

# Accurately Estimating Relatively Small Effects: Air Pollution and Health

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## Abstract

This paper identifies tangible design parameters that might lead to inaccurate estimates of relatively small effects, the short-term health effects of air pollution. Low statistical power not only makes relatively small effect difficult to detect but resulting published estimates also exaggerate true effect sizes. We first document the existence of this issue in the epidemiology and economics literature of interest. Then, we identify its drivers using real data simulations that replicate most prevailing inference methods. Finally, we argue relevance to many other literatures and propose a principled workflow to evaluate and avoid exaggeration when conducting a non-experimental study.

**Keywords:** Acute Health Effects, Air Pollution, Causal Inference, Exaggeration, Simulations, Statistical Power

**Website:** [https://vincentbagilet.github.io/inference\\_pollution](https://vincentbagilet.github.io/inference_pollution)

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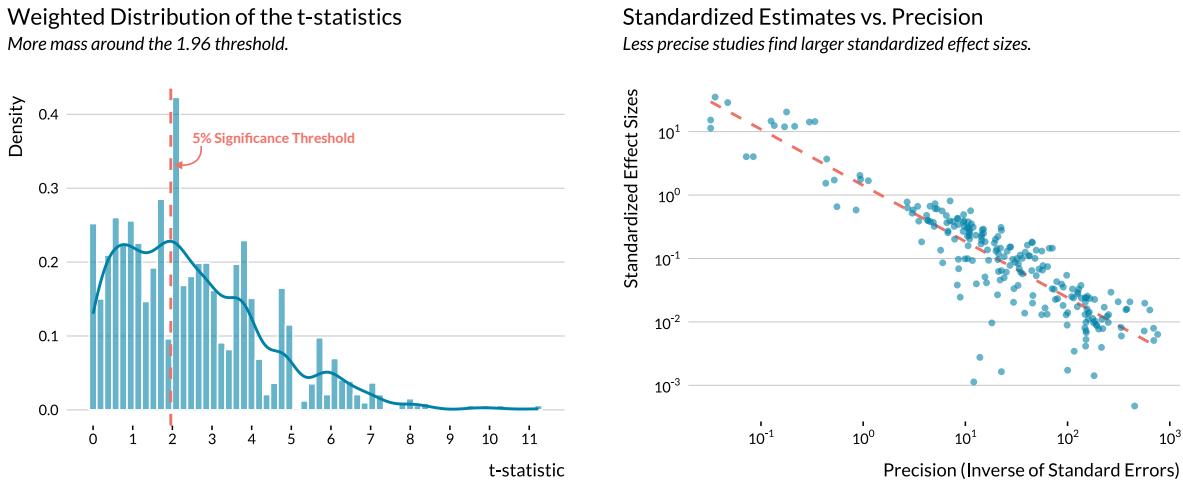
# 1 Introduction

From extreme events such as the London Fog of 1952 to the development of sophisticated time-series analyses, a vast epidemiology literature of more than 600 studies has established that air pollution induces adverse health effects on the very short-term. Increases in the concentration of several ambient air pollutants have been found to be associated with increases in daily mortality and emergency admissions for respiratory and cardiovascular causes (Schwartz 1994, Samet et al. 2000, Le Tertre et al. 2002, Bell, Samet and Dominici 2004, Liu et al. 2019). Based on these results, environmental protection and public health agencies have designed policies such as air quality alerts to mitigate the burden of air pollution. Accurate estimates of these effects are therefore crucial as they are directly used to implement and update policies to address this major public issue.

With this objective in mind, researchers in economics and epidemiology have recently used causal inference methods to improve on the standard epidemiology literature that relied on associations (Dominici and Zigler 2017, Bind 2019). Newly obtained results confirm the short-term health effects of air pollution (Schwartz et al. 2015, Schwartz, Fong and Zanobetti 2018, Deryugina et al. 2019). Yet, causal estimates are substantially larger than what would have been predicted by the standard epidemiology literature, with some estimates being 10 times larger. What can explain such a variation in the magnitude of effect sizes? 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. Our literature review however suggests an alternative but complementary explanation based on statistical power and publication bias.

The left panel of [Figure 1](#) reveals the presence of a publication bias in this literature. Following Brodeur et al. (2016), Brodeur, Cook and Heyes (2020) approach, we show that there is an excess mass in the  $t$ -statistics distribution at the 5% statistical significance threshold. The right panel of [Figure 1](#) produces further evidence of this favoring of significant estimates but also points to a consequence of this publication bias: published estimates from imprecise studies might be exaggerated. If published estimates captured true effects, their standardized effect size should be independent of the precision of the study. However, in this plot we observe that less precise studies display larger standardized effect sizes. Such a pattern emerges when publication favors statistically significant findings. In imprecise settings, only large–too large–effects are significant, e.g., at least 1.96 standard errors away from 0 at the 5% significance level. Such effects are located in the tails of the distribution and do not capture the true effect. Following a similar reasoning, and more generally, relatively imprecise studies will produce inflated published

**Figure 1: Suggestive Evidence of Publication Bias and Exaggeration in the Causal Inference Literature.**



*Notes:* The sample in the left panel includes all 537 estimates reported in articles from the causal literature, including "naive" OLS estimates and placebo tests. Following [Brodeur, Cook and Heyes \(2020\)](#), the weights are equal to the inverse of the number of tests displayed in the same table multiplied by the inverse of the number of tables in the article. In the right panel we exclude the "naive" OLS estimates and placebo tests. Both axes are on a log10 scale. Limiting the sample to economics journal leaves the figures essentially unchanged (see supplemental material). Distinguishing between top 5 and other journals shows that even if there standardized effect sizes are typically smaller in top 5 journals, the same inverse relationship can be observed.

estimates in the presence of publication bias ([Ioannidis 2008, Gelman and Carlin 2014](#))<sup>1</sup>. A lack of relative precision—or equivalently of statistical power—can thus lead to exaggeration and could explain the discrepancy highlighted above. Studies on the short-term health effects of air pollution often display a low relative precision as a result of typically small effect sizes and relatively coarse data, at the city-day level ([Peng, Dominici and Louis 2006, Peng and Dominici 2008](#)). While these factors make them particularly subject to exaggeration issues, many other related or unrelated literature chase relatively small effects and may also suffer from this type of bias.

This paper aims to identify the tangible design parameters that create and drive exaggeration, through the case of studies on the short-term health effects of air pollution. We also document the amount of exaggeration in this literature by gathering 2692 estimates from a unique corpus of 668 articles based on associations and of 36 articles that rely on causal inference methods. For each of these studies, we run calculations to assess whether the design of the study would allow to accurately capture the true effect if it was smaller than the observed estimate ([Gelman and Carlin 2014, Ioannidis, Stanley and](#)

<sup>1</sup>Section 2 illustrates the mechanism in more details using a concrete example.

[Doucouliagos 2017](#), [Lu, Qiu and Deng 2019](#), [Timm 2019](#)). However, such calculations do not highlight the causes of exaggeration. Using real data from the US National Morbidity, Mortality, and Air Pollution Study ([Samet et al. 2000](#)), we therefore implement simulations replicating most prevailing causal and non-causal inference methods to identify the characteristics of research designs that drive exaggeration. We then argue that these tangible parameters should also create exaggeration and bias in other literatures. Finally, we provide a principled workflow to evaluate the risks of exaggeration when carrying out a non-experimental study.

Our literature review results suggest that a substantial share of estimates published in epidemiology and economics could be inflated. To run such calculations, we need to hypothesized true effects sizes. Due to the wide variety of treatments and outcomes considered in this literature, we unfortunately cannot rely on meta-analyses to do so. Since [Ioannidis, Stanley and Doucouliagos \(2017\)](#) and [Ferraro and Shukla \(2020\)](#) find that half of the estimates published in economics and environmental economics are inflated by a factor of at least two, we evaluate the ability of the studies in the review to retrieve effects that would be twice as small as the obtained estimates. Reassuringly, a reasonable share of studies might not suffer from these issues. However, for a quarter of studies, estimated effect sizes would not be able to capture such effects sizes and would exaggerate them by a factor of at least 1.9. For subsets of the two literatures, hypothetical true effect sizes informed by meta-analyzes confirm these exaggeration issues.

Then, our simulation results enable us to identify concrete causes of exaggeration. While our simulations are tuned to study the acute health effects of air pollution, their conclusions likely extend to many other literatures. The intuitions behind the impact of each driver can be applied to most settings, even outside health or air pollution studies. In our context, we first show that as expected exaggeration increases when the sample size decreases. Importantly, we find that for all identification strategies, exaggeration can arise even for large sample sizes. Second, the simulations confirm that the smaller the effect targeted, the larger exaggeration is. They also show that when effect size is small, exaggeration can be substantial. Third, we find that the variation used for identification is a key driver of exaggeration. Using rare exogenous shocks can produce greatly inflated estimates. The number of shocks can represent less than 1% of the observations for some studies leveraging public transportation strikes or thermal inversions as exogenous shocks, leading to large exaggeration ratios even when sample and true effect sizes are large. Similarly, substantial exaggeration can arise when the instrument only explains a limited portion of the variation in air pollution and that, even when F-statistics are large<sup>2</sup>.

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<sup>2</sup>Since this paper focuses on actual implementation of non-experimental studies, it mostly documents

Finally, we show that the count of cases of the outcome is a key driver of exaggeration. Estimated effects of air pollution on the elderly or children can be exaggerated due to the small number of daily hospital admissions or deaths for these groups.

This paper makes three main contributions. First, it contributes to the literature on acute health effects of air pollution and more generally on the impacts of air pollution by underlining the existence of exaggeration issues. We document the presence of a publication bias in this literature and discuss how research design parameters specific to this literature can cause exaggeration.

Second, this paper contributes to a growing literature assessing statistical power and exaggeration issues in various fields (Ioannidis 2008, Gelman and Carlin 2014, Ioannidis, Stanley and Doucouliagos 2017, Ferraro and Shukla 2020, Stommes, Aronow and Sävje 2021, Arel-Bundock et al. 2022). Existing meta-analyses show that the economics literature is plagued with serious power issues but do not usually discuss the determinants of this lack of power. We overcome this key limitation by coupling our literature review with simulations. Outside of meta-analyses, the drivers of exaggeration in non-experimental studies also remain understudied. To our knowledge, only three papers thoroughly address this critical question (Schell, Griffin and Morral 2018, Griffin et al. 2021, Black et al. 2022). We complement these studies focusing on difference-in-differences event-study designs by studying the drivers of exaggeration in a wide array of research designs: standard regression, reduced-form, instrumental variable and regression discontinuity design. We identify design parameters that might drive exaggeration in our literature of interest but also in any applied analysis and invite the reader to pay attention to these tangible characteristics in their own work. We expect power, effective sample size, effect size, the number of shocks or the strength of the instrument to drive the statistical power of most studies, potentially creating important bias in resulting estimates.

Finally, we contribute to a literature discussing the replicability and credibility of empirical findings in non-experimental studies (Button et al. 2013, Open Science Collaboration 2015, Camerer et al. 2018, Brodeur, Cook and Heyes 2020, for instance). We strive to put statistical power at the center of non-experimental analyses since a lack of it can lead to inaccurate published estimates. On the other hand, well-powered studies do not lead to substantial exaggeration even in the presence of publication bias. We thus provide a reproducible workflow to evaluate and avoid exaggeration issues when running a non-experimental study. It suggests to build simulations using fake data or existing datasets before carrying out a study to identify potential exaggeration and its sources.

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tangible drivers. We analyze a theoretical underlying mechanism of exaggeration specific to causal identification strategies (Bagilet and Zabrocki-Hallak 2022).

Once the analysis is completed, we recommend to run a retrospective power analysis to assess whether the design used would have accurately recovered the true effect if it was in fact smaller than the one estimated. We also advocate for reporting these resulting power calculations. To ease the adoption of this workflow, we make all replication and supplementary materials available on the [project's website](#). We also make the algorithm we developed to automatically review the epidemiology literature readily available to almost instantaneously evaluate publication bias and exaggeration issues in other fields reporting point estimates and confidence intervals in plain text.

In the following section, we implement a simple simulation exercise to show why statistically significant estimates exaggerate true effect sizes when studies have low statistical power. In section 3, we present our retrospective analysis of the literature. In section 4, we detail our simulation procedure to replicate empirical strategies. We display the simulation results in section 5 and provide specific guidance to avoid exaggeration when running a non-experimental study in section 6.

## 2 Background on Statistical Power and Exaggeration

In a seminal paper, [Gelman and Carlin \(2014\)](#) point out that statistically significant estimates suffer from a winner's curse in under-powered studies. These estimates can largely exaggerate true effect sizes or can even be of the opposite sign. In this section, we implement a simple simulation exercise to illustrate these two seemingly counter-intuitive issues.

### 2.1 Illustrative Example

Let's simulate an experiment in which a mad scientist is able to increase the concentration of fine particulate matter ( $\text{PM}_{2.5}$ ) to estimate the short-term effects of air pollution on daily non-accidental mortality. The experiment takes place in a major city over the 366 days of a leap year. The scientist increases the concentration of particulate matter by  $10 \mu\text{g}/\text{m}^3$ —a large shock equivalent to a one standard deviation increase in the concentration of  $\text{PM}_{2.5}$ . Concretely, the scientist implements a complete experiment where they randomly allocate half of the days to the treatment group and the other half to the control group. They then measure the treatment effect of the intervention by computing the average difference in means between treated and control outcomes. They find a treatment effect of 4 additional deaths that is statistically significant at the 5% level. The statistical significance of the estimate fulfills the scientist expectations.

Contrary to the scientist, we know the true effect of the experiment since we created the data. [Table 1](#) displays the pair of potential outcomes of each day,  $Y_i(T_i = 0)$  and  $Y_i(T_i = 1)$ .  $Y_i(T_i)$  represents the daily count of non-accidental deaths and  $T_i$  the treatment assignment, equal to 1 when unit  $i$  is treated and 0 otherwise. We first simulate the daily non-accidental mortality counts in the absence of treatment (i.e., the  $Y(0)$  column of [Table 1](#)), by drawing 366 observations from a negative binomial distribution with a mean of 106 and a variance of 402. We choose these parameters to approximate the distribution of non-accidental mortality counts in a large European city. We then define the counterfactual distribution of mortality by adding the treatment effect, drawn from a Poisson distribution (i.e., the  $Y(1)$  column of [Table 1](#)). We choose its parameter to increase the number of death by 1 on average<sup>3</sup>.

**Table 1: Science Table of the Experiment.**

Day Index	$Y_i(0)$	$Y_i(1)$	$\tau_i$	$T_i$	$Y_i^{\text{obs}}$
1	122	124	+2	1	124
2	94	96	+2	1	96
3	96	98	+2	0	96
:	:	:	:	:	
364	96	97	+1	0	96
365	98	98	+0	0	98
366	143	144	+1	1	144

*Notes:* This table displays the potential outcomes, the unit-level treatment effect, the treatment status and the observed daily number of non-accidental deaths for 6 of the 366 daily units in the scientist's experiment.

Following the fundamental problem of causal inference, the daily count of deaths the scientist observes is given by the equation:  $Y_i^{\text{obs}} = T_i \times Y_i(1) + (1 - T_i) \times Y_i(0)$ . Considering that the assignment of the treatment was random, how can the statistically significant estimate found by the scientist be 4 times larger than the true treatment effect size? Replicating the experiment a large number of times explains this apparently puzzling result.

## 2.2 Defining Statistical Power, Exaggeration Ratio and Type S Error

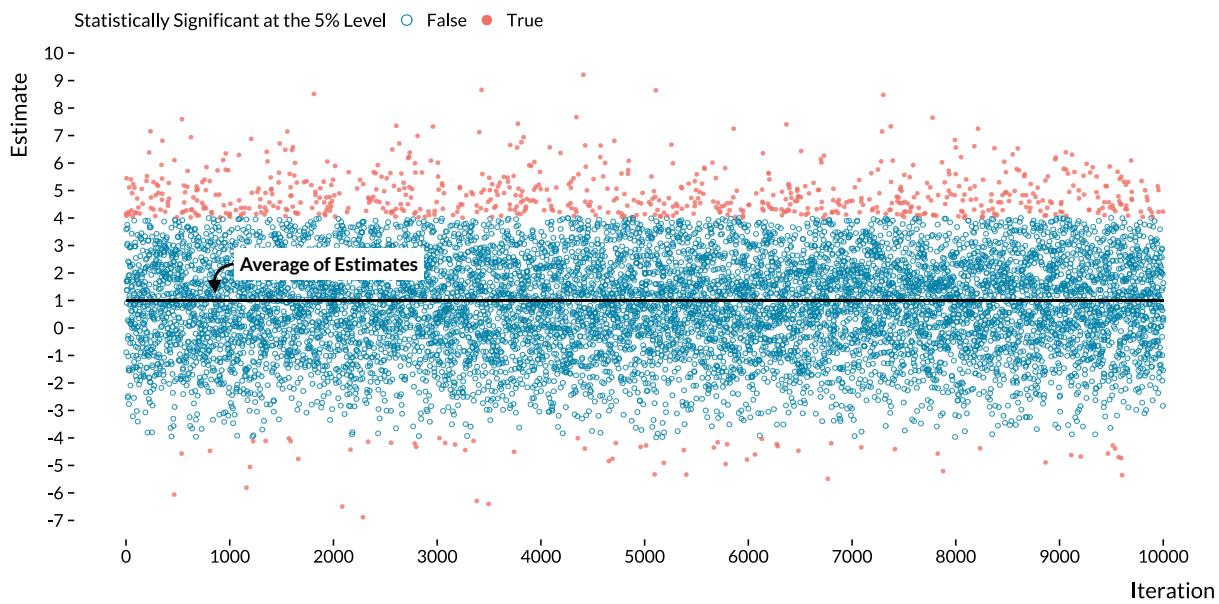
[Figure 2](#) plots the estimates of 10,000 iterations of the experiment. Even if there is

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<sup>3</sup>In relative terms, the treatment effect size we set represents a 1% increase in the health outcome. The magnitude of this hypothetical effect is larger than the one found in a recent and large-scale study based on 625 cities. [Liu et al. \(2019\)](#) estimated that a  $10 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  concentration was associated with a 0.68% (95% CI, 0.59 to 0.77) relative increase in daily all-causes mortality.

a large variation in the effect size of estimates, their average is reassuringly equal to the true treatment effect of 1 additional death. We can however see that estimates close to the true effect size would not be statistically significant at the 5% level. In a world without publication bias, several replications of this experiment would recover the true treatment effect. Unfortunately, despite recent changes in scientific practices and editorial policies, non-statistically significant estimates and replication exercises are still not valued enough ([Brodeur, Cook and Heyes 2020](#)). In a world with publication bias, statistically significant estimates are more likely to be made public. Out of the 10,000 simulation estimates, about 800 are statistically significant at the 5% level. The *statistical power* of the experiment, which is the probability to reject the null hypothesis when there is actually an effect, is equal to 8%. The scientist was therefore lucky to get a statistically significant estimate.

**Figure 2: Replicating 10,000 Times the Experiment.**



*Notes:* Each dot represents a point estimates of one of the 10,000 iterations of the randomized experiment ran by the mad scientist. Red dots are statistically significant at the 5% level while blue dots are not. The black solid line represents the average of estimates, equal to the true average effect of 1 additional death.

With such a low statistical power, statistically significant estimates are however not informative of the treatment of interest. Two metrics, the average type M (magnitude) error and the probability to make a type S (sign) error help assess the negative consequences of a lack of statistical power. The exaggeration ratio, or expected type M error, is defined as the ratio of the absolute values of the statistically significant estimates over the true effect size ([Gelman and Carlin 2014](#)). In the present case, with a statistical power of 8%,

the scientist could expect their statistically significant estimates to be inflated on average by a factor of 5. We also notice in [Figure 2](#) that a non-negligible fraction of statistically significant estimates are of the wrong sign: this proportion is the probability of making a type S error ([Gelman and Carlin 2014](#)). In this experiment, a statistically significant estimate has a 8% probability of being of the wrong sign.

Formally, the statistical power of a test is the probability of rejecting the null hypothesis  $H_0 : \beta = 0$ , where  $\beta$  is the true effect of the estimand of interest. For  $\hat{\beta}$ , a normally distributed unbiased estimate of  $\beta$  with standard error  $\sigma$ , the power of the null hypothesis test at the 5% level is equal to  $\Phi\left(-1.96 - \frac{\beta}{\sigma}\right) + 1 - \Phi\left(1.96 - \frac{\beta}{\sigma}\right)$ , where  $\Phi$  is the cumulative distribution function of the standard normal distribution. It increases with  $\beta$ , the true value of the effect and with the precision of the estimate, *i.e.*, when  $\sigma$  decreases. The exaggeration ratio is  $\mathbb{E}\left(\frac{|\hat{\beta}|}{|\beta|} \mid \beta, \sigma, |\hat{\beta}|/\sigma > 1.96\right)$  and the probability to make a type S error is given by  $\Pr\left(\frac{\hat{\beta}}{\beta} < 0 \mid \beta, \sigma, |\hat{\beta}|/\sigma > 1.96\right)$ . [Zwet and Cator \(2021\)](#) and [Lu, Qiu and Deng \(2019\)](#) derive closed-form expressions for these quantities. They show that both the exaggeration ratio and the probability of type S error decrease with  $\beta$  and the precision of the estimate and thus with statistical power.

To obtain statistically significant estimates that are informative of the true value of the effect size, the scientist would need to improve the design of their study in order to increase its statistical power.

## 3 Retrospective Analysis of the Literature

This section first describes how we ran retrospective analyses of the standard epidemiology and causal inference literatures. We then assess to what extent they could suffer from low statistical power issues.

### 3.1 Our Approach

The formulas for power, exaggeration ratio and type S error described in the previous section all depend on the true magnitude of the estimand of interest. The true effect is however never observed in a given study. We can overcome this limitation with a retrospective power analysis. Essentially, it addresses the following question: would the design of our study be reliable enough to retrieve the true effect if it was in fact smaller than the obtained estimate? A retrospective power analysis can be considered as

a thought-experiment in which we would exactly replicate the study many times under the assumption that the true effect is different from the observed estimate. The reasoning is analogous to the analysis in the previous section. Concretely, [Gelman and Carlin \(2014\)](#) propose to run simulations in which we draw many estimates from the asymptotic distribution of the estimator, a normal distribution with mean equal to the hypothesized true effect and a standard deviation equal to the standard error we obtained in the study. The statistical power is the proportion of sampled estimates that are statistically significant at the 5% level. The exaggeration ratio is computed as the average ratio of the absolute values of statistically significant estimates over the assumed true effect size. The probability to make a type S error is the proportion of significant estimates that are of the opposite sign of the true value. In this project, we use the **R** package `retrodesign` developed by [Timm \(2019\)](#) that implements the closed-form analogue of these simulations ([Lu, Qiu and Deng 2019](#)).

To get a general overview of power issues in the standard epidemiology and causal inference literatures, we first carry out a simple retrospective analysis for each study. These computations rely on hypothesized true effect sizes. Yet, due to variety of treatments and outcomes considered in this literature, it is not possible to make general aggregated assumption on true effect sizes. We have to consider specific hypothesized true effect sizes for each study. Since [Ioannidis, Stanley and Doucouliagos \(2017\)](#) and [Ferraro and Shukla \(2020\)](#) find a typical exaggeration of two in the economics literature. We therefore assess what proportion of studies would have a design reliable enough to retrieve an effect size equal to half of the obtained estimate. On average, by what factor would statistically significant estimates be inflated? A well-designed study should be able to detect a range of plausible effect sizes that are smaller than the observed estimate. This method is however by no means ideal but offers some sort of consistency across studies. To overcome this limitation, for a subset of studies, we also make more elaborate guesses about potential true values of the effect sizes.

## 3.2 Standard Epidemiology Literature

Hundreds of papers have been published on the short-term health effects of air pollution in epidemiology, medicine and public health journals. A large fraction of articles rely on Poisson generalized additive models, which allow to flexibly adjust for the temporal trend of health outcomes and for non-linear effects of weather parameters. This literature spans over 20 years and has replicated analyses in a large number of settings, providing crucial insights on the acute health effect of air pollution.

To gather a corpus of relevant articles, we use the following search query on [PubMed](#) and [Scopus](#):

```
'TITLE(("air pollution" OR "air quality" OR "particulate matter" OR "ozone",  
'OR "nitrogen dioxide" OR "sulfur dioxide" OR "PM10" OR "PM2.5" OR', , "carbon  
dioxide" OR "carbon monoxide"), 'AND ("emergency" OR "mortality" OR "stroke" OR  
"cerebrovascular" OR', , "cardiovascular" OR "death" OR "hospitalization"), 'AND  
NOT ("long term" OR "long-term")) AND "short term"
```

We retrieve the abstracts of 1834 articles. Then, we extract estimates and confidence intervals from these abstracts using regular expressions (regex). Our algorithm available [online](#) detects phrases such as “95% confidence interval (CI)” or “95% CI” and looks for numbers directly before this phrase or after and in a confidence interval-like format. We illustrate the outcome of this procedure (in blue) using one sentence of a randomly selected article from this literature review ([Vichit-Vadakan, Vajanapoom and Ostro 2008](#)):

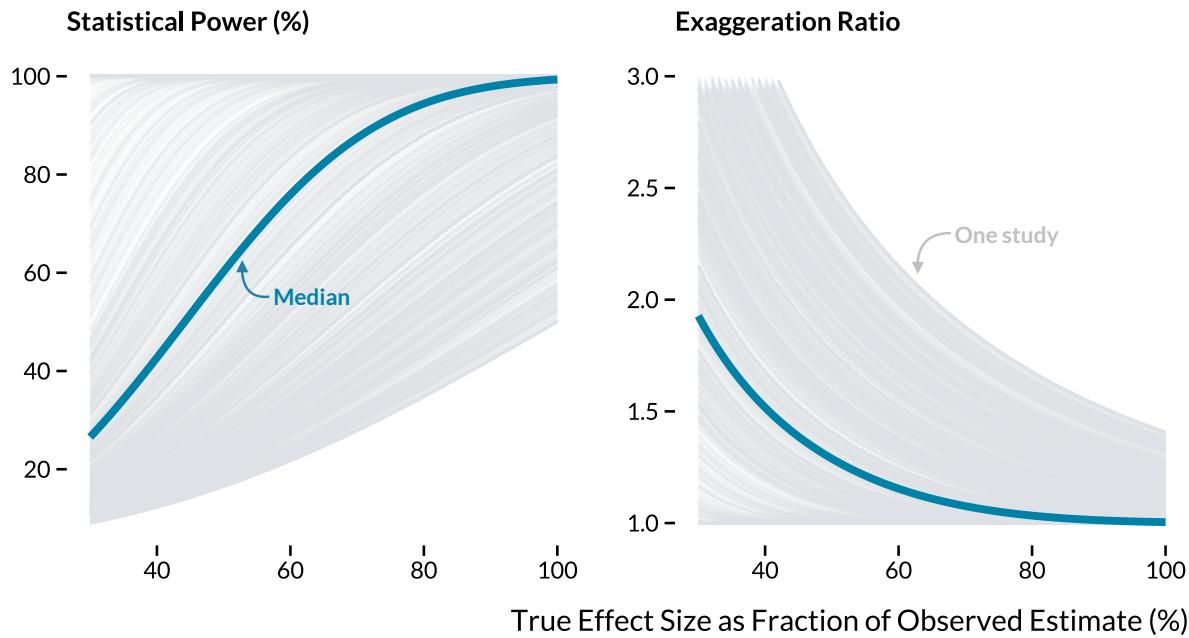
“The excess risk for non-accidental mortality was **1.3% [95% confidence interval (CI), 0.8-1.7]** per 10  $\mu\text{g}/\text{m}^3$  of PM10, with higher excess risks for cardiovascular and above age 65 mortality of **1.9% (95% CI, 0.8-3.0)** and **1.5% (95% CI, 0.9-2.1)**, respectively.”

Using this method, we retrieve 2666 estimates from 784 abstracts. We then read these abstracts and filter out articles whose topic falls outside of the scope of our literature review. The final corpus is thus composed of 668 articles and 2155 estimates. Importantly, the set of articles considered is limited to those displaying confidence intervals and point estimates in their abstracts. We also build regex queries to retrieve other information about the articles such as the air pollutant and health outcome studied, the length of the study and the number of cities considered.

Based on this subset of articles, we first implement a retrospective power analysis to evaluate whether a study could recover an effect size equal to half of the obtained estimate. We carry out this analysis for the 1982 estimates that are statistically significant. [Figure 3](#) displays the power and exaggeration curves for each result. They describe how these metrics vary with the hypothetical true effect sizes.

If the true effect size was equal to half of the obtained estimate, 58% of the studies would have a power below the conventional 80% target used in randomized controlled trials. The median exaggeration ratio would be 1.3 and type S error would not be an issue. These figures however hide a lot of heterogeneity across studies. For one quarter of studies, the exaggeration would be higher than 1.9. We therefore try to apprehend the sources of this heterogeneity.

**Figure 3: Power and Exaggeration Curves for the Epidemiology Literature.**



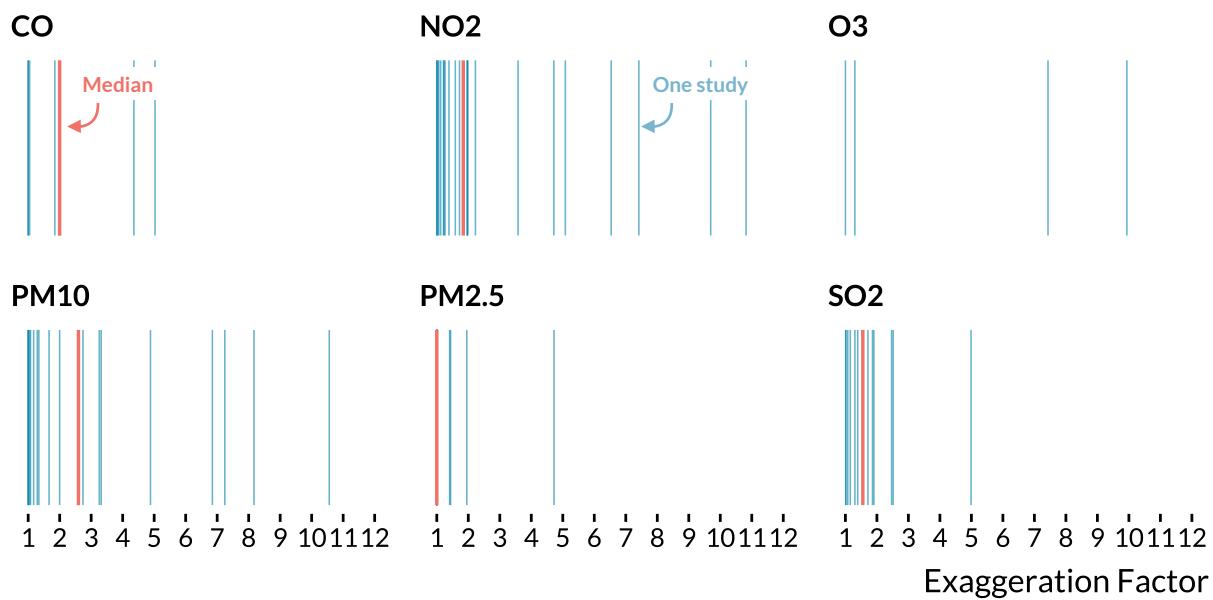
*Notes:* Each gray line is a power curve or an exaggeration curve of a statistically significant result published in the epidemiology literature. The blue lines are the median values. For visual clarity, we drop results for which exaggeration ratios were too large.

We find that inference issues do not depend on the health outcome and the air pollutant studied. Health science journals appear to be less prone to power issues than other journals. Researchers seem to be aware that they should work with large sample size as they often carry out multi-city studies. They also sometimes explicitly state that they investigate non-accidental mortality causes to increase statistical power since the average daily count is higher than for more specific death causes. Yet, the proportion of low power studies has been stagnating since the 2000s, revealing that practices regarding statistical power have not evolved.

Studying the ability of each study to detect an effect that would be half of the obtained estimate gives an overview of power issues in this literature. It can however be viewed as arbitrary. Besides, while our approach enables to study the whole literature, it does not allow to clearly analyze the type of pollutants and outcomes considered in each study. As recommended by [Gelman and Carlin \(2014\)](#) and [Ioannidis, Stanley and Doucouliagos \(2017\)](#), we thus make more informed guesses about potential true effect sizes for a subset of the literature using results from a meta-analysis. [Shah et al. \(2015\)](#) gathered 54 studies on the effects of several air pollutants on mortality and emergency admission for stroke. For each of these studies, we run retrospective power calculations to evaluate their ability

to retrieve the meta-analysis estimates. 63% of studies in [Shah et al. \(2015\)](#) have a statistical power below 80%. The median exaggeration ratio of statistically significant estimate is equal to 1.6. [Figure 4](#) plots for each air pollutant, the distribution of the exaggeration ratios (blue lines) and their medians (orange lines). The median exaggeration varies a lot by air pollutant, from 1 for PM<sub>2.5</sub> up to 13.4 for O<sub>3</sub> (the median is not displayed for visual clarity). More informed guesses about true effect sizes confirm that exaggeration is common in the standard epidemiology literature.

**Figure 4: Distribution of Exaggeration Ratios for Studies in [Shah et al. \(2015\)](#)'s Meta-Analysis.**



*Notes:* Each blue line is the exaggeration ratio of a statistically significant estimate retrieved from [Shah et al. \(2015\)](#)'s meta-analysis. We use the meta-analysis estimates as true effect sizes in the retrospective power calculations. Orange lines are the medians. Extreme exaggeration ratios are removed for visual clarity. The median for O<sub>3</sub> is 13.4.

### 3.3 Causal Inference Literature

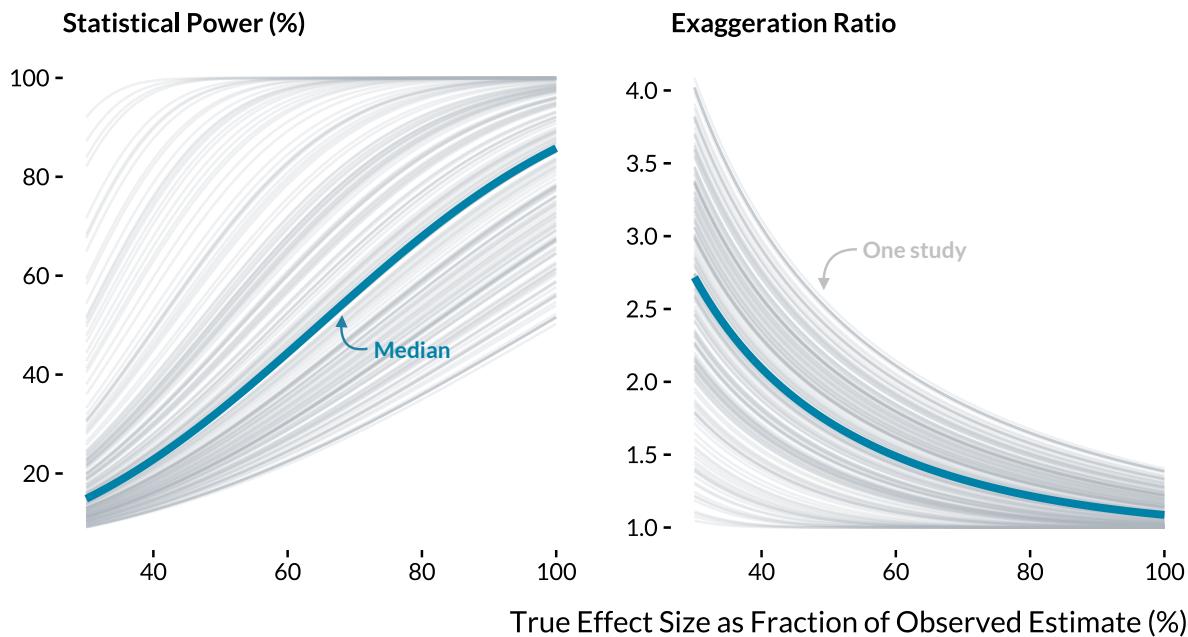
For the causal inference literature, an extensive search strategy on [Google Scholar](#), [IDEAS](#), and [PubMed](#) enables to retrieve studies that (i) focus on the short-term health effects of air pollution on mortality and morbidity outcomes, and (ii) rely on a causal inference methods<sup>4</sup>. Appendix A displays the list of the 36 articles that match the search criteria. For each study, we manually retrieve the method used by the authors, the health outcome

<sup>4</sup>The very recent literature on the effects of air pollution on COVID-19 health outcomes is excluded to gather a relatively homogeneous corpus of studies.

and air pollutant they consider, the point estimate and the standard error of the main specifications.

To evaluate potential statistical power issues in this literature, we follow the same approach as for the analysis of the standard epidemiology literature. [Figure 5](#) plots the power and exaggeration curves for 186 specifications which results are statistically significant at the 5% level. If the true effect size of each study was equal to half of the obtained estimate, the median power would be 33% and the median exaggeration ration would be 1.7. Only 11% of studies would have a power greater than 80%. [Figure 5](#) also shows that there is a wide heterogeneity in statistical power issues among studies. Some of them are relatively well powered while others can run into large exaggeration issues. For instance, one quarter of studies would, on average, exaggerate the true effect sizes by a factor greater than 2. This pattern may help explain the very large effect sizes sometimes observed in the causal inference literature.

**Figure 5: Statistical Power and Exaggeration Curves of Causal Inference Studies.**

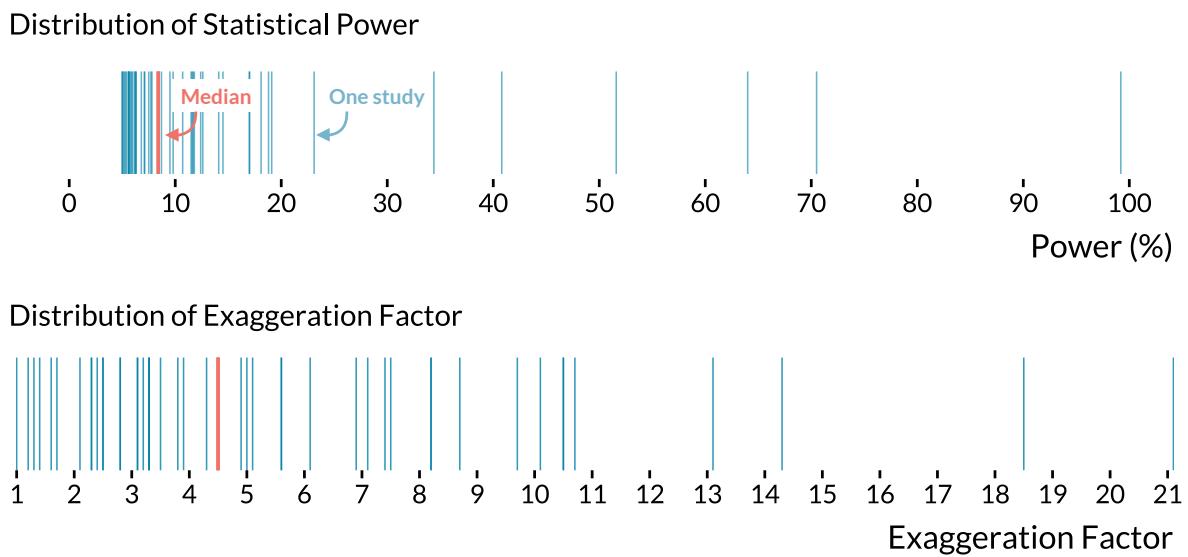


*Notes:* Each gray line is a power curve or an exaggeration curve of a statistically significant result published in the causal inference literature. The blue lines are the median values. For visual clarity, we drop results for which exaggeration ratios were too large.

Then, for the 49 instrumental variable results that are statistically significant and reporting the corresponding naive regression results, we can evaluate whether the 2SLS specifications would recover a true effect closer to that of the naive estimate. [Figure 6](#) displays the distribution of the resulting statistical power and the average exaggeration

ratio. The median power is equal to 8.4%. This results in very large exaggeration ratios: half of the studies would exaggerate a true effect of the size of the OLS estimate by a factor of at least 4.5. Such an inflation of statistically significant estimates could explain part of the gap between the standard and causal literature. This discrepancy could also be explained by a combination of omitted variable bias and attenuation bias caused by classical measurement error in air pollution exposure. It could also come from the fact that the causal estimands targeted by both strategies are different when treatment effects are heterogeneous. Such explanations are not mutually exclusive and the lack of power and inability of the instrumental variables to recover smaller effect sizes remain concerning. In the presence of publication bias, considerable lack of power mechanically causes substantial exaggeration issues.

**Figure 6: Distribution of Power and Exaggeration Ratio for Instrument Variable Designs, Assuming that the Naive OLS Estimates Are the True Effect Sizes.**



*Notes:* For 49 statistically significant 2SLS estimates, we define the true values of effect size as the corresponding OLS estimates. Each blue line represents either the statistical power (%) or the exaggeration factor of a study's result. Orange lines are the median of the two metrics. For visual clarity, we do not display three extreme exaggeration ratios.

## 4 Approach for the Prospective Analysis

The review of the standard epidemiology and causal literatures shows that a sizable portion of articles produce inflated estimates of the short-term health effects of air pollution.

This analysis however does not allow us to clearly identify which parameters of a study influence its statistical power. We therefore implement a prospective analysis to overcome this limitation ([Altoè et al. 2020](#), [Black et al. 2022](#), for other examples of power simulations). We run Monte-Carlo simulations based on real-data to emulate the main empirical strategies found in the literature. We use real data to avoid the difficult task of modeling the long-term and seasonal variations in health outcomes but also the specific effects of weather variables such as temperature. This section describes how these simulations are implemented. Before that, we present the causal identification strategies used to measure the acute health effects of air pollution and then briefly describe the data used for the simulations.

## 4.1 Research Designs to Measure the Short-Term Health Effects of Air Pollution

Several empirical strategies have been leveraged to estimate the short-term health effects of air pollution. We simulate the main ones existing in the literature. We consider the usual setting where data on air pollution, weather parameters, and health outcomes are at the daily-city level.

**Standard regression approach.** The standard strategy consists in directly estimating the dose-response between an air pollutant and an health outcome. In the epidemiology literature, researchers often rely on Poisson generalize additive models where they regress the daily count of an health outcome on an air pollutant concentration, while flexibly adjusting for weather parameters, seasonal and long-term variations. We approximate the workhorse model used by epidemiologists using linear models estimated via ordinary least squares:

$$Y_{c,t} = \alpha + \beta P_{c,t} + \mathbf{W}_{c,t} \lambda + \mathbf{C}_t \gamma + \epsilon_{c,t}$$

where  $c$  is the city index and  $t$  the daily time index.  $Y_{c,t}$  is the daily count of cases of an health outcome and  $P_{c,t}$  the average daily concentration of an air pollutant and  $\epsilon_{c,t}$  an error term. The parameter  $\beta$  captures the short-term effect of an increase in the air pollutant concentration on the health outcome. To address confounding issues, the model adjusts for a set of weather covariates,  $\mathbf{W}_{c,t}$ , and calendar indicators  $\mathbf{C}_t$ .

**Instrumental variable (IV) approach.** The standard strategy could be prone to omitted variable bias and measurement error. A growing number of articles therefore exploits exogenous variations in air pollution. Most causal inference papers rely on IV designs

where the concentration of an air pollutant is instrumented by thermal inversions (Arceo, Hanna and Oliva 2016), wind patterns (Schwartz, Fong and Zanobetti 2018, Deryugina et al. 2019), extreme natural events such as sandstorms or volcano eruptions (Ebenstein, Frank and Reingewertz 2015, Halliday, Lynham and de Paula 2019), or variations in transport traffic (Moretti and Neidell 2011, Knittel, Miller and Sanders 2016, Schlenker and Walker 2016). This approach can be summarized with a two-stage model where the first stage is:

$$P_{c,t} = \delta + \theta Z_{c,t} + \mathbf{W}_{c,t} \eta + \mathbf{C}_t \kappa + \epsilon_{c,t}$$

where  $Z_{c,t}$  is the instrumental variable. The second stage is then:

$$Y_{c,t} = \alpha + \beta \widehat{P}_{c,t} + \mathbf{W}_{c,t} \lambda + \mathbf{C}_t \lambda + \epsilon_{c,t}$$

where  $\widehat{P}_{c,t}$  is the exogenous variation in an air pollutant predicted by the instrument. The causal effect measured by this approach is a weighted average of per-unit causal responses to an increase in the concentration of an air pollutant (Angrist and Imbens 1995).

**Reduced-form approach.** A subset of articles directly estimates the relationship between the health outcome and exogenous shocks to air pollution. For instance, articles using this approach exploit public transport strikes or thermal inversion as exogenous shocks (Bauernschuster, Hener and Rainer 2017, Jans, Johansson and Nilsson 2018, Godzinski, Castillo et al. 2019, Giaccherini, Kopinska and Palma 2021). They estimate a model of the form:

$$Y_{c,t} = \alpha + \beta D_{c,t} + \mathbf{W}_{c,t} \lambda + \mathbf{C}_t \gamma + \epsilon_{c,t}$$

where  $D_{c,t}$  is a dummy equal to 1 when city  $c$  is affected by a shock at time  $t$  and 0 otherwise. The parameter  $\beta$  captures an intention-to-treat effect.

**Regression-discontinuity design (RDD) approach.** The last empirical strategy found in the literature measures the effects of air quality alerts with a regression-discontinuity design (Chen et al. 2018, Anderson, Hyun and Lee 2022). In this approach, the following model is estimated for observations within an air pollution concentration bandwidth around the alert threshold:

$$Y_{c,t} = \alpha + \beta \mathbf{1}\{P_{c,t} > P_c^{(a)}\} + \mathbf{W}_{c,t} \lambda + \mathbf{C}_t \gamma + \epsilon_{c,t}$$

where  $P_c^{(a)}$  is the air pollution alert threshold for city  $c$ . We restrict our simulations to the case of sharp RDD. This model estimates the intention-to-treat effect of air quality alerts.

It can both capture the effect of a subsequent decrease in air pollution caused by traffic restriction policies and inhabitants' avoidance behavior.

## 4.2 Data

The simulation exercises rely on a subset of the US National Morbidity, Mortality, and Air Pollution Study (NMMAPS). The dataset is publicly available and has been used in several major studies in the early 2000s to measure the short-term effects of ambient air pollutants on mortality outcomes ([Peng and Dominici 2008](#)). Specifically, we extract data at the city-day level for 68 cities over the 1987-1997 period. It corresponds to 4,018 daily observations per city, for a total sample size of 273,224 observations. We select observations on the average temperature ( $C^\circ$ ), the standardized concentration of carbon monoxide (CO), and mortality counts for several causes. We focus on CO as it is the air pollutant measured in most cities over the period and its concentration is strongly correlated to that of other pollutants such as particulate matter. Less than 5% of carbon monoxide concentrations and average temperature readings are missing in the initial data set. We impute them using the chained random forest algorithm implemented in the missRanger package ([Mayer 2019](#)).

## 4.3 Simulations Set-Up

**General procedure.** Our simulation procedure follows 7 main steps:

1. Randomly draw a study period and a sample of cities.
2. For instrumental variable, reduced-form and regression-discontinuity designs, randomly allocate days to exogenous shocks/air quality alerts.
3. Modify the health outcome, adding a treatment effect that we will try to recover.
4. Estimate the model.
5. Store the point estimate of interest and its standard error.
6. Repeat the procedure 1000 times.
7. Compute the proportion of statistically significant estimates at the 5% level (the power), the average of the absolute value of significant estimates over the true effect size (the exaggeration ratio), and the proportion of significant estimates of the opposite sign of the true effect (the probability to make a type S error).

**Modeling assumptions.** To only capture the specific issues arising due to low statistical power, we build our simulations such that (i) they meet all the required assumptions of empirical strategies and (ii) make it easier—compared to real settings—to recover the treatment effect. For all research designs, the treatment added to the data is not biased by unmeasured confounders nor measurement errors. For instrumental variable and reduced-form strategies, we only simulate binary and randomly allocated exogenous shocks (*e.g.* the occurrence of a thermal inversion). For the regression discontinuity approach, we only model sharp designs where an air quality alert is always activated above a randomly chosen threshold. The simulations always retrieve on average the true value of the treatment effect.

**Two approaches for simulating research designs.** For the reduced-form and regression discontinuity designs, we follow the Neyman-Rubin causal framework by simulating all potential outcomes (Rubin 1974). Consider that the health outcome value recorded in the NMMAPS dataset corresponds to the potential outcome  $Y_{c,t}(0)$ . To create the counterfactuals  $Y_{c,t}(1)$ , we add a treatment effect drawn from a Poisson distribution whose parameter corresponds to the magnitude of the treatment. We then randomly draw the treatment indicators  $T_{t,c}$  for exogenous shocks or air quality alerts. For reduced-form strategies, the treatment status of each day is drawn from a Bernoulli distribution with parameter equal to the proportion of exogenous shocks desired. For air pollution alerts, we randomly draw a threshold from a uniform distribution and select a bandwidth such that it yields the desired proportion of treated observations. We finally express the observed values  $Y^{obs}$  of potential outcomes according to the treatment assignment:  $Y_{c,t}^{obs} = (1-T_{c,t}) \times Y_{c,t}(0) + T_{c,t} \times Y_{c,t}(1)$ .

To simulate standard regression and the instrumental variable strategies, we rely on a model-based approach. For the standard regression strategy, we first estimate the following statistical model on the data:

$$Y_{c,t} = \alpha + \beta Z_{c,t} + \mathbf{W}_{c,t} \lambda + \mathbf{C}_t \gamma + \epsilon_{c,t}$$

We then predict new observations of a  $Y_{c,t}$  using the estimated coefficients of the model ( $\hat{\beta}$ ,  $\hat{\lambda}$ , and  $\hat{\gamma}$ ) and by adding noise drawn from a normal distribution with variance equal to that of the residuals  $\widehat{\epsilon}_{c,t}$  (Peng, Dominici and Louis 2006). We modify the slope of the dose-response relationship by changing the value of the air pollution coefficient  $\beta$ . For the instrumental variable strategy, we use the same method as for the standard regression approach but first modify observed air pollutant concentrations  $P_{c,t}$  according to the desired effect size  $\theta$  of the randomly allocated instrument:

$$\widetilde{P}_{c,t} = P_{c,t} + \theta Z_{c,t}$$

We draw the allocation of each day to an exogenous shock from a Bernoulli distribution with parameter equal to the proportion of exogenous shocks. We then estimate a two-stage least squares model (2SLS) and modify the coefficient for the effect of the air pollutant on an health outcome. We finally generate the fake observations of the health outcome by combining the prediction from the modified 2SLS model and noise drawn a normal distribution with variance equal to that of the residuals.

**Varying parameters.** To understand which parameters affect statistical power issues, we modify one aspect of the research design while keeping other parameters constant. We study the influence of four main parameters. First, we vary the sample size by drawing a different number of cities and changing the length of the study period. Second, we consider different effect sizes of air pollution or of an exogenous shock on the health outcome. Third, we allocate increasing proportions of exogenous shocks/air quality alerts. Fourth, we vary the number of cases in the outcome by considering different health outcomes.

**Simulations of Case Studies.** The simulations described above help explore the effect of each parameter on statistical power issues. Yet, the resulting set of parameters considered may not be perfectly representative of actual studies. To address this concern, we also calibrate simulation parameters to reproduce three papers published in the literature. We report these analyzes in Appendix C.

## 5 Results of the Prospective Analysis

In this section, we describe how statistical power evolves with the treatment effect size, the number of observations, the proportion of exogenous shocks, the average count of the health outcome, and the strength of the instrument. In Appendix C, we show that statistical power issues can be substantial for actual parameter values found in the literature.

### 5.1 Evolution of Power, Exaggeration Ratio and Type S Error with Study Parameters

We aim to analyze how statistical power, exaggeration ratio and type S error are affected by the value of different study parameters. To do so, we set baseline values for these

parameters and vary the value of each of them one by one. This enables us to get a sense of the impact of each parameter, other things held equal. We consider the following baseline parameters:

- A large sample size of 100,000 observations (2500 days  $\times$  40 cities),
- A 1% effect size, the order of magnitude found in the most precise studies of the literature. A one standard deviation in air pollution or an exogenous shock increases the health outcome by 1%,
- 50% of observations are subject to an exogenous shock. For air pollution alerts analyzed with regression discontinuity designs, we only consider observations close to the threshold, resulting in a smaller proportion of treated units: 10%,
- The health outcome is the total daily number of non-accidental deaths. It is the health outcome with the largest average number of counts (average daily mean of 23 cases).

For all statistical models, we adjust for temperature, temperature squared, city and calendar (weekday, month, year, month $\times$ year) fixed effects. We also repeat the simulations for a smaller sample size of 10,000 observations.

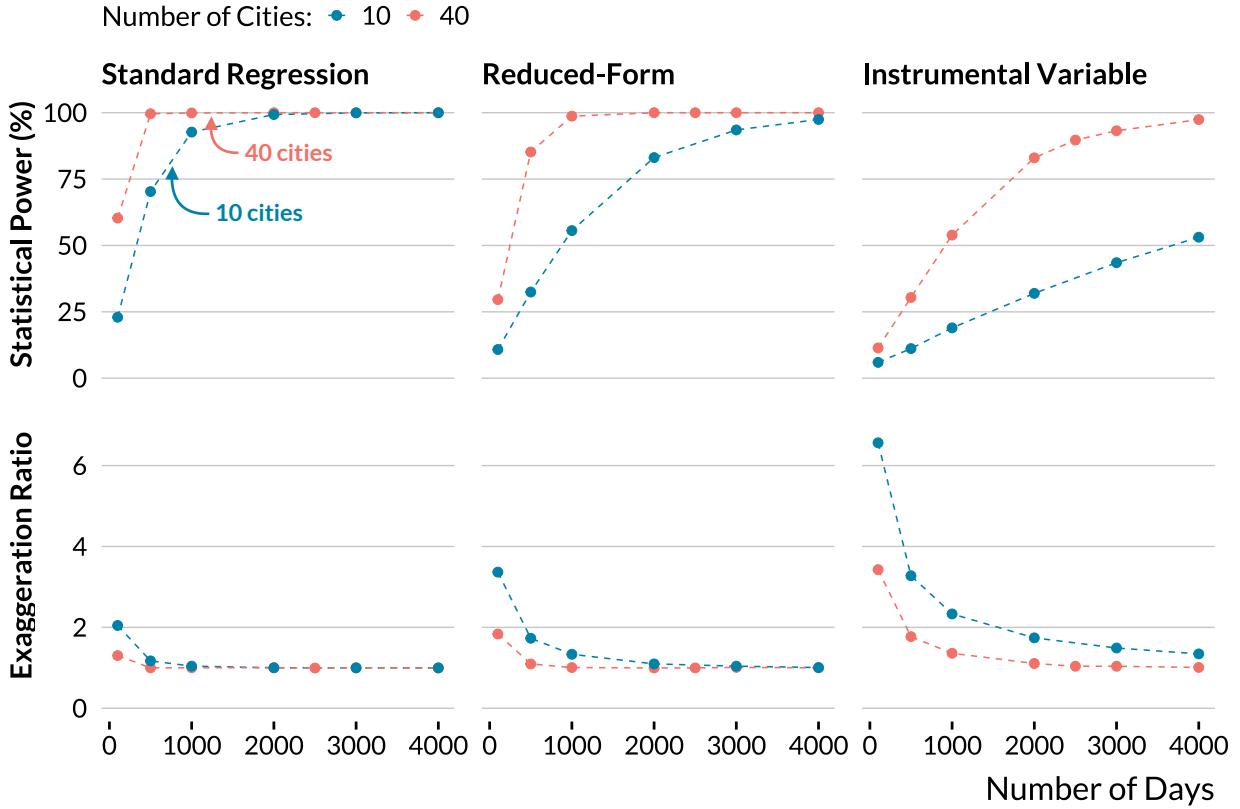
## Sample Size

In [Figure 7](#), we recover the well-known increasing relationship between the number of observations and statistical power. Conversely, the exaggeration ratio decreases with the number of observations.

This results stems from the fact that statistical power increases and exaggeration decreases when the variance of a normally distributed estimator decreases ([Zwet and Cator 2021](#), [Lu, Qiu and Deng 2019](#)). Now, the variance of usual estimators decreases with the number of observations. Then, the relationships between number of observation, power and exaggeration.

We also find that statistical power and exaggeration issues can arise even for a large number of observations. For a sample size of 40,000 observations, the instrumental variable strategy only has a statistical power of 54% and exaggerates the true effect by a factor of 1.4. On the contrary, the standard regression strategy is much less prone to power issues than the instrumental variable strategy. This is explained by the fact that the variance of the two stage least-square estimator is larger than the variance of the ordinary least square estimator. In our simulations, the probability to make a Type S error is null for all identification methods and sample sizes.

**Figure 7: Evolution of Power and Exaggeration with Sample Size.**



*Notes:* The other parameters are set to their baseline values: a true effect size of 1%, 50% of observations subject to an exogenous shock for instrumental variable and reduced-form designs, and the health outcome is the total number of non-accidental deaths.

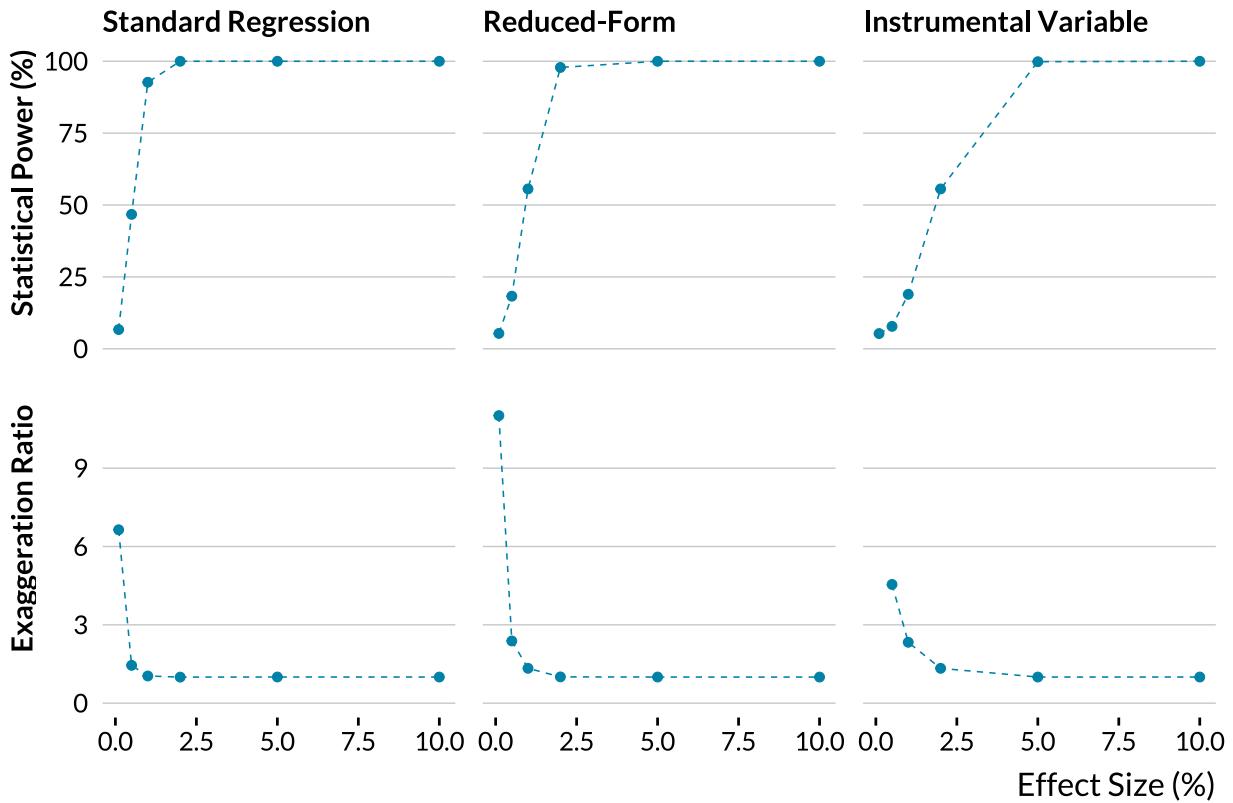
## Effect Size

In Figure 8, we retrieve another familiar result: the larger the effect size, the larger the power. As expected from [Zwet and Cator \(2021\)](#) and [Lu, Qiu and Deng \(2019\)](#)'s results, we also find that the exaggeration ratio decreases with the true effect size. Even for our large baseline sample size, statistical power issues appear for effect sizes routinely found in the epidemiology literature. For instance, for our instrumental variable strategy and an effect size of 0.5%, the average exaggeration ratio is about 1.7. As for results on sample sizes, standard regression and reduced-form strategies are less prone to power issues, even for small effect sizes.

## Proportion of Exogenous Shocks

The link between the proportion of exogenous shocks and statistical power might be less widely known. In Figure 9, we show that statistical power increases with the proportion

**Figure 8: Evolution of Power and Exaggeration with Effect Size.**



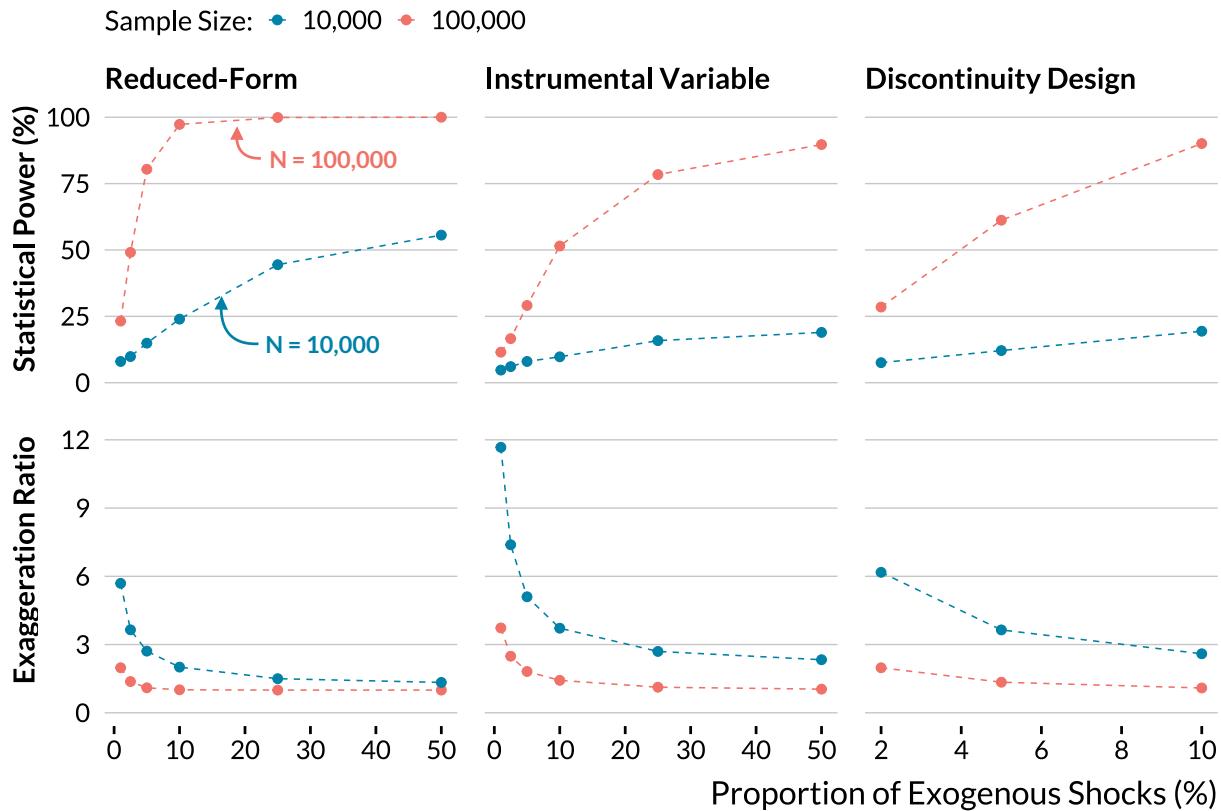
*Notes:* The sample size is 10,000. The other parameters are set to their baseline values: 50% of observations subject to an exogenous shock for instrumental variable and reduced-form designs, and the health outcome is the total number of non-accidental deaths. For an effect size of 1%, we do not display the exaggeration ratio of the instrumental variable design since it is above 20 and it would distort the graph.

of treated units for instrumental variable, regression discontinuity and reduced-form designs. Conversely, the average exaggeration ratio increases as the proportion of exogenous shocks decreases.

This result can be explained by the fact that exaggeration increases and statistical power decreases with the variance of the estimator ([Zwet and Cator 2021](#), [Lu, Qiu and Deng 2019](#)). Now, as routinely discussed for randomized controlled trials but seldom in the case of non-experimental studies, precision is maximized when half of the observations are exposed to the treatment of interest. The variance of the average treatment effect estimator (ATE) is  $\sigma^2/[n \times p(1-p)]$  where  $\sigma$  is the standard deviation of the outcome in the treated and control groups and  $p$  the proportion of treated units. This quantity increases when  $p$  departs from 0.5. Thus, exaggeration increases when the proportion of exogenous shocks decreases, as long as it was initially smaller than 0.5.

Another way to interpret this result is to consider that a small number of exogenous

**Figure 9: Evolution of Power and Exaggeration with the Proportion of Exogenous Shocks.**



*Notes:* The other parameters are set to their baseline values: a true effect size of 1% and the health outcome is the total number of non-accidental deaths. The proportion of exogenous shocks corresponds to the fraction of days in the sample that are allocated to the treatment.

shocks limits the variation that can be leverage to identify the effect of interest. When the proportion of shocks decreases, the variance of the treatment variable decreases and therefore the variance of the estimator increases. A similar reasoning can be applied to IV strategies.

In practice, air pollution alerts, thermal inversion or transportation strikes are generally rare events. In some studies, they represent less than 5% of the observations. With a dataset of 10,000 observations, our simulations return an average exaggeration ratio of 2.7 for the reduced-form strategy. Despite large sample sizes, air pollution studies exploiting few exogenous shocks might be particularly prone to exaggeration issues.

### Average Count of Cases of the Health Outcome

Subgroup analyses are routinely carried out in the literature to evaluate the acute health effects of air pollution on children or the elderly. Yet, the average count of cases can also

**Table 2: Evolution of Power and Exaggeration with the Average Number of Daily Cases of Health Outcomes.**

	Non-Accidental	Respiratory	COPD
Number of Cases	23	2	0.3
Statistical Power (%)	90	16	7.5
Exaggeration Ratio	1	2.4	5.9

*Notes:* This table displays the average number of cases, the power and the exaggeration ratio for three health outcomes: non-accidental deaths, respiratory deaths, and chronic pulmonary deaths for individuals aged between 65 and 75. These figures are obtained for the instrumental variable design with a sample size of 100,000 and 50% of observations subject to an exogenous shock. The instrument variable increases the air pollutant concentration by 0.5 standard deviation. A one standard deviation increase in the instrumented air pollutant leads to 1% relative increase in the health outcome considered.

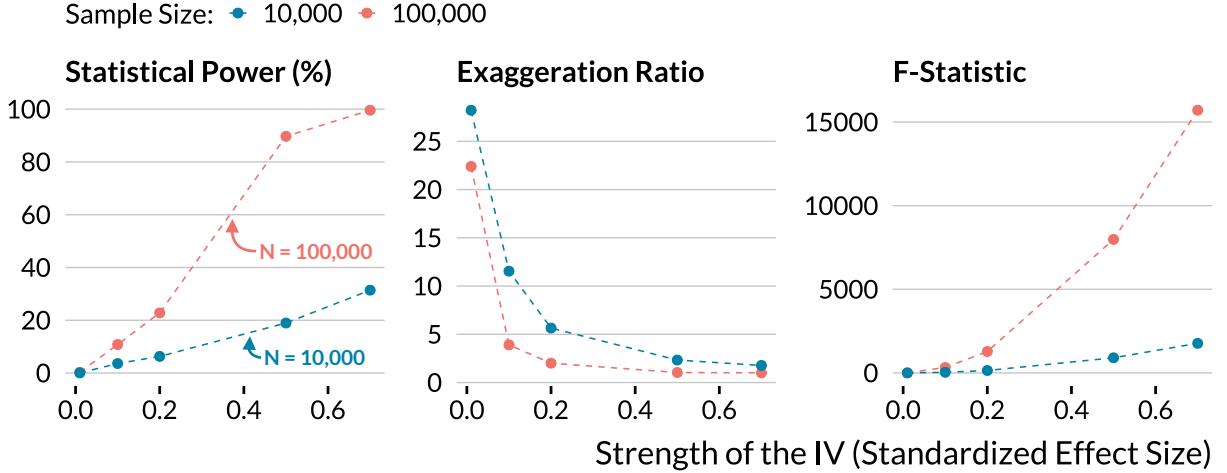
critically affect statistical power as shown in [Table 2](#). For instance, in a setting with only few deaths per day, a 1% increase in the number of deaths will rarely cause additional deaths. The effect will be more difficult to detect. To simulate situations with various number of cases, we consider three different outcome variables, with different counts of cases: the total number of non-accidental deaths (daily mean  $\simeq 23$ ), the total number of respiratory deaths (daily mean  $\simeq 2$ ) and the number of chronic obstructive pulmonary disease (COPD) cases for individuals aged between 65 and 75 (daily mean  $\simeq 0.3$ ). Using baseline parameters and in the case of the large dataset, we find that statistical power is close to 100% for a 1% increase in the total number of non-accidental deaths. However, statistical power drops when the average count of cases decreases. For instance, the instrumental variable strategy has only 16% of statistical power to detect a 1% increase in respiratory deaths. The average exaggeration ratio is then equal to 2.4. For chronic obstructive pulmonary deaths—the health outcome with the lowest number of cases—the situation is even worst since the average exaggeration ratio reaches 5.9. When focusing on subgroups such as children or the elderly, one can expect to find larger effect sizes as those populations are more vulnerable to air pollution. While these larger effect sizes attenuate exaggeration concerns, the lower number of cases exacerbates them. It creates a trade-off for power issues.

### Issues Specific to the Instrumental Variable Design

In the case of instrumental variable strategies, statistical power is affected by the strength of the instrument. In our simulations, we consider a binary instrument (e.g., the occurrence of a thermal inversion or a public transport strike). We define its strength as the

standardized effect size of the instrument on the air pollutant concentration. A strength of 0.2 means that the instrument increases the concentration by 0.2 standard deviation.

**Figure 10: Evolution of Power and Exaggeration with the Strength of the Instrumental Variable.**



*Notes:* The true effect size is a 1% relative increase in the health outcome. The health outcome used in the simulations is the total number of non-accidental deaths. Half of the observations are exposed to exogenous shocks. The strength of the instrumental variable is defined as its effect in standard deviation on the air pollutant concentration.

As shown in [Figure 10](#), we find that statistical power collapses and exaggeration soars when the instrument's strength decreases. Importantly, this issue even arises for large first-stage  $F$ -statistics. In our simulations based the large data set with 100,000 observations, an instrumental variable's strength of 0.2, and an effect size of 1%, we find an average  $F$ -statistics of 1278. The statistical power is however only 23% and the average exaggeration ratio 2. A large  $F$ -statistic could therefore hide large exaggeration issues.

The relationship between IV strength and exaggeration comes from the fact that the variance of the 2SLS estimator decreases with the correlation between the instrument and the instrumented variable. In the homoskedastic case, the asymptotic variance of the 2SLS estimator is  $(\mathbb{E}[XZ']\mathbb{E}[ZZ']^{-1}\mathbb{E}[ZX'])^{-1}\sigma^2$ , where  $\sigma^2$  is variance of the error,  $X$  the endogenous variable and  $Z$  the instrument. When  $\mathbb{E}[XZ']$  and  $\mathbb{E}[ZX']$  decrease, the variance of the estimator increases. Again, since [Zwet and Cator \(2021\)](#) and [Lu, Qiu and Deng \(2019\)](#) show that as the variance of a normally distributed estimator increases, the statistical power decreases and exaggeration increases, we obtain the intuition for the simulation results.

## 6 Discussion

Growing evidence shows the existence of statistical power issues and publication bias towards statistical significance in economics, causing exaggeration (Brodeur et al. 2016, Ioannidis, Stanley and Doucouliagos 2017, Brodeur, Cook and Heyes 2020, Ferraro and Shukla 2020). Although this issue appears to be increasingly acknowledged, discussions about the drivers of low power and therefore actionable guidance to tackle it in non-experimental economic research are still lacking (Altoè et al. 2020, Black et al. 2022). In this paper, we highlighted a list of concrete drivers of power and exaggeration we should pay attention to when carrying a study on the short-term health effects of air pollution. In the present section, we first discuss how this list could translate to other contexts before proposing a principled workflow to assess if and understand why an estimate could be inflated.

While the simulations we ran were specific to studies on the acute health effect of air pollution, we argue that they can provide lessons for other types of non-experimental studies. First, a large literature investigates the short-term impacts of air pollution on different outcomes such as criminality, cognitive skills and productivity (Herrnstadt et al. 2021, Ebenstein, Lavy and Roth 2016, Adhvaryu, Kala and Nyshadham 2022, for instance). These studies use data with a very similar structure, only focusing on different outcomes and find effects of comparable magnitude or smaller than those in our literature of interest. Our results should therefore directly carry to these literatures. More broadly, settings with typically low signal-to-noise ratios can by definition be subject to power and exaggeration issues. Since as described in Section 5, the impact of each driver we identified can be explained theoretically, we expect these drivers to affect power and exaggeration in other settings as well. A limited effect size, effective sample size, number of exogenous shocks, average count of the outcome or strength of the instrument can create exaggeration in many settings. To avoid exaggeration issues when running a non-experimental study, we thus recommend to particularly pay attention to these factors.

In addition to these specific factors, when carrying out a study, we recommend to systematically run retrospective calculations to gauge the risk of exaggeration. They are easy to implement and force us to discuss credible effect size. They allow to evaluate if our research design enables us to confidently estimate a credible range of effect sizes. We implemented and discussed such calculations in our literature review and illustrate this approach in more details in Appendix B by considering the example of Deryugina et al. (2019). As an even simpler first check, we also suggest to consider large confidence intervals verging 0 not only as a sign of uncertainty regarding the exact magnitude of the

effect but also of limited power and potential exaggeration of the obtained point estimate.

Then, we recommend conducting prospective simulations before undertaking a non-experimental study. It allows to verify whether our design can detect effects of a credible magnitude in an almost-ideal setting. Unlike a retrospective analysis, enables us to identify factors that drive exaggeration. Fake-data can be simulated from scratch or simulations can build on datasets used in other studies, as we did in the simulation section of this paper. To facilitate the adoption of this practice, we describe the template we use to run our simulations in the replication material. [Black et al. \(2022\)](#) also provide useful recommendations to implement power simulations.

More generally, we advocate for paying attention to statistical power in non-experimental studies, even after we obtain a statistically significant estimate, as insufficient power can lead to exaggeration and inaccurate published estimates. As such, we recommend reporting power calculations to demonstrate the robustness of the design and its ability to accurately capture smaller effect sizes.

On top of these specific recommendations, we should not forget that published estimates only suffer from exaggeration in the presence of publication bias. The causal inference literature would therefore benefit from adopting a different view towards statistically insignificant results ([Ziliak and McCloskey 2008](#), [Wasserstein and Lazar 2016](#), [McShane et al. 2019](#)). It currently dichotomizes evidence according to the 5% significance threshold, disregarding non-significant results ([Greenland 2017](#)). Instead, if results were published regardless of their significance, the resulting distribution would be centered around the true effect ([Hernán 2022](#)). To replace the null hypothesis testing framework, we recommend to focus on confidence intervals and to interpret the range of effect sizes supported by the data ([Amrhein, Trafimow and Greenland 2019](#), [Romer 2020](#)). Qualifying estimates as "statistically significant" does not acknowledge the actual uncertainty that should be computed and embraced to better help policy-makers. Prospective and retrospective power analyses can help design better studies and improve the interpretation of their results.

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## A List of Studies Included in the Causal Inference Literature

We display below studies included in the retrospective analysis of the causal inference literature. We group them by research designs:

**Instrumental Variable Design:** Moretti and Neidell (2011), Ebenstein, Frank and Reingewertz (2015), Schwartz et al. (2015), Arceo, Hanna and Oliva (2016), He, Fan and Zhou (2016), Knittel, Miller and Sanders (2016), Schlenker and Walker (2016), Sheldon and Sankaran (2017), Schwartz, Bind and Kourakis (2017), Zhong, Cao and Wang (2017), Barwick et al. (2018), Hanlon (2018), Schwartz, Fong and Zanobetti (2018), Halliday, Lynham and de Paula (2019), Deryugina et al. (2019), Cheung, He and Pan (2020), Fan and Wang (2020), He, Liu and Zhou (2020), Giaccherini, Kopinska and Palma (2021), Godzinski and Suarez Castillo (2021), Guidetti, Pereda and Severnini (2021), Kim (2021), Liu and Ao (2021), Xia et al. (2022)

**Reduced-Form Design:** Bauernschuster, Hener and Rainer (2017), Jans, Johansson and Nilsson (2018), Jia and Ku (2019), Godzinski, Castillo et al. (2019)

**Regression Discontinuity Design:** Chen, Guo and Huang (2018), Fan, He and Zhou (2020), Anderson, Hyun and Lee (2022)

**Event-Study Design:** Mullins and Bharadwaj (2015), Simeonova et al. (2021)

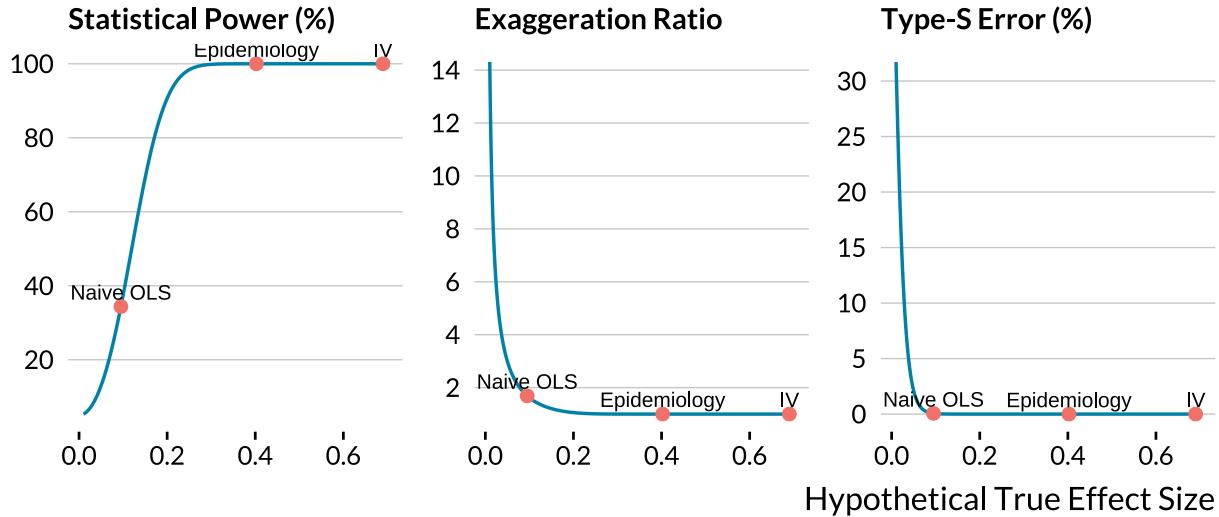
**Matching Design:** Baccini et al. (2017), Forastiere, Carugno and Baccini (2020)

## B Implementing a Retrospective Power Analysis

We explain here how we can easily implement a retrospective power analysis once a study is completed. In a flagship publication, Deryugina et al. (2019) instrument PM<sub>2.5</sub> concentrations with wind directions to estimate its effect on mortality, health care use, and medical costs among the US elderly. They gathered 1,980,549 daily observations at the county-level over the 1999–2013 period; it is one of the biggest sample sizes in the literature. When the authors instrument PM<sub>2.5</sub> with wind direction, they find that “a 1  $\mu\text{g}/\text{m}^3$  (about 10 percent of the mean) increase in PM<sub>2.5</sub> exposure for one day causes 0.69 additional deaths per million elderly individuals over the three-day window that spans the day of the increase and the following two days”. The estimate’s standard error is equal to 0.061. In Figure B.1, we plot the statistical power, the inflation factor of statistically

significant estimates and the probability that they are of the wrong sign as a function of hypothetical true effect sizes.

**Figure B.1: Power, Type M and S Errors Curves for Deryugina et al. (2019).**



*Notes:* In each panel, a metric, such as the statistical power, the exaggeration ratio or the probability to make a type S error, is plotted against the range of hypothetical effect sizes. The "IV" label represents the value of the corresponding metric for an effect size equal to Deryugina et al. (2019)'s two-stage least square estimate. The "Epidemiology" label stands for the estimate found in Di et al. (2017), which is the epidemiology article most similar to Deryugina et al. (2019). The "Naive OLS" label corresponds to the estimate found by Deryugina et al. (2019) when the air pollutant is not instrumented.

The estimate found by Deryugina et al. (2019) represents a relative increase of 0.18% in mortality. We labeled it as "IV" in Figure B.1. Is this estimated effect size large compared to those reported in the standard epidemiology literature? We found a similar article to draw a comparison. Using a case-crossover design and conditional logistic regression, Di et al. (2017) find that a  $1 \mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  is associated with a 0.105% relative increase in all-cause mortality in the Medicare population from 2000 to 2012. The effect size found by Deryugina et al. (2019) is larger than this estimate labeled as "Epidemiology" in Figure B.1. If the estimate found by Di et al. (2017) was actually the true effect size of  $\text{PM}_{2.5}$  on elderly mortality, the study of Deryugina et al. (2019) would have enough statistical power to perfectly avoid type M and S errors. Now, suppose that the true effect of the increase in  $\text{PM}_{2.5}$  was 0.095 additional deaths per million elderly individuals—the estimate the authors found with a "naive" multivariate regression model. The statistical power would be 34%, the probability to make a type S error could be null but the exaggeration factor would be on average equal to 1.7. Even with a sample size of nearly 2 million observations, Deryugina et al. (2019) could make a non-negligible type M error if the true effect size was the naive ordinary least square estimate. Yet,

the authors could argue that their instrumental variable strategy leads to a higher effect size as it overcomes unmeasured confounding bias and measurement error. Besides, for effect sizes down to 0.182 additional deaths per million elderly individuals (a 0.05% relative increase), their study has a very high statistical power and would not run into substantial type M error. A retrospective analysis is thus a very convenient way to think about the statistical power of a study to accurately detect alternative effect sizes.

## C Case Studies

The main simulation results help understand how the various parameters influence the statistical power of studies. Yet, these parameters may not perfectly represent actual studies as we made several conservative assumptions: relatively large sample size, proportion of treated units, average outcome counts and instrumental variable strength. For each research design, we therefore consider a realistic set of parameters based on an example from the literature. We then vary the value of key parameters. As we are working with different data, we cannot exactly reproduce the level of precision found in the articles considered. Our goal is not to claim that the estimates produced by a particular article are inflated, but instead to understand how low power issues could arise for representative parameter values.

### Public Transportation Strikes

Public transportation strikes are unique but rare positive shocks to air pollution as individuals use their cars to reach city centers. Even in a large data set, with several cities and a long study period, the proportion of affected days might be very small. For instance, [Bauernschuster, Hener and Rainer \(2017\)](#) investigate the effect of public transportation strikes on air pollution and emergency admission in the five biggest German cities over a period of 6 years. Despite a sample size of 11,000, there are only 57 1-day strikes during the study period (0.5% of days are actually treated). The authors find that children hospitalizations for breathing issues increase by 34% (SE=8%) on strike days. On average, 0.22 children per day go to the hospital for breathing issues.

We simulate a similar design with our own data. We first randomly sample 2200 observations for five cities and then vary (i) the proportion of exogenous shocks from 0.5% up to 10%, and (ii) the treatment effect size from a 4% increase up to a 34% increase. We focus on elderly mortality due to chronic obstructive pulmonary disease since it has an average daily count of 0.29 cases.

**Figure C.2: Evolution of Power and Exaggeration for Public Transportation Strikes Designs.**



*Notes:* Each panel displays the average value of a metric (power, exaggeration, and standard error) for varying proportions of exogenous shocks and effect sizes. The average standard error of simulations is the raw standard error divided by the mean number of cases of the health outcome. For each combination of parameters, we ran 1000 simulations.

In Figure C.2, we display our simulation results. The first panel from the left shows that both large effect sizes and a large proportion of exogenous shocks are required to reach adequate power. In the middle panel, we show that a proportion of 0.5% of exogenous shocks is associated with very large exaggeration ratios, from 2.2 for a true effect size of 34% up to 14 for one of 4%. Power issues fade for a combination of a proportion of exogenous shocks above 5% and effect sizes above 17%. In the right panel, we plot the average standard error of the estimates, expressed as a fraction of the average of the health outcome. The standard error of Bauernschuster, Hener and Rainer (2017)'s is 8%. In our simulations, we recover that specific precision for a proportion of exogenous shocks of 5%. In that case, a true effect size of 34% would not yield inflated estimates. However, if effect sizes are actually smaller and more representative of those found in the literature, the exaggeration would be consequential.

This simulation exercise shows that exaggeration is likely to arise in practice since the proportion of exogenous shocks is low. It occurs even when true effect sizes are relatively large.

## Air Pollution Alerts

Air pollution alerts are also rare events. Their effects are estimated using regression discontinuity designs that restrict the analysis to observations closed to the air quality

threshold. As a consequence, the effective sample size may be particularly small. For instance, [Chen et al. \(2018\)](#) investigate the effects of air quality alerts on emergency department visits in Toronto, over the 2003-2012 period. While the nominal sample size is 3652, the effective one is only 143 (100 control days and 43 treated days). Only 1.2% of observations are treated. The authors find that eligibility to air quality (the intention-to-treat effect) approximately reduces emergency visits for asthma by 8% (SE=3.8%). The average daily count of cases of their health outcome is 26.

We approximate the setting of [Chen et al. \(2018\)](#) using our data. We first sample one city for a time period of 3652 days and randomly allocate the treatment. We then repeat the process varying the proportion of alerts and effect sizes. Our outcome variable is the total number of non-accidental deaths since it has a daily mean of 23.

**Figure C.3: Evolution of Power and Exaggeration for Air Quality Alerts Designs.**



*Notes:* Each panel displays the average value of a metric (power, exaggeration, and standard error) for varying proportions of exogenous shocks and effect sizes. The average standard error of simulations is the raw standard error divided by the mean number of cases of the health outcome. For each combination of parameters, we ran 1000 simulations.

[Figure C.3](#) displays the simulations results. As in [Figure C.2](#), a combination of large effect sizes and many air quality alerts is needed to avoid low power issues. We get a precision similar to [Chen et al. \(2018\)](#) for a proportion of air quality alerts of 3%. For an effect size of 4%, the average exaggeration ratio is equal to 2.6. In that case, the average average of statistically significant estimates is 10%, which is similar to the effect size found by [Chen, Guo and Huang \(2018\)](#).

Unless true effect sizes are very large, air quality alert designs produce inflated estimates in realistic settings.

## Instrumenting Air Pollution

Finally, we investigate the most commonly used strategy in the causal inference literature, the instrumental variable design. Several studies rely on very large datasets and exploit changes in weather patterns as sources of exogenous variations. For instance, [Schwartz, Fong and Zanobetti \(2018\)](#) instrument PM<sub>2.5</sub> concentration with planetary boundary layer, winds speed, and air pressure. Once the effects of seasonal and other weather parameters are accounted for, the combination of their instruments explains 18% of the variation in PM<sub>2.5</sub> concentration. They find that a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> leads to a 1.5% (SE=0.22%) increase in daily non-accidental mortality. There are on average 23 daily deaths in their dataset of 591,570 observations (135 cities with a length of study of approximately 4382 days).

In our simulations, we assess how the strength of the instrumental variable affects power issues for several health outcomes. We consider a binary instrumental variable and vary its effect on air pollution concentration from a 0.1 to a 0.5 standard deviation increase. The 18% correlation in [Schwartz, Fong and Zanobetti \(2018\)](#) corresponds to a 0.4 standard deviation increase in our case ([Lipsey and Wilson 2001](#)). We assume that half of the observations are exposed to exogenous shocks. We set an effect size corresponding to a 1.5% relative increase in three health outcomes with different average number of cases: non-accidental mortality (mean cases of 23), respiratory mortality (mean of 2), and chronic obstructive pulmonary mortality of elderly (mean of 0.3). Our data set being smaller than the one used in [Schwartz, Fong and Zanobetti \(2018\)](#), we only run simulations for a sample size of 100,000.

In [Figure C.4](#), we see in the top-left panel that power reaches satisfactory level for large instrumental variable strengths but only for non-accidental causes. For respiratory and elderly mortality, exaggeration can be substantial even for large IV strength. While our sample size is large, it is smaller than the one in [Schwartz, Fong and Zanobetti \(2018\)](#). As a consequence, our simulations only have a precision close to theirs for an instrumental variable strength of 0.5 and non-accidental mortality. Yet, our simulations highlight that important exaggeration issues can arise in realistic settings, even for large IV strength. The bottom-right panel of [Figure C.4](#) confirms the result found in the simulations of the previous section: a large first stage *F*-statistic can be a poor indicator of statistical power issues. For instance, for non-accidental mortality and an IV strength of 0.1, the *F*-statistic is equal to 320 but the exaggeration factor is 2.6, with an associated power of 16%. Importantly, as the *F*-statistic does not vary with the number of cases in the outcome it can all the more hide important power issues.

**Figure C.4: Evolution of Power and Exaggeration for Instrumental Variable Designs.**



*Notes:* Each panel display the average value of a metric (power, exaggeration, standard error, and first-stage F-statistic.) for varying proportions of exogenous shocks and effect sizes. The average standard error of simulations is the raw standard error divided by the mean number of cases of the health outcome. For each combination of parameters, we ran 1000 simulations.