large variance may lead to a failure to reject the null of no effect when it is incorrect. Variance is paramount until a statistically significant estimate is obtained. Yet, exaggeration underlines that variance matters, even once a significant estimate has been obtained.

Obtaining a statistically significant estimate from an imprecise estimator should not necessarily be interpreted as a sign of “success” in getting significance despite a large confidence interval. It could instead be a warning that this estimate may come from the tails of the distribution and would thus inaccurately represent the true effect. Conditional on having obtained a statistically significant estimate, a limited precision can hide a bias: exaggeration. This invites to revisit the well-known bias-variance trade-off: a larger variance can also lead to a larger bias, even in (conditional) expectation. When combined with the existing statistical significance filter, the bias-variance trade-off is in fact a bias-bias trade-off. This paper thus invites to pay attention to the implications of our design and modelling choices on the variance of our estimators, even if a large variance did not prevent us from obtaining a statistically significant estimate.

V. 3 THE VARIATION USED FOR IDENTIFICATION

Causal inference strategies only leverage a subset of the variation to avoid confounders. However, when this subset is too small, exaggeration arises. Identifying this variation (and the observations) actually used for estimation can help navigate the confounding-exaggeration trade-off. The measure I outline here both leverages the interpretation of causal inference methods as control approaches and builds on a procedure developed in [Aronow and Samii \(2016\)](#) for a different purpose: evaluating the external validity of standard regressions.

[Aronow and Samii \(2016\)](#) essentially interprets the estimate of the coefficient of the treatment of interest in a simple linear non-causal regression as a weighted average of individual treatment effects. The weight w_i of individual i is simply the squared difference between its treatment status T_i and the value of this treatment status as predicted by the other covariates X : $w_i = (T_i - \mathbb{E}[T_i|X_i])^2$. If treatment effects are heterogenous, the weighting may lead some observations to be disproportionally represented in the average effect of the treatment. In that case, the average of treatment is only representative of a subset of the individual treatments, leading to external validity issues.

The parallel with my setting directly follows from this interpretation regardless of whether the treatment is heterogenous or not: observations whose treatment status is well explained by covariates do not actually contribute to the estimation of the treatment effect. This may lead to a small *effective* sample size and to exaggeration. In the control approach to causal inference

strategies, the more variation in the treatment is absorbed when “controlling” for confoundings, the smaller the effective sample, potentially leading to exaggeration. [Aronow and Samii \(2016\)](#) leverages the representativity of the effective sample for fear of external validity issues. I focus on its size for fear of exaggeration.

It might then seem compelling to define this effective sample by proposing a weight value under which the associated observation does not actually contribute to identification. Yet, considering the specificity of each analysis and that exaggeration depends on several factors, including the true effect size, I instead suggest to visualize the individual weights.⁸ It allows getting a sense of where the variation comes from and which are the observations that actually contribute to the estimation. Since applied economic analyses often rely on panel data, I propose to use a heatmap as a base for visualization, with time on the x -axis and individuals on the y -axis. If the data is geographical, one can directly plot the weights on a map.

V. 4 ATTITUDE TOWARDS STATISTICAL SIGNIFICANCE AND REPLICATION

Exaggeration only arises in the presence of publication bias. As shown in the simulations, if estimates were not filtered by their statistical significance, even under-powered studies would on average recover the true effect, as long as the estimator is unbiased. The exaggeration issue could therefore be addressed by tackling publication bias.

To identify broader pathways to eliminate this filtering of significant results, it is first helpful to discuss the processes that lead to statistically significant results when power and thus the probability of obtaining a significant estimate is low. In such situations, they can be obtained either by “chance” or as an outcome of the garden of forking paths ([Simmons et al. 2011](#), [Gelman and Loken 2013](#), [Kasy 2021](#)). Forks appear at various stages along the path of research, for instance in data preparation, regarding the inclusion of a given cofntrol variable or later, regarding whether to carry on with a research that yields non-significant results. Due to the structural flaw that favors significance, the path followed may be more likely to lead to a statistically significant result. These choices are most often not the result of bad researcher practices but instead a product of a structure that portrays significant results as the end goal of research.

The issue being structural, system level changes in scientific practices could also alleviate exaggeration and the trade-off described in this paper. First, many researchers advocate abandoning statistical significance as a measure of a study’s quality ([McShane et al. 2019](#)). To be effective, this change should be paired with an effort to replicate studies ([Christensen and Miguel 2018](#)).

8. In future developments of this project, I will however develop a measure of this effective sample size. I may also need to modify the weights formula in order to account for the variation in y that is absorbed when controlling.

Replications, even of low powered studies, would eventually enable building the actual distribution of the causal estimand of interest. Meta-analyzes would then reduce the uncertainty around the true value of the causal estimand by pooling estimates (Hernán 2021). Finally, the inflation of statistically significant estimates could be limited by interpreting confidence intervals and not point estimates and thus considering these intervals as compatibility intervals (Shadish et al. 2002, Amrhein et al. 2019, Romer 2020). The width of such intervals gives a range of effect sizes compatible with the data. Confidence intervals will be wide in under-powered studies signaling that point estimates should not be taken at face value, even if statistically significant.

VI CONCLUSION

The economic literature suffers from an extensive lack of statistical power (Ioannidis et al. 2017) and strongly favors statistically significant findings (Rosenthal 1979, Andrews and Kasy 2019, Abadie 2020, Brodeur et al. 2020, for instance). In such situations, estimates published from underpowered studies exaggerate true effect sizes, even when the estimators are “unbiased” in the usual sense of $\mathbb{E}[\hat{\beta}] = \beta$ (Gelman and Tuerlinckx 2000, Ioannidis 2008, Gelman and Carlin 2014). It is therefore not surprising that many estimates published in economics have been shown to be considerably exaggerated (Camerer et al. 2016, Ioannidis et al. 2017), despite the extensive use of convincing causal inference methods. However, determinants for these exaggeration and power issues have remained understudied. I argue that exaggeration is exacerbated by the foundational component of causal inference: the fact that it only leverages subsets of the variation. Although causal methods enable avoiding confounding, they also reduce statistical power and thus increase the risk of exaggeration. The same aspect that makes these methods credible can create another type of bias. A systematic reporting of statistical power calculations and analysis of the variation actually used for identification could help to avoid falling into this exaggeration trap.

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A MATHEMATICAL PROOFS

I. 1 VARIATION OF THE EXAGGERATION RATIO (LEMMA III. 1)

Proof. Lu et al. (2019) and Zwet and Cator (2021) showed this in the case of $b = 0$. To extend it to the biased case, consider $E_b = \frac{\mathbb{E}[|\hat{\beta}_b| \mid |\beta_1, \sigma| \hat{\beta}_b| > z_\alpha \sigma]}{|\beta_1|}$ the exaggeration ratio of interest. Note that, since $\hat{\beta}_b$ is an unbiased estimator of $\beta_1 + b$, $\tilde{E}_b = \frac{\mathbb{E}[|\hat{\beta}_b| \mid |\beta_1, \sigma| \hat{\beta}_b| > z_\alpha \sigma]}{|\beta_1 + b|}$ has the properties described in the lemma. Now, considering that $E_b = \left| \frac{\beta_1 + b}{\beta_1} \right| \tilde{E}_b$ proves the properties when β_1 and b have the same sign. \square

I. 2 ASYMPTOTIC DISTRIBUTION OF $\hat{\beta}_{\text{OVB}}$ (LEMMA 2)

For readability, let us introduce the usual vector notation such that for instance $y = (y_1, \dots, y_n)'$ and set $\beta = (\beta_0, \beta_1)'$ and $x_i = (1, x_i)'$. I also use capital letters to denote matrices (for instance $X = (x_1', \dots, x_n')')$.

Proof. Since, we do not observe w , we consider the projection of y on X only:

$$y = X\beta_{\text{OVB}} + u_{\text{OVB}} \quad (5)$$

where by definition of the projection, $\mathbb{E}[X'u_{\text{OVB}}] = 0$.

We first compute the bias of the estimator. From equation 5 we get:

$$\begin{aligned} X'y &= X'X\beta_{\text{OVB}} + X'u_{\text{OVB}} \\ \Rightarrow \mathbb{E}[X'y] &= \underbrace{\mathbb{E}[X'X]}_{\text{pos. def.}} \beta_{\text{OVB}} + \underbrace{\mathbb{E}[X'u_{\text{OVB}}]}_0 \\ \Leftrightarrow \beta_{\text{OVB}} &= \mathbb{E}[X'X]^{-1} \mathbb{E}[X'(X\beta + \delta w + u)] \quad \text{cf eq. 2} \\ \Leftrightarrow \beta_{\text{OVB}} &= \beta + \mathbb{E}[X'X]^{-1} \mathbb{E}[X'w]\delta \end{aligned} \quad (6)$$

We then compute the asymptotic distribution. We can write:

$$\sqrt{n}(\hat{\beta}_{\text{OVB}} - \beta_{\text{OVB}}) = \left(\frac{1}{n} \sum_{i=1}^n x_i x_i' \right)^{-1} \sqrt{n} \left(\frac{1}{n} \sum_{i=1}^n x_i u_{\text{OVB},i} \right)$$

Applying the Weak Law of Large Numbers (WLLN), the Central Limit Theorem (CLT) and Slutsky's theorem yields:

$$\sqrt{n}(\hat{\beta}_{\text{OVB}} - \beta_{\text{OVB}}) \xrightarrow{d} \mathcal{N}(0, \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} \mathbb{E}[\mathbf{x}_i \mathbf{x}_i' u_{\text{OVB},i}^2] \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1}) \quad (7)$$

We are interested in the second component of $\hat{\beta}_{\text{OVB}}$. To retrieve it we need to compute $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1}$, $\mathbb{E}[\mathbf{x}_i w_i]$ and $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i' u_{\text{OVB},i}^2]$.

$$\begin{aligned} \mathbb{E}[\mathbf{x}_i \mathbf{x}_i'] &= \mathbb{E} \begin{bmatrix} 1 & x_i \\ x_i & x_i^2 \end{bmatrix} = \begin{bmatrix} 1 & \mu_x \\ \mu_x & \sigma_x^2 + \mu_x^2 \end{bmatrix} \Rightarrow \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} = \frac{1}{\sigma_x^2} \begin{bmatrix} \sigma_x^2 + \mu_x^2 & -\mu_x \\ -\mu_x & 1 \end{bmatrix} \\ \mathbb{E}[\mathbf{x}_i w_i] &= \mathbb{E} \begin{bmatrix} w_i \\ x_i w_i \end{bmatrix} = \begin{bmatrix} 0 \\ \underbrace{\mathbb{E}[x_i] \mathbb{E}[w_i] + \text{cov}(x_i, w_i)}_0 \end{bmatrix} = \begin{bmatrix} 0 \\ \underbrace{\gamma \text{var}(w_i)}_{\sigma_w^2} + \underbrace{\text{cov}(\epsilon_i, w_i)}_0 \end{bmatrix} = \begin{bmatrix} 0 \\ \gamma \sigma_w^2 \end{bmatrix} \\ &\Rightarrow \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} \mathbb{E}[\mathbf{x}_i w_i] = \frac{\gamma \sigma_w^2}{\sigma_x^2} \begin{bmatrix} -\mu_x \\ 1 \end{bmatrix} \end{aligned} \quad (8)$$

Note that $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i' u_{\text{OVB},i}^2] \stackrel{\text{LIE}}{=} \mathbb{E}[\mathbf{x}_i \mathbf{x}_i' \mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i]]$. We thus first compute $\mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i]$, noting that:

$$\begin{aligned} u_{\text{OVB},i} &= y_i - \mathbf{x}_i' \beta_{\text{OVB}} \\ &= \delta w_i + u_i + \mathbf{x}_i' (\beta - \beta_{\text{OVB}}) \\ &= \delta w_i + u_i - \underbrace{\mathbf{x}_i' \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} \mathbb{E}[\mathbf{x}_i w_i]}_{\text{projection of } w_i \text{ on } x_i} \delta \\ &= u_i + \delta \underbrace{\left(w_i - \frac{\gamma \sigma_w^2}{\sigma_x^2} (x_i - \mu_x) \right)}_{\text{part of } w_i \text{ orthogonal to } x_i} \\ &= u_i + \delta w_i^\perp \end{aligned} \quad \text{where } w_i^\perp = w_i - \frac{\gamma \sigma_w^2}{\sigma_x^2} (x_i - \mu_x)$$

And thus,

$$\begin{aligned}
\mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i] &= \mathbb{E}[(u_i + \delta w_i^\perp)^2 | x_i] \\
&= \mathbb{E}[u_i^2 | x_i] + 2\delta \mathbb{E}[u_i w_i^\perp | x_i] + \delta^2 \mathbb{E}[(w_i^\perp)^2 | x_i] \\
&= \sigma_u^2 + 2\delta \left(\mathbb{E}[u_i w_i | x_i] - \frac{\gamma \sigma_w^2}{\sigma_x^2} (x_i - \mu_x) \underbrace{\mathbb{E}[u_i | x_i]}_0 \right) + \delta^2 \mathbb{E}[(w_i^\perp)^2 | x_i] \\
&\stackrel{\text{LIE}}{=} \sigma_u^2 + 2\delta \mathbb{E}[w_i \underbrace{\mathbb{E}[u_i | x_i, w_i]}_0 | x_i] + \delta^2 \mathbb{E}[(w_i^\perp)^2 | x_i] \\
&= \sigma_u^2 + \delta^2 \mathbb{E}[(w_i^\perp)^2 | x_i]
\end{aligned}$$

Notice that, by the law of total variance, $\mathbb{E}[(w_i^\perp)^2 | x_i] = \text{Var}(w_i^\perp | x_i) + \mathbb{E}[w_i^\perp | x_i]^2$. Now, since w_i^\perp is the component of w_i that is orthogonal to x_i and by the projection interpretation of the conditional variance, $\mathbb{E}[w_i^\perp | x_i] = 0$. And thus, since by assumption $\text{Var}(w_i^\perp | x_i) = \text{Var}(w_i^\perp)$,

$$\begin{aligned}
\mathbb{E}[(w_i^\perp)^2 | x_i] &= \text{Var}(w_i^\perp | x_i) \\
&= \text{Var}(w_i^\perp) \\
&= \mathbb{E}[(w_i^\perp)^2] - \mathbb{E}[w_i^\perp]^2 \\
&= \mathbb{E} \left[\left(w_i - \frac{\gamma \sigma_w^2}{\sigma_x^2} (x_i - \mu_x) \right)^2 \right] - \left(\underbrace{\mathbb{E}[w_i]}_0 + \frac{\gamma \sigma_w^2}{\sigma_x^2} \underbrace{\mathbb{E}[x_i - \mu_x]}_0 \right)^2 \\
&= \underbrace{\mathbb{E}[w_i^2]}_{\sigma_w^2} - 2 \frac{\gamma \sigma_w^2}{\sigma_x^2} \left(\underbrace{\mathbb{E}[x_i w_i]}_{\gamma \sigma_w^2} - \mu_x \underbrace{\mathbb{E}[w_i]}_0 \right) + \frac{\gamma^2 \sigma_w^4}{\sigma_x^4} \underbrace{\mathbb{E}[(x_i - \mu_x)^2]}_{\sigma_x^2} \\
&= \sigma_w^2 \left(1 - \frac{\gamma^2 \sigma_w^2}{\sigma_x^2} \right)
\end{aligned}$$

Note that this variance is well defined (positive) only if $\sigma_x^2 \geq \gamma^2 \sigma_w^2$. Under this condition,

$$\mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i] = \sigma_u^2 + \delta^2 \sigma_w^2 \left(1 - \frac{\gamma^2 \sigma_w^2}{\sigma_x^2} \right) \quad (9)$$

Thus, under our set of assumptions, $\mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i]$ does not depend on x_i and $\mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i] = \mathbb{E}[u_{\text{OVB},i}^2]$. We denote this quantity $\sigma_{u_{\text{OVB}}}^2$.

We can now compute the variance of the estimator $\hat{\beta}_{\text{OVB}}$, noting that $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i' u_{\text{OVB},i}^2] = \mathbb{E}[\mathbf{x}_i \mathbf{x}_i' \mathbb{E}[u_{\text{OVB},i}^2 | \mathbf{x}_i]] = \mathbb{E}[\mathbf{x}_i \mathbf{x}_i' \sigma_{u_{\text{OVB}}}^2] = \sigma_{u_{\text{OVB}}}^2 \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']$. And thus $\mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} \mathbb{E}[\mathbf{x}_i \mathbf{x}_i' u_{\text{OVB},i}^2] \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']^{-1} = \sigma_{u_{\text{OVB}}}^2 \mathbb{E}[\mathbf{x}_i \mathbf{x}_i']$.

Plugin this and equation 8 into equation 7, we get, for $\hat{\beta}_{\text{OVB}}$, the second component of $\hat{\beta}_{\text{OVB}}$:

$$\hat{\beta}_{\text{OVB}} \xrightarrow{d} \mathcal{N} \left(\beta_1 + \frac{\delta \gamma \sigma_w^2}{\sigma_x^2}, \frac{\sigma_u^2 + \delta^2 \sigma_w^2 \left(1 - \frac{\gamma^2 \sigma_w^2}{\sigma_x^2}\right)}{n \sigma_x^2} \right)$$

Then, noting that $\rho_{xw} = \text{corr}(x, w) = \frac{\text{cov}(\mu_x + \gamma w + \epsilon, w)}{\sigma_x \sigma_w} = \frac{\gamma \sigma_w}{\sigma_x}$, we have:

$$\sigma_{\text{OVB}}^2 = \text{avar} \left(\hat{\beta}_{\text{OVB}} \right) = \frac{\sigma_u^2 + \delta^2 \sigma_w^2 (1 - \rho_{xw}^2)}{n \sigma_x^2}$$

□

I. 3 ASYMPTOTIC DISTRIBUTION OF $\hat{\beta}_{\text{CTRL}}$ (LEMMA 3)

Proof. The proof is the well know proof of the asymptotic distribution of the OLS. I simply compute $\mathbb{E}[x_{w,i} x'_{w,i}]^{-1}$ to retrieve the variance of the parameter of interest β_{CTRL} . We know that we have:

$$\sqrt{n}(\hat{\beta}_{\text{CTRL}} - \beta_{\text{CTRL}}) \xrightarrow{d} \mathcal{N}(0, \mathbb{E}[x_{w,i} x'_{w,i}]^{-1} \sigma_u^2)$$

We are interested in the second component of $\hat{\beta}_{\text{CTRL}}$. To retrieve it we need to compute $\mathbb{E}[x_{w,i} x'_{w,i}]^{-1}$.

$$\mathbb{E}[x_{w,i} x'_{w,i}] = \mathbb{E} \begin{bmatrix} 1 & x_i & w \\ x_i & x_i^2 & x_i w_i \\ w_i & x_i w_i & w_i^2 \end{bmatrix} = \begin{bmatrix} 1 & \mu_x & 0 \\ \mu_x & \sigma_x^2 + \mu_x^2 & \gamma \sigma_w^2 \\ 0 & \gamma \sigma_w^2 & \sigma_w^2 \end{bmatrix}$$

Note that we have $\mathbb{E}[x_i w_i] = \mathbb{E}[x_i] \underbrace{\mathbb{E}[w_i]}_0 + \underbrace{\text{cov}(x_i, w_i)}_{\sigma_w^2} = \underbrace{\gamma \text{var}(w_i)}_{\sigma_w^2} + \underbrace{\text{cov}(\epsilon_i, w_i)}_0 = \gamma \sigma_w^2$.

Now, $\mathbb{E}[x_{w,i} x'_{w,i}]^{-1} = \frac{1}{\det(\mathbb{E}[x_{w,i} x'_{w,i}])} {}^t\text{C}$ with C the comatrix of $\mathbb{E}[x_{w,i} x'_{w,i}]$. We have:

$$\det(\mathbb{E}[x_{w,i} x'_{w,i}]) = (\sigma_x^2 + \mu_x^2) \sigma_w^2 - \sigma_w^2 \mu_x^2 - \gamma^2 \sigma_w^4 = \sigma_w^2 (\sigma_x^2 - \gamma^2 \sigma_w^2)$$

and the “central” component of C, σ_w^2 . Thus the central component of interest of $\mathbb{E}[x_{w,i} x'_{w,i}]^{-1}$ is $\frac{1}{\sigma_x^2 - \gamma^2 \sigma_w^2}$. Therefore, for $\hat{\beta}_{\text{CTRL}}$, the second component of $\hat{\beta}_{\text{CTRL}}$, we have:

$$\hat{\beta}_{\text{CTRL}} \xrightarrow{d} \mathcal{N} \left(\beta_1, \frac{\sigma_u^2}{n (\sigma_x^2 - \gamma^2 \sigma_w^2)} \right) \quad (10)$$

Then, noting that $\rho_{xw} = \text{corr}(x, w) = \frac{\text{cov}(\mu_x + \gamma w + \epsilon, w)}{\sigma_x \sigma_w} = \frac{\gamma \sigma_w}{\sigma_x}$, we have:

$$\sigma_{\text{CTRL}}^2 = \frac{\sigma_u^2}{n \sigma_x^2 (1 - \rho_{xw}^2)}$$

□

I. 4 ASYMPTOTIC DISTRIBUTION OF $\hat{\beta}_{\text{IV}}$ (LEMMA 4)

Proof. Since $u_{\text{IV}} = u_{\text{OVB}} = \delta w + u$, we have $\sigma_{u_{\text{IV}}}^2 = \sigma_u^2 + \delta^2 \sigma_w^2$. Thus, the usual asymptotic distribution of the IV gives:

$$\sqrt{n}(\hat{\beta}_{\text{IV}} - \beta) \xrightarrow{d} \mathcal{N}\left(0, (\sigma_u^2 + \delta^2 \sigma_w^2) \mathbb{E}[z_i x_i']^{-1} \mathbb{E}[z_i z_i'] (\mathbb{E}[z_i x_i']^{-1})'\right)$$

We are interested in the second component of $\hat{\beta}_{\text{IV}}$. To retrieve it we need to compute $\mathbb{E}[z_i z_i']$, $\mathbb{E}[x_i x_i']^{-1}$ and its transpose.

$$\mathbb{E}[z_i z_i'] = \mathbb{E} \begin{bmatrix} 1 & z_i \\ z_i & z_i^2 \end{bmatrix} = \begin{bmatrix} 1 & \mu_z \\ \mu_z & \sigma_z^2 + \mu_z^2 \end{bmatrix}$$

$$\begin{aligned} \mathbb{E}[z_i x_i'] &= \begin{bmatrix} 1 & \mathbb{E}[x_i] \\ \mathbb{E}[z_i] & \mathbb{E}[z_i x_i] \end{bmatrix} = \begin{bmatrix} 1 & \pi_0 + \pi_1 \mathbb{E}[z_i] + \gamma \underbrace{\mathbb{E}[w_i]}_0 + \underbrace{\mathbb{E}[e_i]}_0 \\ \mu_z & \pi_0 \mathbb{E}[z_i] + \pi_1 \mathbb{E}[z_i^2] + \gamma \underbrace{\mathbb{E}[z_i w_i]}_0 + \underbrace{\mathbb{E}[z_i e_i]}_0 \end{bmatrix} = \begin{bmatrix} 1 & \pi_0 + \pi_1 \mu_z \\ \mu_z & \pi_0 \mu_z + \pi_1 (\sigma_z^2 + \mu_z^2) \end{bmatrix} \\ \Rightarrow \quad \mathbb{E}[z_i x_i']^{-1} &= \frac{1}{\pi_1 \sigma_z^2} \begin{bmatrix} \pi_0 \mu_z + \pi_1 (\sigma_z^2 + \mu_z^2) & -\pi_0 - \pi_1 \mu_z \\ -\mu_z & 1 \end{bmatrix} \end{aligned}$$

Thus,

$$\mathbb{E}[z_i x_i']^{-1} \mathbb{E}[z_i z_i'] (\mathbb{E}[z_i x_i']^{-1})' = \frac{1}{\pi_1 \sigma_z^2} \begin{bmatrix} 2\pi_0 \mu_z + \pi_1 (\sigma_z^2 + \mu_z^2) + \frac{\pi_0^2}{\pi_1} & -\mu_z - \frac{\pi_0}{\pi_1} \\ -\mu_z - \frac{\pi_0}{\pi_1} & \frac{1}{\pi_1} \end{bmatrix}$$

And so, for $\hat{\beta}_{\text{IV}}$, the second component of $\hat{\beta}_{\text{IV}}$, we have:

$$\sqrt{n}(\hat{\beta}_{\text{IV}} - \beta_1) \xrightarrow{d} \mathcal{N}\left(0, \frac{\sigma_u^2 + \delta^2 \sigma_w^2}{n \pi_1^2 \sigma_z^2}\right) \quad (11)$$

Now, since $\rho_{xz} = \text{corr}(x_i, z_i) = \frac{\text{cov}(\pi_0 + \pi_1 z_i + \gamma w_i + e_i, z_i)}{\sigma_x \sigma_z} = \pi_1 \frac{\sigma_z}{\sigma_x}$,

$$\sqrt{n} \left(\hat{\beta}_{\text{IV}} - \beta_1 \right) \xrightarrow{d} \mathcal{N} \left(0, \frac{\sigma_u^2 + \delta^2 \sigma_w^2}{\sigma_x^2 \rho_{xz}^2} \right)$$

□

I. 5 ASYMPTOTIC DISTRIBUTION OF $\hat{\beta}_{\text{RED}}$ (LEMMA 5)

Proof. The proof is straightforward: this is the usual univariate, unbiased case, with an error term equal to $(\delta + \beta_1 \gamma)w_i + u_i + \beta_1 e_i$. Since w , u and ϵ_{RED} are uncorrelated, its variance is $(\delta + \beta_1 \gamma)^2 \sigma_w^2 + \sigma_u^2 + \beta_1^2 \sigma_e^2$. □

B MATCHING SIMULATIONS

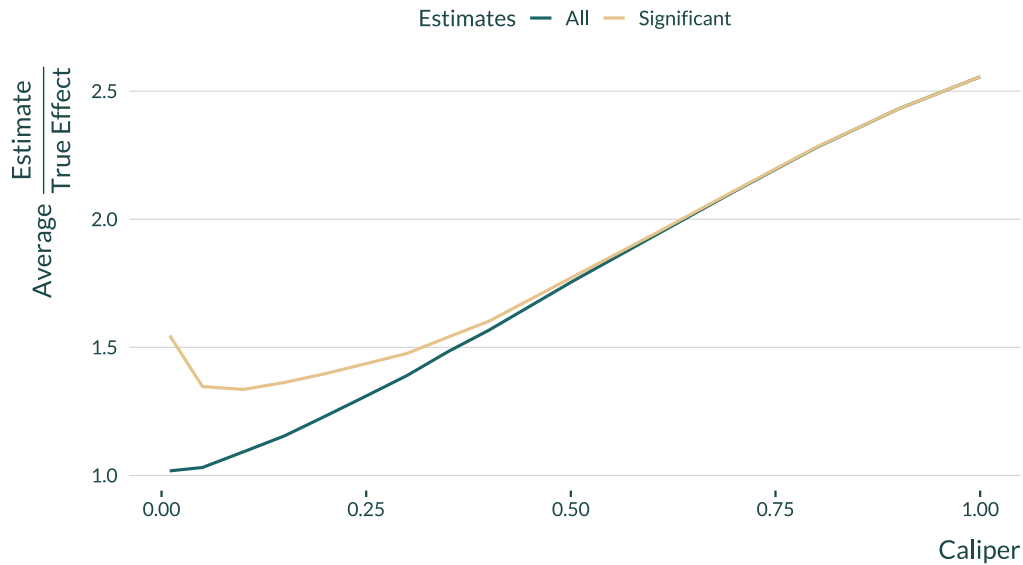
Intuition. Another approach to retrieve a causal effect in a situation of selection on observables is to use matching. This method defines “counterfactuals” for treated units by picking comparable units in the untreated pool. In the case of propensity score matching, treated units are matched to units that would have a similar predicted probability of taking the treatment, *i.e.* couple of units with a difference in propensity score lower than a critical value called the caliper. The smaller the caliper, the more comparable units have to be to be matched and therefore the lower the risk of confounding. Yet, with a stringent caliper, some units may not find a match and be pruned, decreasing the effective sample size. This can lead to a loss in statistical power and produce statistically significant estimates that are inflated. In the case of matching, the confounding-exaggeration trade-off is therefore mediated by the value of the caliper.

Case-study and simulation procedure. I illustrate this issue by simulating a labor training program where the treatment is not randomly allocated (Dehejia and Wahba 1999). Individuals self-select into the training program and may therefore have different characteristics from individuals who do not choose to enroll. To emulate this, I assume that the distribution of the propensity scores differ for treated and control groups: they are drawn from $\mathcal{N}(\mu_T, \sigma_T)$ and $\mathcal{N}(\mu_C, \sigma_C)$ respectively. This can be analogous to considering that matching is done based on the value of a unique covariate. Based on how these propensity scores are created, I define the potential monthly income of each individual i , under the treatment or not.

Based on this simulation framework, I generate 1000 datasets for each propensity score matching procedure with caliper values ranging from 0 to 1. Parameter values of the simulation are set to

make them realistic and can be found [here](#). Once units are matched, I simply regress the observed revenue on the treatment indicator.

Figure 8 – Evolution of Bias with the Caliper in Propensity Score Matching, Conditional on Statistical Significance.



Notes: The green line indicates the average bias for all estimates, regardless of their statistical significance. The beige line represents the inflation of statistically significant estimates at the 5% level. The caliper is expressed in standard deviation of the propensity score distribution. Details on the simulation are available at [this link](#).

Results. Figure 8 indicates that the average bias of estimates, regardless of their statistical significance, decreases with the value of the caliper as units become more comparable. For large caliper values, units are not comparable enough and confoundings bias the effect. For small caliper values, they become comparable but the sample size becomes too small to allow for a precise estimation of the treatment effect and exaggeration arises. Statistically significant estimates never get close of the true effect. This imprecision, and thus exaggeration, results from the fact that the matching procedure does not use information on outcomes that would reduce the residual variance of the model but rather focuses on reducing bias arising from covariates imbalance ([Rubin 2001](#)).