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Documents de travail

N° 2021-004 - Juillet 2021





Institut national de la statistique et des études économiques

G2021/04

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Juillet 2021

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We thank the audience at the AERE Virtual 2020, CREST seminar, Causal Machine Learning Workshop 2020 in St Gallen, the ESEM2019, EAERE 2019, LAGV 2019, ESPE 2019, INSEE seminar, 10th French Econometrics Conference 2018 for their suggestions and useful comments. We thank David Benatia, Jérémy l'Hour and Christophe Gaillac for hindsightful discussions, Laurent Gobillon, Dominique Goux, Sébastien Roux, Antoine Dechezleprêtre and Pauline Givord for their comments and suggestions. We thank the organizers and participants of the Machine Learning for Economics workshop at the Barcelona GSE Forum 2019, whose comments help improve this paper. We are especially grateful to Frédérique Chéruy for the help with the LMDZ model, and to Frédéric Hourdin. We thank Pierre Bayart and Chantal Vilette at Insee for their help with the civil registry data. We thank the CépiDc-Inserm for providing us the mortality by cause database. We thank Alireza Banaei, Fatma Kaci, Anne Bataillard, Max Bensadon and Françoise Bourgoïn from ATIH for providing us the hospital admission database, Anne Laborie, Gilles Levigoureux and Frédéric Penven from ATMO France and the AASQA for providing us the air pollution datasets. This work has been partly funded by a French government subsidy managed by the Agence Nationale de la Recherche under the framework of the Investissements d'avenir programme reference ANR-17-EURE-001. Corresponding author: milena.suarez-castillo@insee.fr.

Distinguer les effets des polluants dans l'air à l'aide de nombreux instruments

Résumé

La pollution atmosphérique constitue une menace majeure pour la santé humaine. Loin d'être unidimensionnelle, la pollution de l'air est multiforme, ce qui complique l'étude des conséquences de polluants multiples dans des études quasi expérimentales. En sélectionnant les instruments optimaux à partir d'un large ensemble de variables instrumentales de météorologie d'altitude, nous démêlons l'impact de cinq polluants atmosphériques dans une évaluation complète de leur impact sanitaire à court terme dans les plus grandes zones urbaines de France sur 2010-2015. Nous constatons que des niveaux journaliers plus élevés d'au moins deux polluants atmosphériques, l'ozone et le dioxyde de soufre, conduisent le jour même à davantage d'admissions aux urgences liées aux maladies des voies respiratoires. Les enfants et les personnes âgées sont les plus touchés. Un niveau plus élevé de monoxyde de carbone conduit à davantage d'admissions aux urgences pour les maladies cardiovasculaires, tandis que des niveaux plus importants en particules fines (PM2.5) et en dioxyde de soufre conduisent à une augmentation du taux de mortalité journalier. En supposant un contexte de cinq polluants atmosphériques, nous montrons qu'un analyste qui aurait ignoré dans son modèle la présence de l'ensemble des polluants atmosphériques aurait tiré des conclusions partiellement fausses.

Mots-clés : polluants de l'air, santé, admissions aux urgences, mortalité, couche limite planétaire, IV Lasso

Disentangling the effects of air pollutants with many instruments

Abstract

Air pollution poses a major threat to human health. Far from unidimensional, air pollution is multifaceted, but quasi-experimental studies have been struggling to grasp the consequences of the multiple hazards. By selecting optimal instruments from a novel and large set of altitude-weather instrumental variables, we disentangle the impact of five air pollutants in a comprehensive assessment of their short-term health impact in the largest urban areas of France over 2010-2015. We find that daily higher levels of at least two air pollutants, ozone and sulfur dioxide, lead to more respiratory-related emergency admissions on the same day. Children and elderly are mostly affected. Carbon monoxide increases emergency admissions for cardiovascular diseases while particulate matter and sulfur dioxide are found responsible for increasing the daily mortality rate. Assuming a five air pollutants context, we show that an analyst who ignored the presence of interrelations between air pollutants would have reached partially false conclusions.

Keywords: air pollution, health, emergency hospital admissions, mortality, planetary boundary layer, IV Lasso

Classification JEL : C26; C55; I18; Q51; Q53

1 Introduction

To protect human health, urban environmental regulations increasingly rely on ambient pollutant concentrations both to inform and take actions. Thanks to the high frequency monitoring systems in place in large cities, local authorities may implement driving restrictions, impose lower speed limits or ban industrial activities when a pollutant concentration exceeds a regulatory threshold. The avoided damage when concentrations fall are central to the design of these environmental policies, first and foremost damage from the respective health impacts of pollutants forming the urban air pollution mixture. Whereas recent quasi-experimental evidence relative to global air quality is clear-cut, disentangling the effect of distinct air pollutants has been a long-discussed challenge. While having received considerable attention, it remains a key difficulty in observational studies. In the discussions for updating the global air quality guidelines, the World Health Organization has set “Causality and independence of effects including multi-pollutant effect estimates as a basis for joint health impact assessment” as a key point for debate, highlighting the relevance of describing and regulating jointly several pollutants ([WHO, 2018](#)).¹

In this paper, we conduct a large-scale quasi-experimental study of the short-term concomitant effects of five air pollutants on daily emergency admissions and mortality in the largest urban areas of France over 2010-2015. We focus on the impact of daily air pollutants levels on same day health outcomes. We address the challenge posed by the highly correlated daily variations of air pollutants by leveraging a novel and large set of instruments which describe extensively altitude weather conditions e.g. winds or temperature profiles. By mining predictive relationships to find instruments for each air pollutant separately, we disentangle the concomitant effects of the main pollutants of the urban mixture. Our first contribution is to provide causal evidence on the separate effects of five air pollutants on both short-term morbidity and mortality, in the real urban environment, while controlling for the other pollutants. Our second contribution is to suggest a novel set of instruments which allows precise estimations when leveraged with the IV-Lasso method by [Belloni et al. \(2012\)](#). We define multipollutant effects as the sum of each air pollutant impact, while controlling for the presence of the others: we do not consider interactions nor threshold nor non-linear effects.

¹The three other discussion points are (i) the shape of the concentration-response function and the identification of thresholds and effects at very low or very high pollutant levels, (ii) effects at different exposure duration times (long-term, short-term) and (iii) considerations regarding vulnerable sub-groups or windows of susceptibility.

Concerns about extrapolating associational estimates have been voiced insistently ([Currie et al., 2011](#), [Dominici et al., 2014](#), [Bind, 2019](#)), but causal estimation remains challenging. Air pollution is not allocated randomly through time and space and may serve as a surrogate for a number of economic and population variables (e.g. traffic, industrial activities, bank holidays...), therefore the well-known challenge to isolate exogenous air pollution variations, even at high frequency. While causal estimates are considered as the gold standard to inform public policies, quasi-experimental studies are still scarce, and typically not able to isolate a given pollutant effect, but rather a cocktail of several ingredients. To identify air pollution effects, the quasi-experimental literature has been very creative in finding external shocks affecting air pollution independently of health outcomes. Authors have taken advantage of plausibly exogenous shocks such as airport congestion ([Schlenker and Walker, 2015](#)), daily boat traffic ([Moretti and Neidell, 2011](#)), changes in local traffic ([Currie and Walker, 2011](#), [Knittel et al., 2016](#), [Simeonova et al., 2018](#)) or recession ([Chay and Greenstone, 2003](#)). The nature of the shocks underpinning the estimations entails quasi-random variations of air quality - but not pollutant-specific variations.

Car traffic engender emissions of particulate matter, carbon monoxide and nitrogen oxides as primary pollutants, and indirectly ozone, a secondary pollutant formed from primary sources. A lower economic activity entails a slowdown in emissions from industry, reducing among other sulfur dioxide and particulate matter. Pollutant concentrations often vary together as they share some common sources, but approximating air pollution by a unidimensional phenomenon might be questionable, not least because some air pollutants are strongly anti-correlated due to chemical equilibrium. As a result, studies relying on these global sources of variations are not well suited to separate the causal effect of distinct air pollutants. Some recent studies resort to finding exogenous shocks specific to one pollutant e.g. [Halliday et al. \(2019\)](#) who use volcano eruptions whose chemical composition is very specific, hence justifying a single-pollutant model. Using change in wind directions as instruments, [Deryugina et al. \(2019\)](#) show that the PM_{2.5} impact on elderly mortality is more robust than that of other pollutants, instrumenting jointly for three pollutants (particulate matter, carbon monoxide and ozone). Yet a broader set of instruments may increase the ability to separate the impact of more air pollutants - showing that not only PMs may be impacting daily mortality. In contrast, most of the existing literature is based on single-pollutant models and authors generally acknowledge that the given pollutant

under study may serve as a surrogate for another.²

In parallel, the emergence of novel econometric and data science techniques has fostered the hope that the causal effects of each air pollutant could be more precisely estimated (Carone et al., 2020). In this spirit, a number of studies intend to explore multi-pollutant exposure consequences with random forest or clustering over pollution profiles but remain ultimately correlative evidence (Zanobetti et al., 2014, Bobb et al., 2015, Tavallali et al., 2020). This paper moves beyond both literature by using a wide set of physical instruments allowing to separate the short-term impact of five air pollutants in a quasi-experimental setting and over a broad set of outcomes.³ We here observe and consider simultaneously six long-regulated hazardous pollutants which have been the focus of the first and often-revised national and international standards for protecting human health: particulate matter of less than 2.5 micrometers PM_{2.5}, of less than 10 micrometers PM₁₀, carbon monoxide CO, nitrogen dioxide NO₂, ozone O₃ and sulfur dioxide SO₂. On a relatively small sample, we bridge the gap between on one hand, quasi-experimental studies which often lacked sufficiently distinct exogenous shocks to disentangle air pollutant effects, and on the other hand, associational studies using data-mining techniques within multi-pollutant models.

For this study, we use a novel and large set of instruments, altitude weather variables: thermal inversions, planetary boundary layer height, altitude winds and altitude pressures derived from a general climate model - the LMDZ model, from the *Laboratoire de Météorologie Dynamique*.⁴ We exploit the richness of a great number of instrumental variables to predict each pollutant variation. Indeed, the atmosphere dynamics, such as wind effects, plays a key role in the mixing, the chemistry and the dispersion of urban air pollution and thus in the ambient air pollution inhaled by the population. The exclusion restriction for this type of IV strategy is that, after largely and flexibly controlling for surface weather variables and city-specific seasonal fixed effects, changes in altitude weather variables are unrelated to changes in population

²This is in particular true for observational studies, as opposed to lab experiments where gases exposure are under the experimenter controls. Although informative, the later are reduced in scope for obvious ethical reasons and may thus be confined to non-representative populations and exposure.

³The long-term causal impact is beyond this study while conveying greater consequences than short-term estimates. For instance, the International Agency for Research on Cancer has recently classified PM mixture as carcinogenic to human (IARC, 2013).

⁴<http://lmdz.lmd.jussieu.fr/> This model among others contributes to fueling Intergovernmental Panel on Climate Change (IPCC) reports (See (Hourdin et al., 2006) and (Dufresne et al., 2013)).

health outcomes except through their influence on air pollutant concentrations. The specification includes very flexible month-by-year-by-city fixed effects and day-of-the-week-by-city fixed effects, so the estimates are identified from deviations within month-year-city cells on similar week days,⁵ and we control for daily temperature, humidity, precipitations and wind strength specified as polynomials of order two, and sunlight and presence of snow. Controlling for surface weather is important inasmuch as they have a direct effect on health and are correlated to our instruments, so a number of robustness checks to the main specification are examined. Individually, some of these instruments have been used to instrument a unidimensional air pollution component. [Arceo et al. \(2016\)](#), [Jans et al. \(2018\)](#), [Chen et al. \(2018\)](#) and [Sager \(2019\)](#) rely on thermal inversions, an inversion of the gradient of vertical temperature profiles which favors polluted conditions. [Deryugina et al. \(2019\)](#) and [Anderson \(2019\)](#) use wind characteristics. [Schwartz et al. \(2016\)](#) use surface wind speed and the planetary boundary layer height, a key driver of ground-level air quality although still under-used in the literature.

To derive pollutant-specific causal effects, we use optimal instrument selection among a high-dimensional set of altitude weather variables, relying on the econometric theory by [Belloni et al. \(2012, 2016\)](#) and [Chernozhukov et al. \(2015\)](#).⁶ These recent techniques allow us to select instruments in an optimal way, avoiding ad-hoc selection and enhancing precision in a setting where it is decisively needed. Compared to the literature drawing causal inference from the unpredictable components of weather variations, the originality here is to use a large set of altitude weather conditions as opposed to a sub-component, and let the data reveal the underlying strongest relationships. We may indeed find many other and more complex phenomena linking altitude weather variables to ground-level pollution by leveraging the rich set of instruments at hand. Isolating different exogenous reasons for each pollutant variation with an IV-Lasso, we prove the empirical added-value of these recent high-dimensional econometric methods, whose applications are too often confined to repeating the existing analysis. Indeed, it is in practice very rare to rely on a naturally-large set of instruments.

This study contributes to the recent literature in economics which estimates the health effects

⁵In other words, the assumptions we need to draw up a causal statement is that we correctly account for the direct effect of surface weather on health, and that across dates within a city-month-year cell, variations in altitude weather phenomena have no direct impact on population health, except through variations in air pollutant concentrations.

⁶For another application, in a different context, see [Gilchrist and Sands \(2016\)](#)

of air pollution in quasi-experimental settings (Currie et al., 2011, Schlenker and Walker, 2015, Deryugina et al., 2019). We combine daily air pollutant concentration data with administrative data on location-specific daily mortality and emergency hospital admissions for cardiovascular and respiratory diseases across age-groups. These data cover over six years (2010-2015) the ten largest urban areas of France, where about 40% of the French population lives. Our results show that ozone and sulfur dioxide impact positively emergency admissions for respiratory diseases, independently from each other and even after controlling for the other pollutants. Quantitatively, we find 4% more respiratory admissions when O₃ goes up by $+ 10 \mu\text{g}/\text{m}^{-3}$ (about half a standard deviation) and 7% more respiratory admissions when SO₂ goes up by $+ 1 \mu\text{g}/\text{m}^{-3}$ (two-third of a standard deviation). These aggregate effects are mostly driven by emergency admissions of young children and elderly. Although not in all specification, some of the models suggest an additional impact of carbon monoxide on respiratory emergency admissions. On cardiovascular diseases, we find an impact of carbon monoxide: $+ 100 \mu\text{g}/\text{m}^{-3}$ (about half a standard deviation) leads to 4% additional emergency admissions. Moreover, we find an effect of PM_{2.5} on cardiovascular-related mortality: $+ 10 \mu\text{g}/\text{m}^{-3}$ (about a standard deviation) leads to a 5% higher mortality rate for deaths with at least one cardiovascular cause (or a 2% increase in the mortality rate). An increase by $+ 1 \mu\text{g}/\text{m}^{-3}$ of SO₂ translates to a 10% higher mortality rate for deaths with at least one respiratory cause (or a 2% increase in the mortality rate). These short-term health effect estimates are significant even when controlling the family-wise error rate of at least one false rejection out of the five hypothesis tests (one per candidate pollutant).

Our last contribution is to shed light on the shortcomings of single-pollutant models compared to multi-pollutant models, by providing an extensive comparison of the results in both paradigms. If most pollutants can be found as having a strong causal effect on short-term health in single-pollutant models, multi-pollutant models offer a more nuanced picture. In single-pollutant models, there may be pollutants acting as surrogate for the others, entailing misleading conclusions. For all outcomes, we reject the equality of estimates from single-pollutant IVs with these of a multi-pollutant IV-Lasso. When instruments are specifically chosen for each pollutant, we reject equality between single and multi-pollutant models for mortality outcomes. These results may question the proxy paradigm which often is the rule in empirical analysis. For instance, if NO₂ has been advocated as a good candidate to proxy for all pollutants in Levy et al. (2014), we find no effect of this pollutant (at short-term) when other pollutants enter the equation. In addition, controlling for four other pollutants and selecting optimally the instru-

ments allow to eliminate the odd finding that O₃ leads to a *decrease* in mortality or emergency admissions. This spurious result is usually explained by the strong negative correlation that this pollutant has with other pollutants.⁷

More generally, our results tie into the literature intending to design policy instruments in a multi-pollutant context e.g. [Montero \(2001\)](#), [Ambec and Coria \(2013\)](#), [Fullerton and Karney \(2018\)](#). Rich economic valuation of environmental policies taking several major pollutants into account, such as [Holland et al. \(2018\)](#) or [Clay et al. \(2019\)](#), substantially rely on integrated assessment models where the health impact measurement is a key step, and is taken from studies which generally use the proxy approach. This paper contributes to quantifying the respective marginal benefit in reducing distinct pollutants, in a context of increasing interest in regulating air pollutants jointly. In addition, our result put into question the current implementation of Air Quality Indexes, which are generally specified as maximum over pollutant sub-indexes, ruling out concomitant effects. Real-time AQI information about air pollution has been shown critical for defensive investment and protective behaviour ([Neidell, 2009](#), [Zhang and Mu, 2018](#), [Barwick et al., 2019](#)).

The article proceeds as follows. In the second section, we introduce background information on pollutants, estimation of health impacts and on pollutants' interaction with weather conditions. In the third section, we present jointly the data and the mechanisms at work. Then, we present and discuss the empirical strategy and the instruments' selection procedure in the forth section. Finally, we present our results and then conclude.

2 Background

2.1 Air pollution or air pollutants?

Air pollutant concentrations are highly correlated in the urban setting but air pollution is without doubt multidimensional in its nature and consequences. The air we breathe contains particulate matter of various sizes and various gases, which may affect differently our health. In this paper, we consider the air pollutants which gather the strongest evidence according to [WHO \(2018\)](#): PM, O₃, NO₂, SO₂ and CO. These are pollutants in WHO's "Group 1", which "should be

⁷For instance, [Deryugina et al. \(2019\)](#) observed this finding in models controlling for PM_{2.5} and ozone when studying all-cause mortality in the U.S. ≥ 65 population.

considered of greatest importance in the process of updating the WHO Air Quality Guidelines”. In terms of regulation, the pollutants considered in this paper have been the main focus of international, national and local air quality standards - being therefore monitored as part of policy packages.⁸ Following the history of policy packages which first focus on industrial pollution and subsequently on traffic-related pollution, pollutant concentrations have known distinct trends. For instance, emissions of SO₂ and CO have been drastically cut in the last decades.⁹ Contrary to the other pollutant concentrations displaying negative trends in most U.S. and European cities (SO₂, CO, PMs, NO_x), ozone concentrations are not at all decreasing. Curbing population exposure is a challenge as it is not emitted directly by human activities, but formed from emitted pollutant precursors (e.g. see [Deschenes et al. \(2017\)](#)). Although non exhaustive in air quality potential hazardous substances, this paper focuses on the long-regulated and high-stake main pollutants - including PM_{2.5}.

Sharing some common sources leads naturally to strong correlations between pollutants and paves the way to approximating air pollution as a unidimensional phenomenon. We here provide some background information on the pollutants sources, gathered from the CITEPA¹⁰ 2019 report on French emissions from 1990 to 2017. In 2017, emissions of SO₂ were for 50% coming from the manufacturing industry, for 25% from the energy industry and for 25% from the building/housing sector. Comparatively, the share of emissions from the transport sector is negligible (< 2%). In contrast, the primary source of NO_x is the transport sector (63% of emissions). The transport sector originates as well particulate matter (31% of emissions of PM₁₀) and carbon monoxide (17% of the emissions of CO). The primary source of emissions of CO and PM_{2.5} remains the housing/building sector (respectively 45% and 50% of emissions). CO and PM_{2.5} are produced as well by the manufacturing industry sector (about 30% of emissions

⁸In the U.S. Clean Air Act of 1970, mandatory air quality standards were set for sulfur dioxide, nitrogen oxides, carbon monoxide, ozone and particulate matter (SO₂, NO_x, CO, O₃, PM₁₀). In the E.U. in 1980, a directive first set air quality limits and guide values for sulfur dioxide and particulate matter (PM₁₀). The 2008 European directive “on ambient air quality and cleaner air for Europe” unifies successive pollutant-specific directives and requires member states to guarantee that limit values shall not be exceeded. In this directive is first introduced a standard for PM_{2.5} (only on the yearly average concentration though), and gathered standards for SO₂, CO, NO₂, O₃, PM₁₀ (for within-day average concentrations along various duration) but also for lead and benzene. Data on these pollutants are very scarce or nonexistent over our time period and cities.

⁹Between 1990 and 2016, sulfur oxide and carbon monoxide emissions dropped respectively by 91 % and 69 % in the EU (European Union emission inventory report 1990-2016, EEA report, 6/2018).

¹⁰Technical Reference Center for Air Pollution and Climate Change, State operator for the French Environment Ministry, the Citepa meets reporting requirements for air pollutants and greenhouse gas emissions from France in different inventory formats. <https://www.citepa.org>

of CO and PM10 come from manufacturing industries).

Yet, approximating air pollution by a unidimensional phenomenon may be dubious. In particular, ozone tends to be anti-correlated with other pollutants in urban settings (Munir et al., 2012). NO₂ is the precursor of O₃ in the reaction $NO_2 + O_2 \leftrightarrow NO + O_3$. There are at least two effects explaining the anticorrelation of O₃ with the other pollutants: NO₂ disappears in the process of producing O₃ in a slow reaction (to a lesser extent it is also the case for CO). Additionally, primary pollutant NO is unstable and reacts quickly with O₃, and it is usually produced in conjunction with PMs by traffic. The latter is known as the urban decrement: primary pollution can at first reduce the concentration in O₃ at the local level.¹¹ The data section complements and illustrates how pollutant concentrations are interrelated.

Given the multidimensional and intricate aspect of air pollution, the need for novel research to better characterize the health effects of multipollutant exposures has been discussed for instance in Mauderly et al. (2010), Vedal and Kaufman (2011) or Johns et al. (2012). While multi-pollutant approaches are widely regarded as desirable, the challenges of implementing them are vast (Dominici et al., 2010) and most of existing literature is based on single-pollutant models. Because some pollutants are highly correlated, the results of many regression models become highly unstable when incorporating more than one pollutant, and very often imprecise. In the quasi-experimental literature in economics, while this challenge is acknowledged in many studies, only partial solutions have been found. Schlenker and Walker (2015) exploits the differential impact of their airport taxi time instruments interacted with wind speed on CO and NO₂ to conclude that CO is responsible for the majority of the observed increase in hospital admissions, while both pollutants in isolation would appear as significantly impacting emergency admissions. The instruments are weak for ozone which is therefore excluded of the analysis. Arceo et al. (2016) study how infant mortality in Mexico City is related to levels of CO and PM10. While not individually significant in the two-pollutant model, the two pollutants are jointly significant in predicting infant mortality and both significant in isolation. They construct a pollution index using the principal components method in order to generate a single endoge-

¹¹A simple way to explain it from Munir et al. (2012): *At the local level freshly emitted nitric oxides (NO) produced by road-traffic react with ozone molecules and produce nitrogen dioxides (NO₂). Hence road-traffic provides a local sink for ground level ozone resulting in ozone concentration in urban areas being lower than the surrounding rural areas. This phenomenon of lower ozone concentration in urban areas is referred to as ozone urban decrement.*

nous variable that captures information on fluctuations of both pollutants. One notable exception is [Deryugina et al. \(2019\)](#), who estimate a three pollutant model for the impact of PM_{2.5}, CO and O₃ on the three-day mortality rate of Medicare beneficiaries and even a five pollutant model in the appendix, adding SO₂ and NO₂ when studying one-day mortality. They find that their IV estimate for PM_{2.5} impact on mortality is robust for simultaneously instrumenting the other pollutants. Their results on mortality are broadly comparable to ours, although less precise (in particular, SO₂ appears with a large but insignificant coefficient), while we rely on a much smaller sample. They also observe that ozone tends to spuriously appear as decreasing the mortality rate given its negative correlation with other pollutants. Our study offers a proposal to disentangle the correlated variations between five pollutants with a method specifically tailored to address the issue, which is applied to all-age morbidity and mortality outcomes.

2.2 Exposure to Pollutants and Health

An extensive literature shows statistical association between air pollution and various health outcomes. Air pollutants are able to accumulate in or pass through lung tissues, thereby triggering or enhancing the severity of respiratory infections. Fragilizing the defense of the respiratory tract, they may favor acute inflammation. Further than endangering respiratory health, air pollution has been shown associated with the onset of acute cardiovascular events (e.g. cardiac arrhythmia, emergency room admissions), including accelerations of the heart rate ([Cakmak et al., 2014](#)). Although widely consistent across a number of settings, most of the existing observational studies refer to correlative evidence.

In the recent literature, concerns rose over the potential endogeneity of environmental exposure which lead environmental economics to an increasing focus on causal methods ([Deschenes and Meng, 2018](#)). Air pollution levels reflect economic activity and air pollution exposure depends on how individuals sort across the territory ([Banzhaf and Walsh, 2008](#)) which leads to bias in naive regression approaches. With longitudinal data, some of this bias can be accommodated with fixed effects (seasonal variation, location-specific heterogeneity) with estimation relying on within-location temporal variations - e.g. [Burkhardt et al. \(2019\)](#). They would capture for instance cross-sectional and time invariant location-specific population characteristics, such as (the invariant component of) socio-economic background. Yet, because human behavior is intrinsically tied to air pollution, important confounders might be forgotten. In our daily observational setting, one potential issue may arise from population daily movements in and out

the urban areas we consider. Population presence is a driver of air pollution (through e.g. car or heater uses) and a prerequisite to hospital visits, death counts, number of crime, accidents... Even more, pollution may just reflect traffic jam or economic upsurge, associated with stressful conditions or extended hours-of-work which may not be unrelated to population health. These mechanisms caution against approaches relying on solely absorbing local seasonal fixed effects when population movements or traffic circumstances are subject to unexpected shocks and justify an IV approach.

Yet, in spite of their limitations, correlative evidence has been broadly confirmed by the quasi-experimental literature. The first findings of the quasi-experimental literature were on the link between air pollution and infant health, birth outcomes and infant mortality (see [Currie et al. \(2011\)](#) and references therein). More recently, evidence on the morbidity and mortality effects of air pollution in other population groups have been added. [Deryugina et al. \(2019\)](#) focus on the senior U.S. population (65 and older) and address the difficult task of measuring the substantial number of life-years lost due to premature deaths induced by air pollution (PM2.5) within this population. [Schlenker and Walker \(2015\)](#) provide a thorough analysis of respiratory and heart-related hospitalizations attributable to air pollution (CO) in communities living near California airports. [Schwartz et al. \(2016\)](#) find a causal association between local air pollution (PM2.5) and the number of daily deaths in the city of Boston.

2.3 Altitude weather variables for causal inference

In the quest for isolating quasi-random variations of air pollution - that is variations plausibly orthogonal to specific-population sorting or confounding human activities, weather instruments gathered particular attention. Indeed, atmospheric mechanisms impact air pollutant concentrations on the ground, while being arguably disconnected from human activities when relative to altitude phenomenon.

Our first instrument is the height of the planetary boundary layer. The planetary boundary layer (PBL) is the part of the atmosphere that is directly and strongly influenced by the presence of the surface of the earth.¹² Pollutants are trapped within this vicinity of the earth.

¹²The air near the ground is indeed sensitive to friction forces with the surface. These forces become negligible in the upper layers where wind circulation is global (the free atmosphere). The planetary boundary layer height is usually defined by the discontinuity in one or several atmospheric variables.

Roughly, the higher the height of the planetary boundary layer (PBLH), the larger the air volume available for pollutants, and the lower the concentration.¹³ Concentrations are expected to be loosely proportional to the inverse of the height (IBLH) due to a dilution effect on the vertical axis, a relationship which we can check on our data. PBLH varies according to various factors. The height of the planetary boundary layer responds to heating flux between the sun and the earth and therefore displays a diurnal pattern. While displaying a seasonality related to surface weather, PBLH may also move under unpredictable large-scale air movements. The planetary boundary layer height reacts to *subsidence*, which brings the top of the layer downward in a high pressure diverging area. It may also be modified when a horizontal movement of cold air brings it under a warmer layer of air (*frontal* inversion at the top of the planetary boundary layer).¹⁴ If some of these phenomena do have a seasonal nature and are partially related to ground-level weather, there is no reason to expect that health would be affected by these phenomena *conditional* on seasonal and ground-level weather conditions. This makes PBLH a strong candidate for instrumentation.

Another distinctive potential instrument is the occurrence of a thermal inversion. Thermal inversions have been used by other authors to instrument air pollution e.g. on mortality in developing countries' cities, [Arceo et al. \(2016\)](#) and closer to us, [Jans et al. \(2018\)](#). Planetary boundary layer height is often defined by the presence of a thermal inversion at the top: the temperature, which usually decreases with height, sharply increases at the top of the PBL.¹⁵ A thermal inversion acts as a lid over the air motion beneath, because an air parcel which is cooler than its environment tends to move down. Its role over pollutant concentrations is widely acknowledged. Thermal inversions are thus closely related to boundary layer height (during the day, it is a thermal inversion that defines the boundary layer height) but they may be multiple and varying in strength within the boundary layer height. During a thermal inversion, polluted air is trapped beneath the inversion height (a warmer layer of air blocks the vertical movement). However, whereas thermal inversions may or may not happen (dummy variable), the height of the planetary boundary layer may always be defined (and is a continuous variable).

Therefore, aside from its height, other characteristics related to the planetary boundary layer

¹³See for instance [Levi et al. \(2020\)](#) for more details.

¹⁴See [Stull \(2016\)](#) for further details.

¹⁵During a thermal inversion, warmer air is held above cooler air; the normal temperature profile with altitude is inverted.

may influence directly pollutant concentrations: thermal inversions, but also winds characteristics (Deryugina et al., 2019, Anderson, 2019) which play a role in air pollution dispersion. Among altitude weather variables, there is a wide set of potential candidates which fulfill the conditional exclusion restriction.

3 Data

In this section, we describe the data sources which have all in common the following scope: the ten most populated urban areas in France over the 2010-2015 period. Table A.1 in Appendix reports the population, and Figure 1 the geographical location and extension of urban areas. The largest urban area is the Paris region where more than twelve million people live. Most of the other urban areas have about a million inhabitants. The urban areas are well spread out on the French territory.

Within these urban areas, many cities do have worrying air pollution levels. Figure A.1 shows the annual mean of particulate matter in municipalities belonging to the ten urban areas, compared to WHO guidelines. The vast majority of cities and in particular the most populated do not respect the guidelines for yearly means in 2014. For instance, Rennes, the smallest urban area in our sample, do not respect the guidelines relative to particulate matter of less than $2.5\mu m$. In the rest of the paper, we call “cities” the urban areas.

3.1 Atmospheric weather characteristics

Data. The altitude weather data come from the LMDZ model (Hourdin et al., 2006), an atmospheric general circulation model developed and maintained by the *Laboratoire de météorologie dynamique* (Z is for zoom).¹⁶ It simulates the full atmosphere over a 3D grid. The development of the model is tested and improved by comparison with atmospheric observations (field or satellite data). The model configuration used to obtain our dataset (stretched grid to zoom over France, with large-scale atmosphere dynamics constrained towards prescribed atmospheric conditions) has been successfully used to simulate a realistic meteorology consistent with observations at the daily time scale (Coindreau et al., 2007, Cheruy et al., 2013).

¹⁶More precisely, the data come from the LMDZOR version, the atmosphere component of the climate model IPSL-CM described in Dufresne et al. (2013) and used for IPCC reports. The LMDZOR version was prepared for the phase 6 of CMIP (coupled model intercomparison project). See <http://lmdz.lmd.jussieu.fr/> for a general presentation.

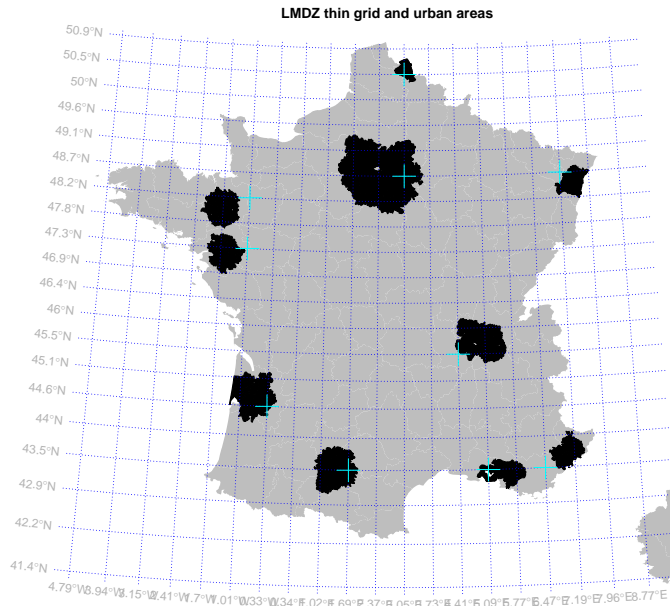


Figure 1: Geographic Location of the Ten Most Populated Urban Areas in France (black areas), LMDZ numerical simulation grid (blue dotted lines) and points representing the urban areas on this grid (cyan cross). (Source: Insee, 2010; LMDZ)

A large set of potential instruments. We were provided the output of an hourly reconstitution of the atmospheric variables for the 2010 to 2015 period along a grid ≈ 50 km x 50 km in which cities are located (the model is used with a zoomed grid over France, see Figure 1). Many variables are present, most importantly, PBLH; but also along a vertical grid parameterizing altitude through pressure levels, wind characterizations (direction, strength), humidity, temperature, and altitude corresponding to the pressure levels. In total, the atmosphere is represented with 79 layers indexed by pressure levels. The first layer corresponds to the layer of air between the surface and pressure level 101,2 kPA (≈ 200 m above the surface) and the 79th layer reaches 1,5 Pa (≈ 78 km above the surface). In particular, the vertical profiles of temperature (temperature gradient) allow to reconstitute thermal inversions indicators within the boundary layer. We acknowledge that measurement errors in the instruments are not taken into account in our approach although likely given that ultimately, these variables are the output of a model.

From the model output which is at the hourly by urban area level, we build an extensive set of 328 instruments at the daily by urban area level to match health data. We first build a set

of variables related to our four main instruments: the planetary boundary layer height PBLH, its inverse IBLH, thermal inversion (TI) presence and thermal inversion strength. PBLH and IBLH are directly obtained at the hourly level from the model output. We compute thermal inversions at the hourly level from temperature altitude profile by considering the first and the ninth layers (ground-level and at 98.1 kPa \approx 450m) similarly to [Jans et al. \(2018\)](#) and define a thermal inversion when ground-level temperature is the lowest, and thermal inversion strength as the difference of these two temperatures.¹⁷ For these four hourly instruments, we build daily average and daily measures specific to six moments of the day (0 to 4 a.m., 4 to 8 a.m., \dots , until 8 p.m. to midnight), that is $(1 + 6)$ statistics times for each 4 variables, hence 28 dimensions. We consider different moment-of-the-day in an attempt to capture differential impacts according to when pollution is mostly emitted (e.g. a thermal inversion during the morning circulation peak is expected to be more damaging than when occurring during the night). As IBLH is expected to provide the maximal explanatory power, we also add interactions of within-day averages of IBLH with urban area indicators to capture potential geographical variations of the phenomenon, that is 10 cities \times 6 moments of the day, 60 dimensions in addition.¹⁸ To this set of 88 instruments, we add daily averages of other model output variables varying through the 79 layers: zonal wind (wind strength when blowing in the direction west to east, u), meridional wind (wind strength blowing in the direction south to north v), total wind strength $\sqrt{u^2 + v^2}$ and altitude of pressure levels (altitude corresponding to each layers which are defined by fixed pressure levels) measured from the 20th to the 79th layer (from 1,5 to 78 km above the surface) - that is 240 dimensions: 60 layers \times 4 variables.¹⁹ Table [B.1](#) in the appendix recap the instruments. We exclude the 19 first layers of the model, below altitude \approx 1,5km to prevent using close-to-the-surface weather as an instrument. Indeed, altitude weather variables are arguably less likely than surface variables to have a direct impact on health. Table [1](#) reports descriptive statistics on 16 of these instruments providing an overview of the substantial daily variation,

¹⁷We also experimented with more complex measures of inversion such as defining a thermal inversion when at least 50% of the layers between 101,2 kPa (\approx 200 m) and 89,7 kPa (\approx 1,2 km) have temperature above temperature at ground level (in the lower layer). As it does not make differences in the results, we resort to the simplest measure.

¹⁸We also tried to add the same set of 60 interactions with thermal inversions but it did not improve the precision and kept the result similar.

¹⁹In a previous version of this work, we also included altitude humidity in the set of instruments with the agnostic view that any weather variable measured in high altitude could enter as a credible instruments. The exception was altitude temperature: misspecification in surface temperature controls could become an issue as surface temperature is known to have a direct impact on health. Given that there is evidence of a direct impact of humidity on mortality ([Barreca, 2012](#)) we now treat humidity as temperature in our specification: it only appears as a surface-level control and not as an instrument. In the appendix, we nonetheless report our main results when we amplify or restrict the set of instruments.

including between layers.

3.2 Ground-level weather data

Weather conditions play a key role in human activity and air pollution formation, but also directly on health (Deschênes and Greenstone, 2007). Plus, ground-level weather data is likely correlated to altitude weather data, so that it is important to condition on ground-level weather in our regression, the assumption being that high altitude atmospheric variations are exogenous with respect to health, except through pollution, conditional on ground-level weather controls. We hence consider a full set of weather conditions. Data come from Météo France and are available on an hourly basis for our ten urban areas. We consider six weather parameters: temperature, rainfall, wind speed, sunshine exposure, presence of snow and humidity. Measurement stations are located at nearby airport,²⁰ except for Paris, where the measurement station is located in a garden in the center of Paris urban area.

In our regressions, ground-level weather are specified as polynomials of order two for daily temperature, rainfall, humidity and wind strength; and linear controls for sunshine and for the presence of snow, variables described in Table 1. Given their importance for our identification strategy, we perform a number of robustness to their specification.

3.3 Pollutant data

Air quality is measured by regional associations called AASQA (*associations agréées de surveillance de la qualité de l'air*), which are grouped in a national federation called ATMO France. The Ministry of Environment delegates the surveillance of “regulated” pollutants to the AASQA. They operate numerous air quality measurement stations all over France. We consider the stations located in the 10 more populated urban areas. We focus on a rich set of air pollutants: the 6 pollutants that are widely available on an hourly basis are carbon monoxide (CO), particulate matter of less than 2.5 micrometers (PM2.5), particulate matter of less than 10 micrometers, nitrogen dioxide (NO2), ozone (O3) and sulfur dioxide (SO2). We usually have data for several monitoring stations per urban area,²¹ which we average at the urban area and

²⁰Specifically for sunshine exposition in Lille, we use the measurement station in Lillers, nearby Lille, as this parameter was not available in Lille-Lesquin airport station over the whole studied period.

²¹We have at least one monitoring station for each pollution in each urban area.

Table 1: Descriptive Statistics

<i>Daily Observations, Ten Most Populated Urban Areas in France, 2010-2015</i>				
	D1	Mean	D9	# Missing Values
<u>Pollutant concentrations ($\mu\text{g}/\text{m}^{-3}$)</u>				
PM2.5	6.5	16.5	30.1	5,451
PM10	12	25.8	43.3	7,389
NO2	17.1	37.4	59.8	6,790
O3	18.7	51.5	81.6	5,661
CO	179.7	407.6	691.9	7,525
SO2	0	1	2.8	6,792
<u>Air Pollutant Index</u>				
Air Pollutant Index	-1.4	0	1.7	0
<u>Emergency admissions per 100,000 inhabitants</u>				
All admissions	8.7	14.6	20.6	0
Cardiovascular Diseases	0.9	1.6	2.4	0
Respiratory Diseases	0.5	1.4	2.1	0
Digestive Diseases	0.8	1.4	2.1	0
<u>Mortality Rate per 100,000 inhabitants</u>				
Mortality Rate	1.4	2.1	2.9	0
with at least one cause from:				
- Cardiovascular Diseases	0.5	0.9	1.3	0
- Respiratory Diseases	0.2	0.5	0.8	0
- Digestive Diseases	0	0.2	0.4	0
<u>Ground-level Weather Variables</u>				
Precipitations (mm)	0	0.1	0.3	93
Temperature (degree Celsius $^{\circ}\text{C}$)	4.4	13.2	22	15
Wind Strength (m/s)	1.7	3.5	5.7	98
Relative Humidity (%)	55.3	73.1	89.2	19
Sun Light	10.7	54	106.9	253
Snow (Dummy)	0	0.021	0	0
<u>Subset of Instruments From Altitude Weather</u>				
PBLH (m)	399.5	889.8	1,408	0
IBLH ($1/\text{m} \times 1,000$)	0.7	1.4	2.5	0
Thermal Inversions (# Hours during the day)	0	0.2	0.5	0
Thermal Inversion Strength ($T_{up} - T_0$, $^{\circ}\text{C}$)	-2.5	-1.2	0.4	0
Zonal Wind (Layer 20) (m/s)	-4.1	3.5	12	0
Zonal Wind (Layer 40) (m/s)	-5.8	12.9	32.1	0
Zonal Wind (Layer 60) (m/s)	-13.3	8.2	36.6	0
Meridional Wind (Layer 20) (m/s)	-8.2	-0.4	7.5	0
Meridional Wind (Layer 40) (m/s)	-23.5	-3.1	15.9	0
Meridional Wind (Layer 60) (m/s)	-7.2	-1.3	3.6	0
Total Wind Strength (Layer 20) (m/s)	2.5	8.2	15.3	0
Total Wind Strength (Layer 40) (m/s)	8	22.5	38.9	0
Total Wind Strength (Layer 60) (m/s)	4.5	17.5	38.6	0
Altitude Pressure of Layer 20 (m)	1,350	1,485.5	1,758.1	0
Altitude Pressure of Layer 40 (m)	9,793.8	10,123.7	10,423	0
Altitude Pressure of Layer 60 (m)	28,157.7	28,777.7	29,416.7	0

Total Observations

21910

Sources: AASQA, ATIH, Insee, Météo France, LMDZ.

daily level on a constant set of monitoring stations. Table 1 presents descriptive statistics on the sample.

To gauge the impact of aggregated ambient air pollution, we create a pollutant index with a principal component analysis (PCA) over the 6 standardized pollutants concentration and keep the first component as the pollution index.²² As we have missing values in pollutant concentration, the PCA is combined with an EM algorithm to deal with missing values (Josse and Husson, 2012). The index is therefore available even though one or several pollutant concentrations are missing (keeping observations for which all six pollutants are observed leads to drop 80% of the sample while most of the time only one pollutant measurement is missing). We do not use the normative indexes because they are designed as communication tools and consist in a large simplification of the information.²³

Table 2: Correlations Between Pollutant Concentrations

	Air Pollution index	PM2.5	PM10	NO2	O3	CO	SO2
Air Pollution index	1	0.85	0.82	0.77	-0.47	0.77	0.42
PM2.5	0.85	1	0.83	0.40	-0.32	0.46	0.27
PM10	0.82	0.83	1	0.53	-0.14	0.40	0.26
NO2	0.77	0.40	0.53	1	-0.22	0.69	0.24
O3	-0.47	-0.32	-0.14	-0.22	1	-0.40	-0.08
CO	0.77	0.46	0.40	0.69	-0.40	1	0.22
SO2	0.42	0.27	0.26	0.24	-0.08	0.22	1

Source: AASQA and Authors computations.

Table 2 shows how pollutants are correlated. Two important points should be noted for what follows. First, PM2.5 are a subsample of PM10 (60 to 70% of PM10 particulates are PM2.5 particulates according to Airparif).²⁴ To preview our results, we will not be able to disentangle their effect separately as we will find no clear distinction in their response to our instruments. Second, O3 is anticorrelated with all pollutants, in particular to its precursors NO2 and CO. On average, high levels of nitrogen oxides are associated with low levels of ozone. This

²²Arceo et al. (2016) use a similar index approach when considering the joint effect of PM10 and CO.

²³In the decree of the 22 of July, 2004, the air quality indexes are presented as a communication tools, a simple qualitative information, which should be an integer between 1 and 10. It is specified that they are not meant to inform public action. In addition, the index design is by now old and has been questioned - its design is currently being updated to be aligned with the European Air Quality Indexes, which has only five classes.

²⁴Bilan de la qualité de l'air 2017.

emphasizes the multi-dimensional aspect of air pollution, which should ideally not be treated as a whole. The dynamics of O₃ is singular as it is a secondary pollutant, reacting with the other pollutants (see section “Background”). These elements emphasize how ambient air pollution is multifaceted. Notably, if there exists an air pollution common component, ozone tends to vary in opposition.

3.4 Daily health data

The first data set is obtained from the ATIH (*Agence Technique de l’Information Hospitalière*) that gathers an administrative and exhaustive database which records all admissions in public and private hospitals, the PMSI (*Programme de médicalisation des systèmes d’information*). Its primary use is to compute hospitals’ funding based on their activity. By final diagnostic and by urban area in which the hospital is located, we were provided the daily count of emergency admissions. Further, this information breaks down by age groups defined as 5-years breakdown (0-4, 5-9, up to 75-79 plus over 80). More precisely, an emergency admission is an entrance through the hospital emergency unit that led to an admission from patients coming from their residence (i.e. not transferred from another hospital) or from public space.²⁵ Therefore, programmed admissions, long-term and recurring care are excluded. The diagnostic used here is coded at the end of the patient stay. It represents the main diagnostic which gave rise to the highest care resources. We divide the daily count of admissions by the age-range and urban-area corresponding population (2013 legal population produced by INSEE, the French national statistical office). In the regression, our variable of interest is the emergency rate of admission per 100 000 inhabitants (of the corresponding age group, when applicable).

In addition, we consider two sources for mortality rates: INSEE’s data on civil registry records and Inserm’s data CépiDc (Epidemiological Center on the Medical Causes of death) on death causes. Each death is recorded in both data sets, with civil information but no medical information in the first while the second records medical information but is anonymous. From the civil registry as produced by INSEE at the municipality level, we observe for each urban areas mortality rates by age groups. From the extraction of CépiDc, we observe for each urban

²⁵When due to hospital organization, emergency room is the main entry point, doctors should not use the code “emergency” systematically but only when the individual situation in the views of the patient, his relatives or his general practitioner is an emergency.

area mortality rates related to either cardiovascular or respiratory diseases,²⁶ but we do not observe age. After similarly normalizing with the legal population, our variable of interest is the mortality rate per 100 000 inhabitants. On average in our sample, there are 2.1 deaths per 100,000 inhabitants a given day, 0.9 related to at least one cardiovascular cause and 0.5 related to at least one respiratory cause, 1.4 emergency admissions for respiratory diseases and 1.6 emergency admissions for cardiovascular diseases (Table 1).

4 Empirical strategy

4.1 The causal damages of air pollution: preliminary evidence.

In this section, we provide basic preliminary evidence on the detrimental impact of air pollution on health by relying on isolated instruments. We show how these instruments are strong predictors of air pollution. We make a case empirically for the need to rely on instrumentation for eliminating confounders. We caution against single-pollutant models when instruments are far from being pollutant-specific.

Eliminating confounders. In this section, we empirically evidence that air pollution is intrinsically linked with some observed human activities, thereby suggesting that potential confounders might be plenty and that instrumentation is required. We also show that the “unexpected”²⁷ component of our instruments is not related to human activity as captured by various proxies. As an example of a potential confounding effect, air pollution could capture the population presence in the urban area (through e.g. car/heaters use, congestion, polluting economic activities). Inbound and outbound trips variations may therefore correlate with air pollution variations. For instance, mobile phone data have been used to infer urban emissions inventory in the environmental literature (Gately et al., 2017). Hospital emergency admissions typically increase with the population at risk (e.g. touristic areas tend to have a boom in emergencies admissions correlated with inbound tourism). Both statements if true entail a spurious correlation between air pollution and hospital emergencies admissions through a simple population volume

²⁶That is, death events which contains at least one cause encoded with “I” or “J” from CIM-10 chapters (the same codes are used to categorize emergency admissions).

²⁷Unexplained by other covariates and usual seasonal patterns

effect.²⁸

To empirically assert this claim, we exhibit correlations between various proxies of population presence and on one hand air pollution and on the other hand hospital emergency admissions. We regress the PCA-derived index of air pollution Y (or the rate of emergency admissions) over dummies indicating whether the date is a bank holiday, belongs to an holiday, or to an extended week-end (a bank holiday adjacent to a week-end) with a wide set of seasonal and weather controls. In addition, we include the hostel occupancy rate to capture “unexpected” visits in the urban area. In our data, hostel occupancy is on average 69% on Tuesday and Wednesday but 50% and 55% on Friday and Saturday, suggesting that hostel occupancy is not only holiday-induced but also driven by business travel hence economic activity. Denoting these proxy for population volume as V , the regression writes

$$Y_{ct} = V'_{ct}d + X'_{ct}b + \eta_{d,c} + \gamma_{my,c} + \epsilon_{ct} \quad (1)$$

$\eta_{d,c}$, $\gamma_{my,c}$ are respectively day-of-the-week, and month-year fixed effect which are specific to the urban area to capture usual pattern of pollution in our period. We additionally introduce a wide set of weather controls X_{ct} specified as polynomials of order two for temperature, rainfall, humidity and wind strength; controls for sunshine and for the presence of snow. Table 3 reports the result of this regression for four outcomes. First in columns (1) and (2), air pollution is lower on bank holidays or extended-weekend, when the outbound journeys are the highest (but also when the economic activity tends to decrease). When the urban area experiences a higher number of inbound trips, as captured by hostel occupancy, air pollution is higher as well. Strikingly, the direction of the correlations is the same when the outcome is instead the total hospital emergency admissions in columns (3) and (4). The latter suggests that hospital emergencies react to human activities such as day trips (or reduced staff) at the daily level, in much the same way as air pollution. This simple exercise emphasizes that air pollution and hospital admissions are intrinsically linked with human activity²⁹ and confounders are possibly numerous. To eliminate concerns about spurious correlations arising just because air pollution is a very good proxy for any human activities, one may use instrumentation. Columns (5), (6), (7) and (8)

²⁸This is in general acknowledged in studies controlling for holidays dummies in regressions. Nevertheless, big cities do experience daily-level population variations which are not holiday-induced on a regular basis (e.g. business travels linked to economic activity, visitors of particular events such as festivals...).

²⁹For instance, air pollution reacts at daily frequency to strike events (Bauernschuster et al., 2017), which may have unintended health consequences (Adda, 2016, Godzinski and Suarez Castillo, 2019).

show that provided that we have controlled correctly with ground-level weathers and seasonal fixed-effects, the residuals variations of our instruments (IBLH and thermal inversions) are not linked with our proxy for human activities, supporting (but not demonstrating) the exclusion restriction statement.

Table 3: Air pollution, Hospital Emergency Admissions and Human Activities

	<i>Dependent variable:</i>							
	Air pollution		All hospital emergencies		Inverse of PBL height		Thermal inversions	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Hostel Occupancy		0.279*** (0.067)		0.507*** (0.107)		0.017 (0.061)		-0.027 (0.018)
Bank holiday	-0.282*** (0.030)	-0.248*** (0.030)	-1.310*** (0.074)	-1.249*** (0.074)	-0.007 (0.027)	-0.005 (0.029)	0.005 (0.007)	0.001 (0.007)
Holidays	-0.067** (0.029)	-0.041 (0.029)	-0.142*** (0.032)	-0.095*** (0.032)	0.010 (0.022)	0.011 (0.021)	-0.0002 (0.006)	-0.003 (0.006)
Extended W-E	-0.058** (0.027)	-0.070** (0.027)	-0.107** (0.049)	-0.129*** (0.049)	-0.016 (0.026)	-0.017 (0.026)	-0.007 (0.007)	-0.006 (0.007)
Observations	21,459	21,459	21,459	21,459	21,459	21,459	21,459	21,459

All regressions includes month-year and day-of-the-week fixed effects, interacted with city fixed effects; and weather controls. Standard errors are clustered at the month-year-city level. Hotel occupancy is derived from the INSEE frequentation survey (See [Godzinski and Suarez Castillo \(2019\)](#) for a description of the data). Hospital emergencies aggregate all emergencies (source: PMSI). Significance: *p<0.1; **p<0.05; ***p<0.01

Instrumenting Air Pollution Variations. Among our set of potential instruments, the inverse of planetary boundary layer height (IBLH) stands as a natural choice given the well-identified physical mechanism which ties this instrument to air pollution. Figure A.2 shows how an increase from IBLH leads to a close to linear increase of 5 pollutant concentrations out of 6, as expected from a vertical dilution effect where pollutant concentrations would be inversely proportional to boundary layer height.³⁰ Figure A.2 clearly shows how all pollutant concentrations except O3 are driven upward when the boundary layer height goes down, conditional on weather controls and city-level temporal patterns of pollution. Therefore, we see the limitation of using IBLH to instrument a single pollutant: it triggers for sure an increase in most of the air pollutants, but a decrease in ozone. This is a form of exclusion restriction violation: as

³⁰These figures are built upon the unusual component of both variables to emphasize that the link between both variables does not arise merely from seasonality. Nevertheless, without controls and seasonal fixed effects, the same patterns can be observed (see Figure B.1 in Appendix).

these instruments are not pollutant-specific they may capture complex variations in air pollution mixture.

In fact, the very same limitation applies to the instrument built on thermal inversion. We report in Table 4 the results of the following regression - which mirrors Figure A.2:

$$P_{ct} = Z_{ct}\eta + X_{ct}b + \alpha_{dc} + \beta_{myc} + \epsilon_{ct} \quad (2)$$

P_{ct} is either a given pollutant concentration or a pollution index, X_{ct} the aforementioned ground-level weather controls and seasonal fixed effects (day-of-the-week \times city and month-year \times city). In addition to the inverse of the planetary boundary layer height $IBLH_{ct}$, we test as well the number of hours with thermal inversion during the day TI_{ct} . All pollutants except SO2 respond strongly to both instruments. O3 responds negatively, in the opposite way compared to the other pollutants, probably because of the increases in concentration in nitrogen oxides. For the sake of comparison between mono-pollutant models and multi-pollutant models which are hindered with more missing values as they require to observe all pollutants at each date, we report the results for two distinct samples, the full sample (A) for which a pollutant index can be computed, and the restricted sample (B) where PM2.5, O3, CO, SO2 and NO2 are all observed, which is the focus in the rest of the paper. The second panel of Table 4 reproduces the regression from equation (2) but with normalized air pollutant concentrations and instruments so as to characterize the respective shock triggered by both instruments. We observe that a standard deviation in IBLH triggers relatively more variations in particulate matters and carbon monoxide. In contrast, the number of hours under thermal inversion triggers relatively less negative variations in ozone compared to IBLH and more in sulfur dioxide and nitrogen oxides. If each pollutant has a specific health-impact and is more or less affected by the instrument, the composition of the instrument-induced air pollution shock would be reflected in the health impact estimates.

Reduced form evidence. Although triggering a complex shock, both instruments induce a strong and plausibly exogenous deterioration in air quality. We resort to the following regression to provide preliminary evidence on the causal detrimental effect of air pollution on health:

$$R_{ct} = Z_{ct}\delta + X_{ct}d + a_{d,c} + e_{myc} + \nu_{ct} \quad (3)$$

with R the rate of admissions in emergency (or of deaths) per 100 000 inhabitants in urban area c and date t with the same set of weather controls and seasonal patterns. Importantly,

Table 4: Effect of the Inverse of Planetary Boundary Layer Height and of Thermal Inversions on Pollutant Concentrations.

	Pollution		Concentration ($\mu\text{g}/\text{m}^{-3}$)					
	index		PM2.5	PM10	NO2	O3	CO	SO2
	(A)	(B)						
$IBLH_{c,t}$	247.0*** (15.8)	249.2*** (26.7)	2,439.2*** (162.7)	2,567.9*** (198.1)	476.1*** (171.1)	-2,814.3*** (216.3)	31,079.0*** (3,753.2)	-31.5 (22.8)
$TI_{c,t}$	0.4*** (0.04)	0.4*** (0.1)	2.8*** (0.5)	3.5*** (0.6)	7.0*** (0.5)	-2.0*** (0.7)	24.8*** (8.4)	0.5*** (0.1)
Obs.	21,459	6,135	16,095	14,220	14,875	15,968	14,109	14,820
F-Stat	1050.2	291.5	610.1	432.9	324.6	292.8	352.0	49.18

	Pollution		Scaled Concentration					
	index		PM2.5	PM10	NO2	O3	CO	SO2
	(A)	(B)						
$IBLH_{c,t}$ (scaled)	0.23*** (0.01)	0.23*** (0.02)	0.18*** (0.01)	0.15*** (0.01)	0.02*** (0.01)	-0.09*** (0.01)	0.10*** (0.01)	-0.01 (0.01)
$TI_{c,t}$ (scaled)	0.10*** (0.01)	0.09*** (0.02)	0.05*** (0.01)	0.05*** (0.01)	0.07*** (0.01)	-0.02*** (0.01)	0.02*** (0.01)	0.06*** (0.01)
Obs.	21,459	6,135	16,095	14,220	14,875	15,968	14,109	14,820
F-Stat	1050.2	291.5	610.1	432.9	324.6	292.8	352.0	49.18

IBLH (resp. TI) stands for the Inverse of Planetary Boundary Layer Height averaged by date and urban area (resp. the number of hours with a Thermal Inversion by date and urban area). All regressions includes month-year and day-of-the-week fixed effects, interacted with urban-area fixed effects, and weather controls. Standard errors are clustered at the month-year-urban-area level. The F-statistics corresponds to the hypothesis of joint nullity of the two instruments. Significance: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

our exclusion restriction condition is that altitude weather variables influence air pollution at given ground-level weather, and should not impact health directly. Our identification strategy only mobilizes variations within month-year-city cells through a flexible and wide set of fixed effects. That is we only compare admissions or mortality and altitude weathers between similar working days in the same city a given month. This flexible set of fixed effects already captures large amount of ground-level weather variations.³¹ The first order threat to identification is the potential confounding effect of ground-level weather variables, which are both correlated to our instruments and may have a direct impact on health, in particular, ground-level temperature and humidity. These variables are therefore not used in the instrument set, although present in LMDZ model output. To limit concerns about spurious findings, we introduce a number of weather controls, introduced as polynomial in temperature, humidity, rainfalls, wind strength, sunlight and snow, and perform a number of tests when altering or amplifying this set. We show however that once accounted for month-year-city fixed effects, the direct and very short-term link between mortality or emergencies and ground-level weather is in fact rather weak.

Table A.2 in Appendix shows that indeed, both instruments are associated with an increase in both respiratory and cardiovascular mortality rate. As for emergency admissions, respiratory emergencies respond positively to thermal inversions and cardiovascular emergencies to the inverse of planetary boundary layer height. Under the assumption that these effects are mediated only by the instrument-induced variations in air quality, these results provide strong evidence on the short-term detrimental effect of air pollution on health. As a falsification test, we add digestive diseases as a common emergency admissions, and another potential cause of death. As expected, we find no effects of the inverse of the boundary layer nor of thermal inversions for these outcomes.

4.2 Multi-pollutant models and optimal instruments

Instrument selection. Starting from the first stage equation 2, we expand the set of instruments under consideration. From the very large set of 328 potential instruments, we perform an optimal selection following Belloni et al. (2012, 2016). Optimal selection should be understood as unveiling a true predictive power, not as an unprincipled over-fitting of the data at

³¹For instance, 82% (resp. 53%) of the variance of temperature (resp. humidity) in our sample is captured with month-year-city fixed effects. This aggregate level of analysis is often used to evidence the temperature-mortality link, and is not used in our analysis.

hand. The instrumental variable setting is particularly fitted for using prediction tools coming from the machine learning literature for traditional econometric identification. In a two-stage least square, the first stage can be thought of as a *prediction step in the service of estimation* (Mullainathan and Spiess, 2017). Model selection is performed thanks to the LASSO (Least Absolute Shrinkage and Selection Operator, Tibshirani (1996)). It introduces a penalization to the OLS objective, the errors’ sum of squares, by adding a scalar penalty multiplied by the l_1 -norm of the (possibly high-dimensional) parameter of interest. The solution has a limited number of non-zero coefficients, whose number depends on the penalty level: as such it performs model selection. In our setting, the high dimensional parameter is the effect of *many* altitude weather characteristics on pollutant concentrations.

To abstract from seasonality, we first take out the estimated seasonal fixed effects from any of the variables considered in the following equations: pollution, ground-level and altitude weather characteristics, health outcomes. Lower case letters designate residuals from linear regressions over month-year \times city and day-of-the-week \times city fixed effects. Selection on these effects is not appropriate: we want to maintain the conditional exclusion restriction and use identifying variations which do not come from mere seasonality. We therefore study all variables after partialling-out seasonal fixed effects, which boils down to a Frisch-Waugh transformation. This is the first step of the treatment required for panel data in such a setting (Belloni et al., 2016). The selection equation thus writes:

$$p_{kct} = w_{ct}\eta^k + x_{ct}b^k + \epsilon_{kct} \quad (4)$$

where k indexes pollutants, p_{kct} the residual variation in air pollutant k concentration at date t in urban area c , and w_{ct} is a high dimensional set of instruments built from the altitude weather variables³² and η^k is a high dimensional vector to be estimated. We formulate the assumption that η^k is at least approximately sparse, i.e. that only a “small” number of dimensions of this vector is non negligible. That is, only some of the introduced instruments variables do have a non negligible impact on pollutants’ concentration. The pollutant-specific set of selected instruments are derived from the following penalized minimization problem, for each k (for the

³²For IBLH, PBLH, and thermal inversion number of hours and strength, we build many possible functions of the variable at the city-date (c, t) level: averages over 24 hours; over 6 time windows of 4 hours and specific to the city. For IBLH we allow for various predictive patterns per city. We add daily average altitude variables in 60 altitude layers: altitude pressure, zonal wind, meridian wind and wind strength. The full set of instruments comprise 328 variables.

sake of readability, the index k is not mentioned):

$$\operatorname{argmin}_{\eta, b} \sum_{c, t} (p_{ct} - w_{ct}\eta - x_{ct}b)^2 + \lambda \sum_j \phi_j |\eta_j| \quad (5)$$

where j indexes the instruments set. In this step, there is no selection on ground-level weather controls. The parameter b is not penalized, to maintain the exclusion restrictions, for similar reasons to these applying to seasonal fixed effects.³³ Belloni et al. (2012) show how to choose “rigorously” the penalty λ and the instrument-specific penalty loadings ϕ_j to ensure asymptotic convergence and inference in a IV-setting where LASSO is used to select instruments in a first step. The unfeasible choice which guarantees favorable performance is to take for each j a penalty ϕ_j associated to η_j which dominates the noise by verifying $\frac{\phi_j}{N} > 2c|\frac{1}{N} \sum_c \sum_t x_{ct}\epsilon_{ct}|$, where N is the number of observations, and c a constant greater than 1 - which is possible by allowing ϕ_j to depend on x and ϵ . This condition ensures that the “regularization event” (correct selection) happens with high probability. In practice, as ϵ is unobserved, Belloni et al. (2012) suggest to use an iterative algorithm, first using conservative penalty loadings and then plugging estimated residuals and so on. Belloni et al. (2016) shows how to modify the penalty choice to take into account within-group dependence in ϵ - introducing a clustered version of the IV Lasso for panel data, which is used here. We start from routines developed in the *hdm* package implementation (Chernozhukov et al., 2016) and additionally implement selection by clustered-lasso and allow for unpenalized dimensions. To use the clustered-lasso version, we specify the level of clustering as the month-year-city level. This level of clustering stems from the choice of the unobserved heterogeneity specified as month-year-city fixed effects that are differentiated in the procedure and then define the level for clustering robust standard errors. However, in a classic IV setting, a natural choice of clustering would have been the city-level, to allow for arbitrary serial correlation within a given location across all dates. We can not define robust standard errors at these levels without strongly biasing downward the standard errors because of the small number of clusters. As a check for our main results inference, we compute wild cluster bootstrap standard errors at these alternative levels of clustering under the null hypothesis of no pollutant effect, following Cameron et al. (2008) who advocate this technique to tackle the issue of clustering with a small number of clusters.

³³However, seasonal fixed effects are somewhat too numerous to be forced into the model in the same way. Theoretical properties after selection from a high dimensional set of instruments, conditional on covariates, assume the later to be low dimensional.

The IV-Lasso method is attractive as complex relationships between altitude weather variables and air pollution may be recovered from the data, taking an agnostic and data-driven approach on the dimensions which should enter the first stage: which specific variables, measured at which hours-of-the-day, altitude layers or in which city? It avoids an ad hoc choice of variables and ensures that the selection is reproducible. At the same time, a strong first stage should improve the precision of our estimates. Other estimators could have been considered in a setting where the exclusion restriction applies to a high number of instruments, which are possibly interacting. We favored a Lasso selection because in our setting, not all instruments are expected to be worthwhile which fits precisely in the Lasso sparsity assumption.³⁴ In addition, with the selection step we can clearly identify, per pollutant, which instruments are active in the estimation.

For each pollutant, which instruments will be selected? We present selected instruments in Table 5 on the five-pollutant sample : each pollutant is regressed over the selected variables. After selection, we run OLS on selected instruments and the same ground-level weather variables controls. This is known as *post Lasso* estimation and alleviates the Lasso bias which shrinks point estimates toward zero. To compare the relationships with altitude weather between pollutants, all the variables are standardized in this table. Cluster-lasso with the rigorous penalty selects 3 instruments for CO, 3 for SO₂, 10 for O₃, 14 for NO₂ and 15 for PM_{2.5}. Different dimensions are selected, and when the same dimension appears for several pollutants, the sign and magnitude differ. Thermal inversions and planetary boundary heights are selected for all air pollutants. Leaving O₃ and geographical differences apart, all air pollutants respond positively to the following atmospheric phenomena: thermal inversion strength (as the temperature gradient is higher, the lid is more hermetic, and air pollutants cannot escape above), presence of thermal inversion, inverse of planetary boundary layer height (the lower the planetary boundary layer, the lower the volume available and the higher the air pollutant concentration). Selected characteristics of thermal inversions and planetary boundary layer, as the times of the day or the precise indicator considered, however differ among air pollutants. Wind patterns are almost exclusively selected for predicting PMs. For instance, altitude zonal wind coming from the west

³⁴On one hand, instead of having many weak instruments, which would favor estimators such as JIVE (Hansen and Kozbur, 2014), many of them are clearly strong. On the other hand, the regularization of the first stage with the LASSO could have been replaced by other forms of regularization, as presented in Carrasco and Tchuente (2015), to keep all the instruments active in the estimation. However, among our instruments, if some of them are known to be related to air pollution, some of them are likely weak or close to noise when it comes to form the conditional expectation of pollutants given the instruments, which suggest to favor a selection setting.

Table 5: Lasso Selection of Instruments for Predicting Pollutant Concentrations.

	PM2.5	CO	O3	NO2	SO2
Thermal inversions					
- #Hours between 0 and 4am				0.02 (0.01)	
- #Hours between 8 and 12am			-0.03*** (0.01)		
- #Hours between 8 and 12pm				0.03*** (0.01)	
- Strength between 0 and 4am	0.03* (0.02)	0.03** (0.01)		0.02 (0.02)	0.07*** (0.01)
- Strength between 4 and 8am				0.02 (0.02)	
Planetary Boundary Layer					
- Height Inverse (Day Average)	0.06** (0.02)		-0.01 (0.01)		
- Height Inverse (0-4 am)	0.08*** (0.02)			0.05*** (0.01)	
- Height Inverse (4-8 am)				0.01 (0.01)	
- Height Inverse (8-12 am)	0.003 (0.02)	0.06*** (0.01)			
- Height Inverse (4-8 pm)		0.11*** (0.02)	-0.05*** (0.01)	0.07*** (0.01)	
- Height (0-4 am)	0.001 (0.01)		0.07*** (0.01)		
- Height (8-12 am)			0.02 (0.02)		
- Height (0-4 pm)			0.04** (0.02)		
- Height Inverse (0-4 am - Nice)					-0.06*** (0.01)
- Height Inverse (4-8 am - Nice)			0.10*** (0.02)		
- Height Inverse (8-12 am - Nice)	0.03** (0.01)				
- Height Inverse (0-4 am - Lille)	0.05*** (0.01)				
- Height Inverse (4-8 am - Lille)					0.09*** (0.01)
- Height Inverse (8-12 am - Lyon)	0.06*** (0.02)				
- Height Inverse (0-4 am - Paris)				0.07*** (0.02)	
- Height Inverse (8-12am - Nantes)				0.03*** (0.01)	
- Height Inverse (0-4pm - Nantes)			-0.02** (0.01)		
- Height Inverse (4-8pm - Nantes)				0.02** (0.01)	
- Height Inverse (8-12pm - Strasbourg)				0.06*** (0.01)	
- Height Inverse (8-12pm - Marseille)				0.07*** (0.01)	
Altitude wind					
- Zonal wind (layer 20)	-0.12*** (0.02)			0.05*** (0.01)	
- Zonal Wind (layer 40)	-0.02 (0.02)				
- Meridional Wind (layer 32)	0.06*** (0.01)				
- Total strength (layer 38)	0.02 (0.09)				
- Total strength (layer 39)	-0.08 (0.09)				
- Total strength (layer 45)	-0.04** (0.02)				
- Total strength (layer 52)			0.02* (0.01)		
Altitude Pressure levels					
- Average (layer 25)	1.07*** (0.12)				
- Average (layer 46)			-0.20*** (0.02)		
- Average (layer 78)				-0.13*** (0.03)	
Selected (Optimal Constraint)	15	3	10	14	3
Weather controls	Yes	Yes	Yes	Yes	Yes
Observations	6,135	6,135	6,135	6,135	6,135
R ²	0.32	0.30	0.48	0.53	0.05

This table presents post-clustered-lasso models by pollutants on the sample where the five pollutants are simultaneously observed. The optimal "rigorous" lasso constraints is used. Before all regressions, we partial out fixed effects. For each pollutants, a first step of instruments' selection is performed with no selection on weather controls. Then, OLS is run per pollutant on the set of selected instruments, which are shown here. Significance: * p<0.1; ** p<0.05; *** p<0.01

(resp. from east) predicts a lower (higher) concentration, which is coherent with clean oceanic winds from the west and/or polluted air imported from the eastern regions. However, the opposite sign for altitude zonal wind appears for NO₂. Consistent with Table 4, thermal inversions patterns seems to have a stronger relationships with NO₂ and SO₂ than they have for PMs and CO. Thermal inversion strength is selected for SO₂ with a rather strong magnitude (0.07), while 5 dimensions describing thermal inversions are selected for NO₂. All 5 dimensions appear with a positive coefficient indicating that thermal inversions conditions (occurrence, strength) favor higher concentrations of NO₂ at various moments of the day. For predicting COs, IBLH is selected at two distinct moments of the day, 8-12 a.m. and 4-8 p.m. - possibly related to high traffic emissions at these hours. Patterns in IBLH are numerous for PMs, and city-specific patterns are also often selected for NO₂. As for O₃, this pollutant stands out with negative signs on thermal inversions and IBLH, and selection of PBLH with a positive sign. CO selection is somewhat close to the selection NO₂ (2 instruments selected for CO are also selected in the largest selection set for NO₂), but NO₂ may be distinguished from CO with the other dimensions. Table 5 suggests distinct relationships between these altitude weather variables and each pollutant, which is a first requirement to be able to disentangle the role of the various pollutants. The exceptions are PM_{2.5} and PM₁₀: the first models one obtains after Lasso are very similar for both pollutants, and therefore PM₁₀ is excluded in the rest of the analysis. The final pooled-instrument set for the five air pollutants has 35 selected dimensions: 20 related to the planetary boundary layer height, 6 related to thermal inversions, 7 related to altitude winds, 3 related to altitude pressure.

After the lasso selection and with the pooled selected instruments (one set of instruments has been selected for each pollutant), we run a two stage least squares with multiple pollutants, taking into account the same ground-level weather controls. In this post-lasso IV, the first stage writes as follows

$$p_{kct} = w_{ct}^{(s)} \mu + x_{ct}c + u_{ct} \quad (6)$$

where $w^{(s)}$ are altitude weather variables selected by Lasso in a first step among w . The full first stage is reported in Tables A.3 and A.4, with previously Lasso-selected dimension in bold. Once controlling for the instruments selected for the other pollutants, thermal inversions patterns (occurrence, strength) are unambiguously affecting positively NO₂ and SO₂, while the relationship with other pollutants is less clear-cut and depends on moments of the day. IPBLH patterns are strong predictors of PMs, CO, O₃ and NO₂, although the precise magnitudes, signs, involved

moments of the day or interactions with cities depends on each pollutant. Wind patterns are mostly related to PMs, and to a lesser extent to the other pollutants. Pressure altitudes are also strong predictors for all pollutants with various magnitude, layers and signs.

The second stage is as follows

$$r_{act} = \sum_{k=1}^K \hat{p}_{ct} \delta_k + x_{ct} \beta + \nu_{ct} \quad (7)$$

where each \hat{p}_{kct} is derived from the post-lasso first stage linear regression, and r is the rate of emergencies admissions or of mortality, possibly indexed by age a , and x ground-level weather controls. Note that we consider a linear specification for the impact of air pollutants on r , which excludes cocktail (interactions) or threshold effects (non-linearities). Lasso selection for interaction or non-linear terms did not lead to select strong instruments, as opposed to linear terms, preventing further analysis.

5 Results

We first disentangle separately the impact of distinct air pollutants on three short-term health outcomes using the Lasso-selected set among the large set of potential altitude weather instruments. To discuss the shortcomings of single-pollutant models, we then provide results when considering separately distinct pollutants instead as it is classic in this literature.

5.1 Disentangling the impact of distinct air pollutants

In this section, we derive pollutant-specific causal estimates within multi-pollutant specifications. Table 6 presents our main set of results, derived from Equations 6 and 7 in the post-lasso IV setting. The five-pollutant specification is run for mortality and emergency admissions related to cardiovascular, respiratory or digestive diseases, the later being a placebo outcome. We consider these results as giving the separate impact of pollutants, once controlled for the other main pollutants.³⁵ Being agnostic beforehand on which pollutants out of the five impact each outcome, we have to test five null hypotheses (no effect of a given pollutant) per outcome.

³⁵We never consider both types of particulate matter together as we did not succeed in disentangling their separate effects, even in regressions with only these two pollutants. This is probably because they are strongly interrelated: PM2.5 represents a large subset of PM10. Their most significant predictors tend to be the same, hampering our identification strategy.

False rejections of at least one null hypothesis are thus about five times more likely to happen by chance than if we were only considering a single treatment dimension. Accordingly, we report the significance level with the Bonferroni-Holm multiple hypothesis testing correction for controlling the Family-Wise Error Rate (FWER, the probability of at least one false rejection) at level α . As explained in [Holm \(1979\)](#), we rank the 5 p-values obtained for each outcome from the smallest to the highest and successively compares them to the threshold $\frac{\alpha}{5}, \frac{\alpha}{4}, \frac{\alpha}{3}, \frac{\alpha}{2}, \alpha$ until the m -th p-value falls above the m -th threshold. If $m > 1$, we reject the $m - 1$ assumptions of no pollutant effect with control of the FWER at level α . We focus on the sample where all the considered pollutants are observed, a condition which significantly alters the sample size. That is why we also provide intermediate multi-pollutant specifications based on more observations but the same set of instruments in [Tables A.5, A.6, A.7 and A.8](#) in the Appendix with an emphasis on the main culprits within the five-pollutant models - to discuss regularities.

For daily emergency admissions for respiratory diseases, we find compelling evidence of the detrimental and pollutant-specific effect of two pollutants: ozone (O3) and sulfur dioxide (SO2). They are found to have a positive and independent effect from other pollutants with a control of the FWER at 5%. A standard deviation of O3 ($24\mu g/m^{-3}$) causes 10% more emergency admissions for respiratory diseases while in addition, a standard deviation of SO2 ($1.5\mu g/m^{-3}$) causes an increase by 10%. These impacts are very stable when we successively add more pollutants to the equation to test both the sample-robustness due to missingness in air pollutant measures and to accounting for other observed pollutants. [Table A.5](#) in the Appendix reports a response between 7 to 13% to an O3 standard deviation and between 8 to 11% to a SO2 standard deviation, depending on which pollutant enters the model. While CO has no significant effect in our main and most complete specification, we tend to be cautious in excluding its effect on respiratory diseases. First because in models up to 4 pollutants based on more observations, estimates in [Table A.5](#) are statistically significant. Second, because as detailed later, when studying the short-term timing of the effects of air pollutants, the impact of carbon monoxide is significant in five-pollutant models as long as two lags in concentrations are introduced. One potential explanation is that in some estimations, the impact of carbon monoxide may still be confounded with that of NO2 (correlation of 0.69).

Overall, these results are consistent with the evidence from controlled per-pollutant chamber-exposure on small samples of volunteers, which evidence impaired respiratory func-

tions for all these three pollutants (e.g. for SO₂, [Johns and Linn \(2011\)](#), for O₃ and CO, [Ferris Jr \(1978\)](#)). O₃ has a well-known effect on respiratory functions, which is mediated by a number of mechanisms (see e.g. for a review, [Bromberg \(2016\)](#)). Our results offer, however, a larger external validity and evidence of worst health consequences (hospital admissions) in the general population. We find no effect of PM_{2.5} at short-term on emergency admissions for respiratory diseases, while they have the potential to cause lung inflammation. One reason might be that the frontier between particulate matter and sulfur dioxide is porous as the latter can serve as a primary pollutant for a sub-component of the former, in which case sulfur dioxide might be a better proxy to sulfur-rich particulates than total particulate matter.

Table 6: Causal effect of Air Pollutants on Mortality and Morbidity

<i>Multi Pollutant IV-Lasso Models</i>						
	<i>Emergency Admissions</i>			<i>Mortality</i>		
	Respiratory	Cardiovascular	Digestive	Respiratory	Cardiovascular	Digestive
PM _{2.5}	0.0009 (0.0024)	0.0008 (0.0021)	-0.0036 (0.0018)	0.0019 (0.0011)	0.0042** (0.0016)	0.0010 (0.0007)
CO	0.0002 (0.0003)	0.0007** (0.0003)	-0.0001 (0.0002)	0.0001 (0.0001)	0.0001 (0.0002)	-0.00002 (0.0001)
O ₃	0.0056** (0.0019)	0.0020 (0.0018)	-0.0026 (0.0017)	-0.0007 (0.0010)	0.0012 (0.0015)	0.0006 (0.0006)
SO ₂	0.0957** (0.0350)	-0.0250 (0.0342)	-0.0049 (0.0296)	0.0381* (0.0159)	0.0196 (0.0238)	-0.0020 (0.0109)
NO ₂	0.0026 (0.0027)	-0.0028 (0.0026)	0.0010 (0.0021)	0.0004 (0.0014)	0.0007 (0.0019)	-0.0007 (0.0008)
Observations	[6,135]	[6,135]	[6,135]	[6,135]	[6,135]	[6,135]
Instruments	35	35	35	35	35	35

This Table presents the results of post-clustered-lasso IV models. Before all regressions, we partial out fixed effects. All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with city fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant clustered-lasso selection is performed, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations. Standard errors are clustered at the month-year-city level, consistently with the post clustered-lasso estimation. In this Table, we correct significance for testing 5 hypothesis - that is one per pollutant - separately for each outcome, using the Bonferroni-Holm method. Multiple Hypothesis Testing adjusted significance: * Null rejected with control of the FWER<0.1; ** Null rejected with control of the FWER<0.05

For cardiovascular diseases, the unique air pollutant which is ever significant in multi-pollutant specifications is carbon monoxide (CO) - as reported in Table 6 and complementary Table A.6 in the Appendix. Quantitatively, a standard deviation of CO ($216\mu\text{g}/\text{m}^{-3}$) causes 9% more emergency admissions for cardiovascular diseases. The five pollutant model estimate is the highest, the single-pollutant model is the lowest estimate but will still imply a 3% response of cardiovascular emergency admissions to a standard deviation in CO concentration. The well-known accidental poisoning deaths caused by a high-level of carbon monoxide exposure are just the tip of the iceberg. Carbon monoxide causes adverse effects by combining with hemoglobin, preventing the blood from carrying oxygen. Therefore, the mechanisms of action of carbon monoxide are well understood and established (*hypoxia induced by formation of carboxyhemoglobin*), although the most recent research suggests other potential mechanisms to investigate.

For the mortality rate related to respiratory diseases, we find evidence of a detrimental impact of SO₂ although only controlling for the FWER at 10%. A one standard deviation of SO₂ would imply an increase by 14% of respiratory-related mortality - or 2% of the mortality rate, which is a relatively large effect compared to the literature, and given that SO₂ levels are much below what they were two decades ago.³⁶ Our results suggest that even at extremely low level, SO₂ harmful effect is still there. This is broadly consistent with the evidence on emergency admissions, which are not necessarily vital emergencies and are very frequent at the youngest age. In larger samples involving less pollutants as reported in Table A.8 this impact is very stable and at least of 3% and PM_{2.5} appears as well with a positive impact on this type of mortality but with more marginal significance levels (10% in the main specification before correcting for multiple hypothesis testing).

For the mortality rate related to cardiovascular diseases, we find strong evidence of the detrimental effect of particulate matter PM_{2.5} both in the most complete specification (Table 6) and across varying-pollutant models in Table A.7. Quantitatively, a standard deviation of PM_{2.5} ($11\mu\text{g}/\text{m}^{-3}$) causes an increase between 1.5 and 2 % of the mortality rate, or 5 to 9% of the cardiovascular-related mortality. We may compare our PM_{2.5} estimate to that from [Schwartz](#)

³⁶SO₂-induced mortality has been recently studied in China where the average level of SO₂ concentration is way above typical concentrations in Europe. [Kan et al. \(2010\)](#) and [Chen et al. \(2012\)](#) point to a statistically significant association at short-term in single-pollutant model but which tend to disappear when controlling for NO₂. Evidence in Europe is somewhat older. e.g. ([Tertre et al., 2002](#))

et al. (2016), which find for Boston over 2000-2009 with an IV strategy, that for an increase by about $6\mu\text{g}/\text{m}^{-3}$ of PM_{2.5} leads to an increase by 0.9% of daily deaths. Our estimate is of similar magnitude - only slightly higher. Particulate matter are thought to exacerbate pre-existing inflammatory diseases (oxidative stress induced by inhaled particles, causing lung inflammation). A PM-induced lung inflammation mechanism is widely suggested by the literature, while the evidence on PM-induced cardiovascular events need to be strengthened, in particular pointing out the mechanisms (Scapellato and Lotti, 2007). Many mechanisms, and therefore a number of associated diseases involving exposure to PMs are possibly mediating death outcomes. We can not exclude that other weakening factors are at play at the individual level to lead to death, especially since the eldest are the most affected population (see next section).

One may however ask why we see no effect of particulate matter on cardiovascular diseases emergency admissions³⁷ in Table 6 while this pollutant impacts cardiovascular mortality. In addition, we may ask why we see no effect of carbon monoxide on cardiovascular-related mortality which would be expected if emergency admissions were largely vital emergencies. Several assumptions may be formulated. First, although cardiovascular events could be mediated by PMs, the impact could be acute enough to lead directly to death, without the occurrence of an emergency admissions on average - in particular if the eldest are concerned.³⁸ Second, it could be the case that we do not have enough statistical power to find a small effect. Third, PMs could act in a context of complex co-morbidities, involving a cardiovascular mechanism but not only - in particular because they are themselves a mixture with various potential toxicological pathways. Last but not least, it could be the case that the existing evidence of PMs on short-term emergency admissions is confounded by the impact of other pollutants. In 2017 in France, among persons hospitalized for a stroke 12,5% died in hospital (on the day of the admissions or later)³⁹ and probably the persons with the most severe events did not reach the hospital. In our data, we observe 80% more admissions than deaths related to cardiovascular diseases. Therefore and especially with an analysis at the daily level, cardiovascular emergency admissions are on their vast majority not followed by a death event on the same day - and perhaps, short-term cardiovascular emergency and deaths with at least a cardiovascular cause

³⁷This does not exclude the role of PMs in cardiovascular events due to long-term exposure.

³⁸In 2006, 58% of the deaths in the French population happened in an hospital - a proportion which was rather stable over the previous decade. This proportion typically decreases with age as the eldest die relatively more at home (IGAS, 2009).

³⁹ATIH, Analyse de l'activité hospitalière, bilan 2017

events may be described with distinct causes. In this sense, our approach lacks specificity by focusing on aggregated outcomes. From [Franchini and Mannucci \(2007\)](#), “*Particulate matter are the type of air pollutant that causes the most numerous and serious effects on human health, because of the broad range of different toxic substances that it contains*”. Thus one interpretation of our results could be that PMs mediate a larger number of more severe events, building on co-morbidities which may explain its link with mortality, while CO may help in triggering cardiovascular emergencies not necessarily as severe as those leading to deaths.

Finally, we perform a falsification test by examining our models’ results on emergency admissions for digestive diseases (the other most common emergency admissions, with our two main outcomes) or on mortality related to digestive diseases - for which we do not reject a null impact of all five air pollutants. In addition, we test whether our standard errors are appropriate in size. In our baseline, we only allow arbitrary correlation within city-month-year cells - our fixed effects level, but we could allow for arbitrary correlation within city clusters of observations. To account for the small number of cluster, we do not resort to cluster robust standard errors at the city-level (which are biased downward) but compute wild clustered bootstrap standard errors ([Cameron et al., 2008](#)) and report the associated p-values for testing the null hypothesis of no effect of a given pollutant. Results are reported in [Table A.9](#), with indications on the control of the FWER under this alternative set of p-values. Virtually all our main results are confirmed but the impact of SO₂ on respiratory-related mortality which is still significant at the 5% level but not after accounting for testing five distinct hypotheses. Results on pollutants with a higher number of selected instruments (that is PMs, O₃) turn to be even more significant while results on pollutants with a smaller number of selected instruments (SO₂, CO) are then only significant at the 2% level, that is with a control of the FWER at 10%. In the rest of the paper, we stick to the clustering at the month-year-city level.

5.2 Contrasting Mono-Pollutant and Multi-Pollutant models

Since the existing short-term literature mostly relies on single-pollutant models, we now turn to the comparison of single-pollutant and multi-pollutant estimates to provide some hindsight on the limitations of single-pollutant results. With such a strategy, a single pollutant could drive all the results attributed to other pollutants. This may be mitigated by having pollutant-specific instruments. In this paper, our main set of results are derived within multi-pollutant models with lasso-selected and pollutant-specific instruments. These results may be compared with

two other setups: single-pollutant models with lasso-selected and pollutant-specific instruments or the single-pollutant models with a simple IV, with first stage as in Table 4. Both strategies ignore the exclusion restriction violation due to the presence of other pollutants, but the former's first stage is tailored to predict each pollutant while the later does not adapt the instrument set to the pollutant under scrutiny - but consider two strong hand-picked instruments: IBLH and TI. We consider the sample where all five pollutants are observed to perform the comparison.⁴⁰

We begin with simple IV results reported in Table 7, where in columns (1P-IV) each coefficient is from a separate regression, the dependent variable being either emergency admissions for respiratory or cardiovascular diseases or respiratory or cardiovascular-related mortality. The later estimates are broadly comparable to the existing literature, which do not control for the presence of other pollutants. For each specific pollutant, concentrations are expressed in $\mu\text{g}/\text{m}^{-3}$. Quantitatively, + 10 $\mu\text{g}/\text{m}^{-3}$ in PM2.5 (about a standard deviation) leads to + 12% more respiratory-related mortality, + 7% more admissions for cardiovascular-related mortality and a emergency admissions for cardiovascular diseases higher by +4%. For CO, + 200 $\mu\text{g}/\text{m}^{-3}$ (about a standard deviation) leads to +6% more cardiovascular admissions, +11% of the cardiovascular mortality rate and + 16% of the respiratory mortality rate. Notably, all pollutants are found to have an effect on both types of mortality rates. In addition, ozone is even found to have a large and *negative* effect on mortality rates, a finding at odds with the existing medical literature and in particular with chamber experiments.⁴¹ As ozone concentration is strongly anti-correlated with the other air pollutant concentrations, a high concentration of ozone most probably proxies for the absence of the other air pollutants. In addition, negative variations of ozone triggered by the instrument are accompanied by positive variations in all other pollutants (Figure A.2). This result indicates that the exclusion restriction does not hold in single-pollutant models and in particular when pollutants are interrelated. To study O3, we will need to control at least for the other pollutants involved in equilibrium with O3, that are anti-correlated (carbon monoxide, nitrogen oxides). Omitted controls and the exclusion restriction violation lead to some pollutants variations acting as a surrogate for the others' variations. These results seem in sharp contrast with our main multi-pollutant results, reported in columns labelled (5P-IVLasso).

⁴⁰While single-pollutant models could be run on larger samples, we show in appendix Tables B.5, B.6, B.7 and B.8 that single-pollutant IV-Lasso models estimates are very stable across samples used for selection and/or estimation, so the conclusion are similar if we compare multi-pollutant models and single-pollutant models on their respective "natural" sample for estimation.

⁴¹However, it was also observed by Deryugina et al. (2019).

To go beyond these qualitative comparisons, we report p-values for testing for the equality of coefficients across both specifications. We simultaneously estimate each single-pollutant model jointly with the five-pollutant model by GMMs which allows for testing a linear constraint of equality between both model coefficients. We also report the hypothesis which will be rejected if we control the Family-Wise Error rate for testing for five equality of coefficients at 1, 5 or 10% level. We strongly reject equality of estimates between both approaches, and systematically across outcomes for ozone.

Single-pollutant models with pollutant-specific instruments, as selected by Lasso and reported in columns (IP-IVLasso) in Table 8, provide better results than simple IVs. They tend to be more consistent with multi-pollutant models. The impact of ozone on respiratory emergency admissions - even without controls for the anti-correlated pollutants - appear as expected positive and significant. The negative effect of ozone on respiratory mortality tends to persist - and disappears only when controlling for other pollutants. Aside from the particular case of ozone, there are many significant effects which disappear when other pollutants are introduced. For instance, our results suggest that NO₂ is as a general fact a surrogate for the other pollutants. NO₂ is found to affect our three health outcomes in all single-pollutant models whereas this effect never survives in the five-pollutant context. In terms of pathology, the surrogate phenomenon tends to be the strongest for the mortality rate: single-pollutant models tend to accuse almost all of the available pollutants, which is not the case in multi-pollutant models that designate particulate matter or SO₂. For emergency admissions, the issue seems less relevant as except for the effect of NO₂ on cardiovascular emergencies, we do not reject equality of coefficients across specifications. All in all, our results suggest a cautionary review of the literature based on single-pollutant models when instruments are not pollutant-specific.

5.3 Robustness and further results

Results by Age Group. We then conduct an heterogeneity analysis along the age ladder. We restrict the pollutants entering the model to those found to have significant impact in the general population, a choice which preserves the sample size. We report as well the more noisy estimates derived within the baseline five-pollutant models. We note that these results by age are not as precise as our main results (we do not attempt any MHT correction here) but they may still provide some useful information. Results are graphically reported in Figure 2, and corresponding Tables are provided in Appendix B.9. For respiratory diseases, both hands of the

Table 7: Comparison of Single Pollutants in Classical IV and Multi Pollutants models with Lasso-selected Instruments.

	Panel A: Mortality					
	<i>Respiratory</i>			<i>Cardiovascular</i>		
	(5P-IVLasso)	(1P-IV)	Equality Test	(5P-IVLasso)	(1P-IV)	Equality Test
PM2.5	0.0019* (0.0011)	0.0056*** (0.0014)	$p = 0.0324^*$	0.0042*** (0.0016)	0.0066*** (0.0018)	$p > 0.1$
CO	0.0001 (0.0001)	0.0004*** (0.0001)	$p = 0.0091^{**}$	0.0001 (0.0002)	0.0005*** (0.0002)	$p = 0.0535$
O3	-0.0007 (0.0010)	-0.0050*** (0.0012)	$p = 0.0021^{***}$	0.0012 (0.0015)	-0.0059*** (0.0016)	$p = 0.0002^{***}$
NO2	0.0004 (0.0014)	0.0081*** (0.0019)	$p = 0.0008^{***}$	0.0007 (0.0019)	0.0089*** (0.0024)	$p = 0.0010^{***}$
SO2	0.0381** (0.0159)	0.1283*** (0.0430)	$p = 0.0268^*$	0.0196 (0.0238)	0.1295** (0.0533)	$p = 0.0305^*$
Instruments	35	2		35	2	
Observations	6,135	6,135		6,135	6,135	
	Panel B: Emergency Admissions					
	<i>Respiratory</i>			<i>Cardiovascular</i>		
	(5P-IVLasso)	(1P-IV)	Equality Test	(5P-IVLasso)	(1P-IV)	Equality Test
PM2.5	0.0009 (0.0024)	0.0047* (0.0025)	$p > 0.1$	0.0008 (0.0021)	0.0061** (0.0026)	$p = 0.0850$
CO	0.0002 (0.0003)	0.0003 (0.0002)	$p > 0.1$	0.0007*** (0.0003)	0.0005** (0.0002)	$p > 0.1$
O3	0.0056*** (0.0019)	-0.0040* (0.0022)	$p = 0.0003^{***}$	0.0020 (0.0018)	-0.0056** (0.0024)	$p = 0.0055^{**}$
NO2	0.0026 (0.0027)	0.0101*** (0.0033)	$p = 0.0593$	-0.0028 (0.0026)	0.0054* (0.0031)	$p = 0.0337$
SO2	0.0957*** (0.0350)	0.2134** (0.0814)	$p > 0.1$	-0.0250 (0.0342)	0.0290 (0.0571)	$p > 0.1$
Instruments	35	2		35	2	
Observations	6,135	6,135		6,135	6,135	

This Table compares the results of single pollutant models estimated with a classical IV with multi-pollutant models after Lasso selection. Columns (5P-IVLasso) correspond to multi-pollutants regressions estimated with an IV Clustered-Lasso. In columns (1P-IV), each coefficient corresponds to a separate regression with one pollutant and two hand-picked strong instruments. We test for the equality of coefficients by estimating jointly 5P and 1P models by solving the joint GMM model, and report the p-value for the hypothesis of equality of coefficients. These p-values are adjusted for clustering at the city-month-year level. Significance: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$. Multiple Hypothesis Testing adjusted significance: * Null rejected with $\text{FWER} < 0.1$; ** $\text{FWER} < 0.05$; *** $\text{FWER} < 0.01$

Table 8: Comparison of Single and Multi Pollutants models with Lasso-selected Instruments.

Panel A: Mortality						
	<i>Respiratory</i>			<i>Cardiovascular</i>		
	(5P-IVLasso)	(1P-IVLasso)	Equality Test	(5P-IVLasso)	(1P-IVLasso)	Equality Test
PM2.5	0.0019* (0.0011)	0.0035*** (0.0008)	$p = 0.0509$	0.0042*** (0.0016)	0.0045*** (0.0011)	$p > 0.1$
CO	0.0001 (0.0001)	0.0003*** (0.0001)	$p > 0.1$	0.0001 (0.0002)	0.0004*** (0.0001)	$p > 0.1$
O3	-0.0007 (0.0010)	-0.0022*** (0.0008)	$p = 0.0236^*$	0.0012 (0.0015)	-0.0016 (0.0012)	$p = 0.0029^{**}$
NO2	0.0004 (0.0014)	0.0042*** (0.0011)	$p = 0.0037^{**}$	0.0007 (0.0019)	0.0055*** (0.0016)	$p = 0.0014^{**}$
SO2	0.0381** (0.0159)	0.0718*** (0.0178)	$p = 0.0137^*$	0.0196 (0.0238)	0.0659** (0.0262)	$p = 0.0171^*$
Instruments	35	15;3;10;14;3		35	15;3;10;14;3	
Observations	6,135	6,135		6,135	6,135	
Panel B: Emergency Admissions						
	<i>Respiratory</i>			<i>Cardiovascular</i>		
	(5P-IVLasso)	(1P-IVLasso)	Equality Test	(5P-IVLasso)	(1P-IVLasso)	Equality Test
PM2.5	0.0009 (0.0024)	0.0007 (0.0018)	$p > 0.1$	0.0008 (0.0021)	0.0018 (0.0015)	$p > 0.1$
CO	0.0002 (0.0003)	0.0003 (0.0002)	$p > 0.1$	0.0007*** (0.0003)	0.0004* (0.0002)	$p > 0.1$
O3	0.0056*** (0.0019)	0.0038** (0.0015)	$p > 0.1$	0.0020 (0.0018)	0.0003 (0.0016)	$p > 0.1$
NO2	0.0026 (0.0027)	0.0063*** (0.0021)	$p > 0.1$	-0.0028 (0.0026)	0.0040** (0.0020)	$p = 0.0052^{**}$
SO2	0.0957*** (0.0350)	0.0840** (0.0385)	$p > 0.1$	-0.0250 (0.0342)	-0.0242 (0.0379)	$p > 0.1$
Instruments	35	15;3;10;14;3		35	15;3;10;14;3	
Observations	6,135	6,135		6,135	6,135	

This Table compares the results of single and multi pollutant models after Lasso selection, for four health outcomes. Columns (5P-IVLasso) correspond to multi-pollutants regressions. In columns (1P), each coefficient corresponds to a separate regression with one pollutant and the pollutant-specific selected instruments. Before all regressions, we partial out fixed effects (month-year \times city and day-of-the-week \times city) and variables are replaced by the corresponding residuals. A first step of per-pollutant clustered-lasso selection is performed, conditional on weather variables which are forced into the model (no selection). In five pollutant models, selected instruments are pooled in 5P models, in 1P model only pollutant-specific instruments are used. Standard errors are clustered at the month-year-city level, following the post clustered-lasso estimation. We test for the equality of coefficients by estimating jointly 5P and 1P models by solving the joint GMM model, and report the p-value for the hypothesis of equality of coefficients. These p-values are adjusted for clustering at the city-month-year level. Significance for unadjusted p-values * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$. Multiple Hypothesis Testing adjusted significance: * FWER < 0.1 ; ** FWER < 0.05 ; *** FWER < 0.01

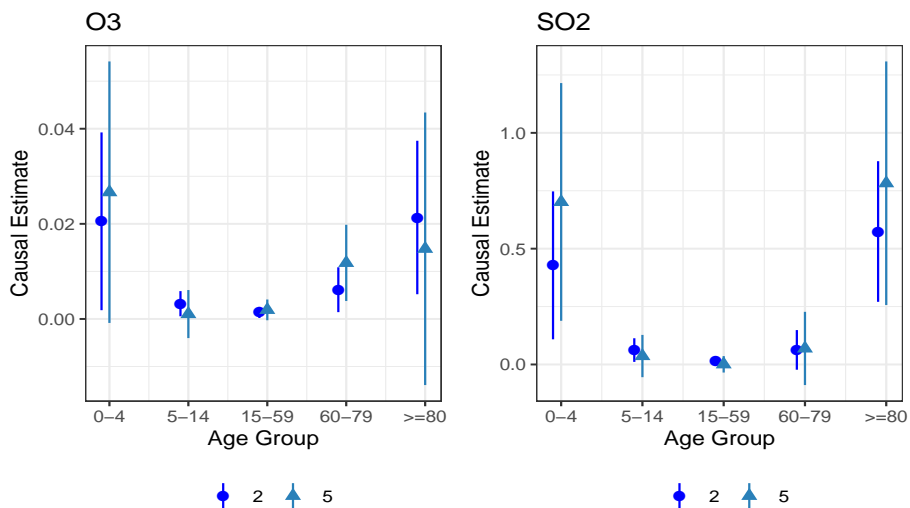
age distribution stand out with the largest impacts. Emergency admissions are higher for young children (less than 4) by 9% when O₃ is higher by one standard deviation ($+24\mu\text{g}^{-3}$) and by 11% when SO₂ is higher by one standard deviation ($+1.5\mu\text{g}^{-3}$). For the eldest (≥ 80), it would be higher by respectively 7% and 12%.

As expected, and although not always statistically significant, the impact is concentrated on the eldest age group (≥ 80) for mortality and cardiovascular diseases. $+11\mu\text{g}/\text{m}^{-3}$ of PM_{2.5} (one standard deviation) leads to an increase in the mortality rate of the eldest by 2.5% and the impact of SO₂ on the mortality rate is the highest of the eldest.⁴² For cardiovascular diseases, a standard deviation of CO ($216\mu\text{g}/\text{m}^{-3}$) implies emergency admissions higher by 6.6% for the 80 and plus age group. This is consistent with air pollution affecting a fragile population, and may raise questions about harvesting effects (that is, mortality displacement). The latter is however not dealt with in this paper, at least for health events displacement over a few days.

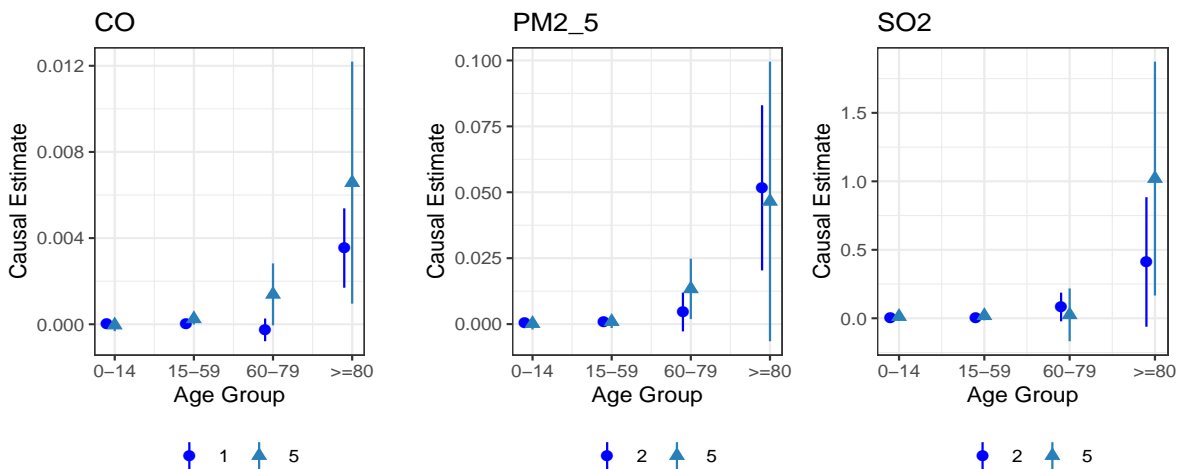
Specification of Weather Controls. Accounting correctly for ground-level controls is one of the requirements of our exclusion restriction. We provide in Table 9 three robustness exercises relative to weather controls for each health outcome. Column (1) reports the baseline result where controls are specified as a polynomial of order 2 in temperature, average precipitations and average wind strength, plus controls in humidity, in sunlight and a dummy for the presence of snow. From the literature, the most sensitive control in our specification is temperature. Its impact on human health has long been documented - and although we do not use directly altitude temperature in our estimation, ground-level temperature is not unrelated with our instruments. Precipitations have an impact on human activities (e.g. remain home) and on air pollution, but no clear short-term impact on health. Studies on the temperature-mortality link typically focus on extreme temperature e.g. Barreca et al. (2016). In column (2), we replace average temperature and average precipitations with both their daily minimum and maximum - each entering the specification with a polynomial of order two. Estimates remain basically the same while the cardiovascular point estimate decreases in significance at the 5% level.

Ideally and given the non-linear link between mortality and temperature, weather controls should be preferably specified as bins. It is for instance the case in (Deryugina et al., 2019)

⁴²We do not observe mortality rate by both age *and* causes due to privacy protection reasons. We consider in this section all-causes mortality.



(a) Respiratory Diseases: O3 and SO2



(b) Cardiovascular Diseases: CO

(c) Mortality Rate: PM2.5 and SO2

Figure 2: Causal Effect of Main Pollutants by Age Group. *Note:* Post IV Lasso models with 90% confidence intervals are represented. Causal effects are estimated either with all five pollutants (triangle) or with the specified pollutants (circle).

where ground-level weather controls are specified in 29,000 theoretical interactions between 17 bins of minimum and maximum temperature intervals, deciles of daily precipitations and wind-speed for a sample size of 1.9 million observations, 300 instruments, and county, state-by-month, and month-by-year fixed effects. Our weather controls are certainly not as flexible. However, we tend to absorb more variations in our set of fixed effects (city-by-month-by-year + day-of-the-week-by-city) letting only a few observations left for identification within month-year-city cells. In the end, the impact of ground-level weather on health outcomes is found rather weak (see Table A.10 in Appendix) and we show in the Appendix Table B.3 that starting from the simplest specification to the full specification of ground-level controls does not affect our results. Our sample size does not allow as many ground-level interactions as done in Deryugina et al. (2019). Yet this does not preclude an issue in the validity of our exclusion restriction due to misspecification of ground-level weather controls. To address this concern, in column (3), temperature control is further specified with the daily number of hours spent in each decile of temperature. Both precision and point estimates are very close to column (2), except for the cardiovascular emergency outcome where significance falls at 10% level. Finally, in column (4), the main controls are all specified in bins: temperature, wind strength and humidity are specified with the number of hours in quartile bins of the variable, and precipitations in three intervals as the median precipitation is zero. While the estimates on respiratory and cardiovascular emergencies remain very similar, mortality rate estimates lose their significance, and decreases in magnitude. However, with this quite demanding weather controls specification and consistent with the fact that the sample size is binding rather than the exclusion restriction invalid, restricting the set of pollutants to PM_{2.5} and SO₂ leads to recover our main set of results with a larger sample where both pollutants are observed and instruments specific to both pollutants. We conclude that in this most demanding specification, we succeed in disentangling the effect of five air pollutants on respiratory and cardiovascular emergency admissions but we can not disentangle the impact of more than two pollutants on mortality rates.

Finally, we report in the online appendix two additional robustness exercises relative to air pollutant measurement error and to delayed effects. In this paper, we measure air pollutant concentrations with the 24-hour average over a constant set of hourly-monitoring stations located within the urban area. Instead of the average, we use day minimum and maximum to test how our baseline results would change and report the updated results in Table B.4 in the Online Appendix. In addition to gauging how robust are our estimates to concentration measurements, this test may possibly inform on health sensitivity to sustained versus acute pollutant concen-

Table 9: Causal effect of Air Pollutants on Health Outcomes. Robustness checks to weather controls.

	Emergency admissions							
	Respiratory Diseases				Cardiovascular Diseases			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
PM2.5	0.0009 (0.0024)	0.0017 (0.0022)	0.0036 (0.0029)	-0.0002 (0.0024)	0.0008 (0.0021)	0.0020 (0.0021)	0.0037 (0.0026)	0.0012 (0.0023)
CO	0.0002 (0.0003)	0.0001 (0.0003)	0.0001 (0.0003)	0.0001 (0.0003)	0.0007*** (0.0003)	0.0006** (0.0002)	0.0004* (0.0002)	0.0005** (0.0002)
O3	0.0056*** (0.0019)	0.0056*** (0.0019)	0.0053*** (0.0020)	0.0040* (0.0022)	0.0020 (0.0018)	0.0020 (0.0018)	0.0015 (0.0018)	0.0010 (0.0019)
NO2	0.0026 (0.0027)	0.0038 (0.0028)	0.0036 (0.0026)	0.0036 (0.0027)	-0.0028 (0.0026)	-0.0015 (0.0029)	-0.0013 (0.0026)	-0.0015 (0.0025)
SO2	0.0957*** (0.0350)	0.0942*** (0.0343)	0.1034*** (0.0360)	0.1141*** (0.0350)	-0.0250 (0.0342)	-0.0069 (0.0325)	-0.0146 (0.0332)	-0.0119 (0.0340)
<i>Weather Controls</i>	Baseline	Min/Max	Bins (1)	Bins (2)	Baseline	Min/Max	Bins (1)	Bins (2)
<i>Observations</i>	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135
<i>Instruments</i>	35	38	37	40	35	38	37	40
	Mortality with Respiratory Causes				Mortality with Cardiovascular Causes			
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
PM2.5	0.0019* (0.0011)	0.0026** (0.0011)	0.0029** (0.0014)	0.0012 (0.0012)	0.0042*** (0.0016)	0.0048*** (0.0016)	0.0055*** (0.0021)	0.0032* (0.0018)
CO	0.0001 (0.0001)	-0.00002 (0.0001)	0.0001 (0.0001)	0.0001 (0.0001)	0.0001 (0.0002)	0.00001 (0.0002)	0.00004 (0.0002)	0.0002 (0.0002)
O3	-0.0007 (0.0010)	-0.0005 (0.0010)	-0.0007 (0.0010)	-0.0006 (0.0010)	0.0012 (0.0015)	0.0010 (0.0014)	0.0011 (0.0015)	0.0011 (0.0015)
NO2	0.0004 (0.0014)	0.0010 (0.0016)	0.0006 (0.0015)	0.0013 (0.0014)	0.0007 (0.0019)	0.0015 (0.0021)	0.0013 (0.0020)	0.0013 (0.0018)
SO2	0.0381** (0.0159)	0.0412** (0.0164)	0.0337** (0.0160)	0.0244 (0.0155)	0.0196 (0.0238)	0.0179 (0.0227)	0.0258 (0.0241)	0.0191 (0.0228)
<i>Weather Controls</i>	Baseline	Min/Max	Bins (1)	Bins (2)	Bins (2)	Bins (2)	Bins (2)	Bins (2)
<i>Observations</i>	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135
<i>Instruments</i>	35	38	37	40	35	38	37	40

This Table presents the results of post-clustered-lasso IV models. Weather controls are in the baseline (1) a polynomial of order 2 in average temperature, average precipitations and average wind strength, a linear control in sunlight and in humidity and a dummy for the presence of snow. In specification (2), temperature and precipitations are specified as an order two polynomial in their daily minimum and maximum. In specification (3), temperature control is specified with the daily number of hours spent in each decile of temperature. In specification (4), temperature, wind strength and humidity are specified with the number of hours spent in quartile bins of the variable, and precipitation in three intervals. All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant clustered-lasso selection is performed, conditional on various weather variables specification (1) to (4) which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations with the same weather controls. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

trations. Estimation with min and max leads to results consistent with our baseline estimation. Throughout the whole analysis, we have assumed that the impact of air pollution on short-term health indicators was exclusively contemporaneous. In this last part, we check whether we find lagged effects when introducing day-level leads and lags in the IV, with both contemporaneous and lagged instruments. For most of our results, these regressions suggest that the (short-term) effect is mostly contemporaneous.

6 Conclusion

This paper shows how distinct pollutants have strong and independent effects on the short-term respiratory health of the urban population. We develop a two-step strategy, showing first how air pollution is causally linked to daily emergency admissions and mortality rates and second how optimally selecting many more instruments allows to disentangle the effects of several pollutants. We provide causal evidence on the separate effects of ozone and sulfur dioxide on respiratory diseases, jointly and independently, in the real urban environment, and controlling for the other pollutants. Moreover, we find a significant impact of carbon monoxide on cardiovascular diseases as well as of particulate matter and sulfur dioxide on the mortality rate, while controlling for the other pollutants in presence. Our results suggest a reassessment of the evidence derived in single-pollutant models, by providing a comparison with multi-pollutant models. In addition, we show how high dimensional data from a general climate model can be leveraged to provide a large set of instruments which prove very insightful for clean evidence of ambient pollution levels on health. Our results point out to large effects of relatively small amounts of ozone, sulfur dioxide, carbon monoxide and particulate matter, borne in priority by children and elderly. While European norms have improved air quality as e.g. carbon monoxide is concerned, ozone concentrations are not at all decreasing in modern European cities. Moreover, our estimates could be considered for the production of a short-term pollution index reflecting the joint and independent impact of several pollutants - as opposed to the current standard of aggregation, a maximum over air pollutant sub-indexes, or for designing age-specific public alert. That being said, effective communication about air quality or correct economic valuation of multi-pollutant environmental policies remain complex questions which lie outside the scope of the present paper. This work only considers a limited number of air pollutants in the pool of pollutants suspected to jeopardize human health and well-being. However, this paper contributes to the discussion by proposing a framework which could, when the data is

recorded and available, be extended to allow for more substances - proving useful in a world where the unveiling of new sources of dangers is becoming more and more frequent.

The recent literature has emphasized the need for tackling the total health effect associated with the exposure to multiple pollutants, including non-linearities and interactions (Bobb et al., 2015), but a fully convincing method would have to deal with causality as well. In this paper, we have focused on deriving linear causal estimates within a multi-pollutant framework, but emphasize that future research bringing the theoretical econometrics literature on nonparametric instrumental variables to implementation could be promising continuation for our application. The problem is not simple in our view: in the first stage one needs a regularization step to incorporate the many instruments in the spirit of Belloni et al. (2012) or Carrasco and Tchuente (2015) and in the second stage, finding a causal response function for five pollutants means finding a non parametric function of five arguments, which is very computationally and data intensive. However, the air pollution mixture effects, modeled in a non-parametric way, may provide better insight into the features of the air pollution mixes which lead to the worst health impacts. Indeed, in the words of the WHO, high-impact future research should focus on “the shape of the concentration-response function, the identification of thresholds and the effects at very low or very high pollutant levels”.

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7 Appendix

Table A.1: The Ten Most Populated Urban Areas in France

Urban area	Population in thousands		
	All age	0-4	over 70
Paris	12,470	845	1,203
Lyon	2,259	152	249
Marseille - Aix-en-Provence	1,744	103	231
Toulouse	1,312	81	137
Bordeaux	1,195	67	135
Lille	1,182	80	111
Nice	1,006	52	171
Nantes	934	61	97
Strasbourg	777	45	89
Rennes	708	46	70

For Lille and Strasbourg urban area, only the French part is considered.

Source: 2013 census

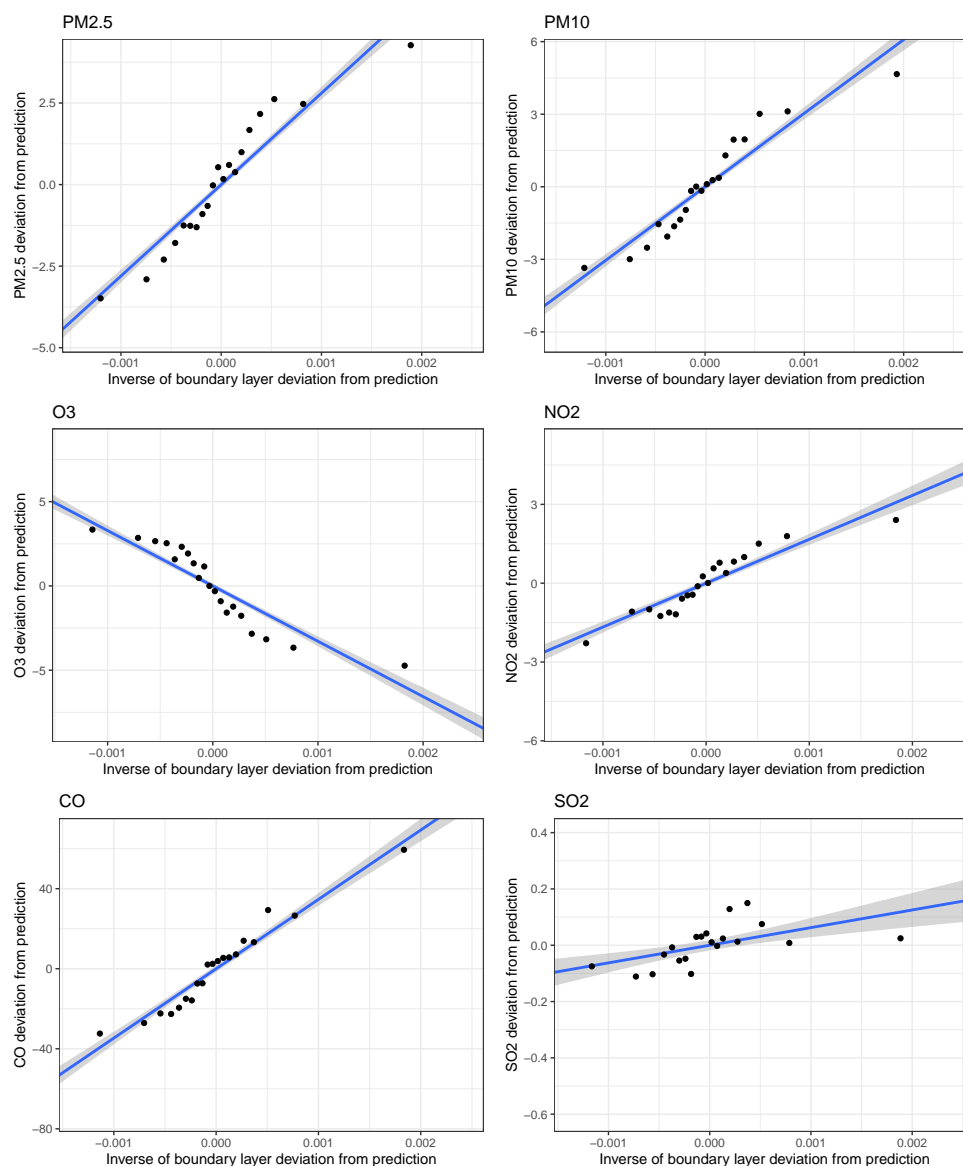


Figure A.2: Pollutant concentrations and Inverse Planetary Boundary Layer Height: Unusual Components.

Note: "Unusual" refers to the deviation of the variable from a set of weather and seasonal controls. On top of the regression line, for each quantile from p5 to p95 of unusual inverse boundary layer height, are represented the mean of unusual pollutant concentration.

Table A.2: Mortality Rate, Emergency Admissions, Inverse of Planetary Boundary Layer Height and Thermal Inversions

<i>Dependent variable, per 100 000 inhabitants:</i>						
	<i>Emergency Admissions</i>			<i>Mortality</i>		
	Respiratory	Cardiovascular	Digestive	Respiratory	Cardiovascular	Digestive
	(1)	(2)	(3)	(4)	(5)	(6)
$IBLH_{c,t}$	5.11 (5.08)	7.82* (4.04)	2.22 (3.59)	10.35*** (2.29)	7.44** (3.13)	-2.00 (1.27)
Observations	21,459	21,459	21,459	21,459	21,459	21,459
	(1)	(2)	(3)	(4)	(5)	(6)
$TI_{c,t}$	0.05*** (0.02)	0.02 (0.01)	-0.001 (0.01)	0.03*** (0.01)	0.03*** (0.01)	-0.005 (0.004)
Observations	21,459	21,459	21,459	21,459	21,459	21,459

IBLH stands for the Inverse of Planetary Boundary Layer Height averaged by date and urban area. TI stands for the number of hours with a thermal inversion a given date in the urban area. All regressions includes month-year and day-of-the-week fixed effects, interacted with urban area fixed effects; and weather controls. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.3: Post Lasso First Stage : Air Pollutants and Instruments (Part I)

# Selected	PM2.5 (15)	CO (3)	O3 (10)	NO2 (14)	SO2 (3)
Planetary Boundary Layer					
- Height Inverse (Day Average)	0.122*** (0.039)	0.092* (0.051)	0.000 (0.024)	-0.161*** (0.027)	-0.055 (0.044)
- Height Inverse (0-4a.m.)	0.133*** (0.025)	0.076*** (0.019)	0.015 (0.019)	0.101*** (0.017)	-0.034 (0.029)
- Height Inverse (4-8a.m.)	-0.090*** (0.030)	-0.056** (0.023)	-0.004 (0.019)	-0.016 (0.018)	0.032 (0.035)
- Height Inverse (8-12a.m.)	0.024 (0.029)	0.063** (0.025)	-0.020 (0.018)	0.087*** (0.019)	0.021 (0.035)
- Height Inverse (4-8p.m.)	-0.018 (0.021)	0.068*** (0.023)	-0.035** (0.014)	0.136*** (0.018)	0.020 (0.026)
- Height (0-4a.m.)	0.001 (0.015)	0.049*** (0.013)	0.082*** (0.011)	0.018* (0.010)	0.005 (0.020)
- Height (8-12a.m.)	-0.026 (0.020)	-0.000 (0.016)	0.005 (0.017)	-0.011 (0.015)	-0.016 (0.030)
- Height (0-4p.m.)	0.096*** (0.021)	0.093*** (0.021)	0.028 (0.020)	0.036** (0.014)	0.007 (0.026)
- Height Inverse (0-4a.m. - Nice)	-0.026 (0.040)	-0.011 (0.035)	-0.002 (0.044)	-0.050* (0.027)	-0.004 (0.040)
- Height Inverse (4-8a.m. - Nice)	0.031 (0.059)	-0.040 (0.044)	0.098** (0.044)	0.046 (0.039)	-0.053 (0.052)
- Height Inverse (8-12a.m. - Nice)	0.042 (0.026)	0.012 (0.023)	-0.001 (0.022)	-0.034 (0.023)	-0.023 (0.031)
- Height Inverse (0-4a.m. - Lille)	0.011 (0.035)	0.004 (0.017)	-0.033** (0.015)	-0.014 (0.016)	0.052 (0.037)
- Height Inverse (4-8a.m. - Lille)	0.053 (0.036)	0.019 (0.016)	0.025 (0.018)	-0.003 (0.016)	0.042 (0.033)
- Height Inverse (8-12a.m. - Lyon)	0.068*** (0.020)	-0.017 (0.020)	0.023** (0.011)	0.031 (0.019)	0.013 (0.020)
- Height Inverse (0-4a.m. - Paris)	0.079 (0.053)	0.056*** (0.021)	-0.001 (0.025)	0.064*** (0.016)	0.023 (0.027)
- Height Inverse (8-12a.m. - Nantes)	-0.010 (0.020)	0.006 (0.014)	0.012 (0.012)	0.018* (0.011)	-0.050** (0.020)
- Height Inverse (0-4p.m. - Nantes) S	0.047** (0.023)	0.006 (0.015)	-0.026* (0.013)	0.024** (0.012)	0.024 (0.016)
- Height Inverse (4-8p.m. - Nantes)	-0.010 (0.013)	-0.002 (0.013)	-0.008 (0.008)	0.008 (0.011)	-0.011 (0.014)
- Height Inverse (8-12p.m. - Marseille)	0.011 (0.018)	0.021 (0.023)	-0.013 (0.018)	0.058*** (0.011)	-0.033 (0.022)
- Height Inverse (8-12p.m. - Strasbourg)	0.003 (0.014)	0.013 (0.015)	0.001 (0.012)	0.054*** (0.013)	0.020 (0.016)
...					

Table A.4: Post Lasso First Stage : Air Pollutants, Instruments and Weather Controls (Part II)

# Selected	PM2.5 (15)	CO (3)	O3 (10)	NO2 (14)	SO2 (3)
			...		
Thermal inversions					
- Hours between 0 and 4a.m.	-0.009 (0.019)	-0.020 (0.015)	0.020* (0.011)	0.010 (0.011)	-0.009 (0.019)
- Hours between 8 and 12a.m.	-0.013 (0.018)	-0.003 (0.017)	-0.048*** (0.011)	-0.009 (0.012)	-0.019 (0.024)
- Hours between 8 and 12p.m.	-0.034*** (0.012)	0.002 (0.009)	-0.016** (0.007)	0.036*** (0.008)	0.012 (0.018)
- Strength between 0 and 4a.m.	0.074*** (0.028)	0.083*** (0.027)	-0.008 (0.018)	0.040** (0.016)	0.069* (0.036)
- Strength between 4 and 8a.m.	-0.060** (0.030)	-0.082*** (0.029)	0.037** (0.018)	0.002 (0.017)	0.013 (0.039)
Altitude Wind					
- Zonal Wind (Layer 20)	-0.121*** (0.018)	0.038*** (0.011)	0.037*** (0.012)	0.052*** (0.011)	0.053*** (0.020)
- Zonal Wind (Layer 40)	-0.016 (0.017)	-0.015 (0.013)	-0.006 (0.011)	-0.011 (0.011)	-0.032* (0.018)
- Meridional Wind (Layer 32)	0.055*** (0.014)	0.020** (0.009)	-0.015 (0.009)	-0.037*** (0.009)	-0.019 (0.015)
- Total Wind Strength (Layer 38)	0.012 (0.092)	-0.064 (0.060)	0.061 (0.063)	0.087 (0.060)	-0.015 (0.096)
- Total Wind Strength (Layer 39)	-0.066 (0.094)	0.051 (0.060)	-0.068 (0.065)	-0.083 (0.060)	0.025 (0.099)
- Total Wind Strength (Layer 45)	-0.044** (0.020)	0.004 (0.016)	-0.029* (0.016)	-0.009 (0.013)	-0.007 (0.022)
- Total Wind Strength (Layer 52)	0.001 (0.020)	-0.025 (0.016)	0.038*** (0.013)	-0.010 (0.012)	0.000 (0.021)
Altitude Pressure levels					
- Average (Layer 25)	1.316*** (0.155)	0.418*** (0.105)	-0.273*** (0.092)	0.420*** (0.091)	0.416*** (0.152)
- Average Pressure (Layer 46)	-0.075 (0.060)	-0.051 (0.042)	-0.137*** (0.034)	-0.115*** (0.030)	-0.118** (0.048)
- Average Pressure (Layer 78)	-0.063 (0.043)	-0.095*** (0.027)	0.057*** (0.021)	-0.122*** (0.027)	-0.063 (0.043)

All variables are scaled with their in sample standard deviation. All regressions includes month-year and day-of-the-week fixed effects, interacted with urban area fixed effects. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.5: Emergency Admissions for Respiratory Diseases: Causal effect of Air Pollutants in Multi-Pollutant Models.

<i>Emergency Admissions for Respiratory Diseases per 100,000 inhabitants</i>								
<i>Models from 1 to 5 pollutants</i>								
	- 1 -	- 2 -		- 3 -		- 4 -		- 5 -
PM2.5	0.0006 (0.0010) [16,095]	-0.0014 (0.0015)		-0.0005 (0.0019)		0.0022 (0.0020)	-0.0004 (0.0020)	0.0009 (0.0024)
CO	0.0003*** (0.0001) [14,109]		0.0007*** (0.0002)	0.0007*** (0.0002)	0.0004 (0.0003)		0.0005** (0.0002)	0.0002 (0.0003)
O3	0.0039*** (0.0010) [15,968]		0.0078*** (0.0013)	0.0071*** (0.0016)	0.0060*** (0.0016)	0.0054*** (0.0018)	0.0064*** (0.0017)	0.0056*** (0.0019)
NO2	0.0050*** (0.0015) [14,875]				0.0014 (0.0027)	0.0038* (0.0021)	0.0037 (0.0024)	0.0026 (0.0027)
SO2	0.0852*** (0.0187) [14,820]	0.1030*** (0.0253)			0.0878*** (0.0310)	0.0760** (0.0313)		0.0957*** (0.0350)
Obs.	[in bracket]	11,573	9,027	8,513	8,935	6,992	7,795	6,135
Inst.	35	35	35	35	35	35	35	35

This Table presents the results of post-clustered-lasso IV models. In column labeled 1, each coefficient corresponds to a separate regression with one pollutant. Other columns correspond to a multi-pollutants regression. All regressions use the 35 instruments of the baseline model. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.6: Emergency Admissions for Cardiovascular Diseases: Causal effect of Air Pollutants in Multi-Pollutant Models.

<i>Emergency Admissions for Cardiovascular Diseases per 100,000 inhabitants</i>								
<i>Models from 1 to 5 pollutants</i>								
	- 1 -	- 2 -		- 3 -		- 4 -		- 5 -
PM2.5	0.0002 (0.0008) [16,095]	0.0011 (0.0011)		-0.0007 (0.0015)		0.0026 (0.0016)	-0.0007 (0.0017)	0.0008 (0.0021)
CO	0.0002*** (0.0001) [14,109]		0.0003*** (0.0001)	0.0004** (0.0002)	0.0007*** (0.0002)		0.0007*** (0.0002)	0.0007*** (0.0003)
O3	0.0007 (0.0009) [15,968]		0.0010 (0.0013)	0.0004 (0.0015)	0.0026* (0.0014)	0.0022 (0.0016)	0.0014 (0.0017)	0.0020 (0.0018)
NO2	0.0022** (0.0011) [14,875]				-0.0019 (0.0024)	0.0019 (0.0019)	-0.0024 (0.0023)	-0.0028 (0.0026)
SO2	0.0025 (0.0151) [14,820]	-0.0100 (0.0221)			-0.0284 (0.0293)	-0.0237 (0.0295)		-0.0250 (0.0342)
Obs.	[in bracket]	11,573	9,027	8,513	8,935	6,992	7,795	6,135
Inst.	35	35	35	35	35	35	35	35

This Table presents the results of post-clustered-lasso IV models. In column labeled 1, each coefficient corresponds to a separate regression with one pollutant. Other columns correspond to a multi-pollutants regression. All regressions use the 35 instruments of the baseline model. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.7: Mortality Rate for at least a Cardiovascular cause: Causal effect of Air Pollutants in Multi-Pollutant Models.

<i>Mortality (Cardiovascular cause) per 100,000 inhabitants</i>								
<i>Models from 1 to 5 pollutants</i>								
	- 1 -	- 2 -		- 3 -		- 4 -		- 5 -
PM2.5	0.0028*** (0.0007) [16,095]	0.0024*** (0.0009)		0.0029** (0.0014)		0.0033** (0.0013)	0.0039*** (0.0015)	0.0042*** (0.0016)
CO	0.0002*** (0.0001) [14,109]		0.0003** (0.0001)	0.0001 (0.0001)	0.0003* (0.0002)		-0.00003 (0.0002)	0.0001 (0.0002)
O3	-0.0008 (0.0007) [15,968]		0.0008 (0.0009)	0.0008 (0.0012)	0.0008 (0.0012)	0.0004 (0.0013)	0.0006 (0.0013)	0.0012 (0.0015)
NO2	0.0024*** (0.0008) [14,875]				-0.0003 (0.0017)	0.0007 (0.0014)	0.0023 (0.0017)	0.0007 (0.0019)
SO2	0.0407*** (0.0111) [14,820]	0.0267* (0.0159)			0.0333 (0.0204)	0.0328 (0.0216)		0.0196 (0.0238)
Obs.	[in bracket]	11,573	9,027	8,513	8,935	6,992	7,795	6,135
Inst.	35	35	35	35	35	35	35	35

This Table presents the results of post-clustered-lasso IV models. In column labeled 1, each coefficient corresponds to a separate regression with one pollutant. Other columns correspond to a multi-pollutants regression. All regressions use the 35 instruments of the baseline model. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.8: Mortality Rate for at least a Respiratory cause: Causal effect of Air Pollutants in Multi-Pollutant Models.

<i>Mortality (Respiratory cause) per 100,000 inhabitants</i>								
<i>Models from 1 to 5 pollutants</i>								
	- 1 -	- 2 -		- 3 -		- 4 -		- 5 -
PM2.5	0.0025*** (0.0004) [16,095]	0.0019*** (0.0006)		0.0023*** (0.0009)		0.0019** (0.0009)	0.0024** (0.0010)	0.0019* (0.0011)
CO	0.0002*** (0.0001) [14,109]		0.0002** (0.0001)	0.0001 (0.0001)	0.0002* (0.0001)		0.00004 (0.0001)	0.0001 (0.0001)
O3	-0.0014*** (0.0005) [15,968]		-0.0001 (0.0007)	0.0003 (0.0008)	-0.0006 (0.0008)	-0.0010 (0.0009)	-0.0001 (0.0009)	-0.0007 (0.0010)
NO2	0.0031*** (0.0006) [14,875]				-0.0007 (0.0014)	0.0012 (0.0009)	0.0016 (0.0013)	0.0004 (0.0014)
SO2	0.0316*** (0.0081) [14,820]	0.0230** (0.0109)			0.0361** (0.0142)	0.0305** (0.0148)		0.0381** (0.0159)
Obs.	[in bracket]	11,573	9,027	8,513	8,935	6,992	7,795	6,135
Inst.	35	35	35	35	35	35	35	35

This Table presents the results of post-clustered-lasso IV models. In column labeled 1, each coefficient corresponds to a separate regression with one pollutant. Other columns correspond to a multi-pollutants regression. All regressions use the 35 instruments of the baseline model. Significance: *p<0.1; **p<0.05; ***p<0.01

Table A.9: Main Results and Wild-bootstrap p-values.

	<i>Dependent variable:</i>			
	(Respiratory)	(Cardiovascular)	(Cardiovascular)	(Respiratory)
PM2.5	0.0009	0.0008	0.0042	0.0019
(Baseline)	p = 0.7118	p = 0.6828	p = 0.0091**	p = 0.0957
(Wild Bootstrap)	p = 0.7778	p = 0.7257	p = 0.0020***	p = 0.1772
CO	0.0002	0.0007	0.0001	0.0001
(Baseline)	p = 0.4215	p = 0.0091**	p = 0.4221	p = 0.5526
(Wild Bootstrap)	p = 0.4204	p = 0.0170*	p = 0.5596	p = 0.5195
O3	0.0056	0.0020	0.0012	-0.0007
(Baseline)	p = 0.0039**	p = 0.2831	p = 0.3945	p = 0.5115
(Wild Bootstrap)	p = 0.0000***	p = 0.3403	p = 0.3634	p = 0.4434
NO2	0.0026	-0.0028	0.0007	0.0004
(Baseline)	p = 0.3398	p = 0.2841	p = 0.6991	p = 0.7787
(Wild Bootstrap)	p = 0.5015	p = 0.0791	p = 0.7077	p = 0.7497
SO2	0.0957	-0.0250	0.0196	0.0381
(Baseline)	p = 0.0063**	p = 0.4642	p = 0.4082	p = 0.0166*
(Wild Bootstrap)	p = 0.0190*	p = 0.5405	p = 0.5245	p = 0.0390
Observations	6,135	6,135	6,135	6,135

Baseline p-values p are obtained by clustering by month-year-city cells and are compared to p-values obtained using an alternative wild-bootstrap inference. They are built with 1,000 bootstrap samples by bootstrapping by city and clustering standard errors by city. In this Table, we correct significance for testing 5 hypothesis - that is one per pollutant - separately for each outcome, using the Bonferroni-Holm method. * Null rejected with $\text{FWER} < 0.1$; ** $\text{FWER} < 0.05$

Table A.10: Causal effect of Air Pollutants on Mortality and Morbidity with Weather Controls.

<i>Multi Pollutant IV-Lasso Models</i>						
	<i>Emergency Admissions</i>			<i>Mortality</i>		
	Respiratory	Cardiovascular	Digestive	Respiratory	Cardiovascular	Digestive
PM2.5	0.0009 (0.0024)	0.0008 (0.0021)	-0.0036* (0.0018)	0.0019* (0.0011)	0.0042*** (0.0016)	0.0010 (0.0007)
CO	0.0002 (0.0003)	0.0007*** (0.0003)	-0.0001 (0.0002)	0.0001 (0.0001)	0.0001 (0.0002)	-0.00002 (0.0001)
O3	0.0056*** (0.0019)	0.0020 (0.0018)	-0.0026 (0.0017)	-0.0007 (0.0010)	0.0012 (0.0015)	0.0006 (0.0006)
SO2	0.0957*** (0.0350)	-0.0250 (0.0342)	-0.0049 (0.0296)	0.0381** (0.0159)	0.0196 (0.0238)	-0.0020 (0.0109)
NO2	0.0026 (0.0027)	-0.0028 (0.0026)	0.0010 (0.0021)	0.0004 (0.0014)	0.0007 (0.0019)	-0.0007 (0.0008)
Snow	-0.0030 (0.0066)	0.0092 (0.0065)	0.0036 (0.0059)	0.0014 (0.0036)	0.0125*** (0.0045)	0.0023 (0.0020)
Precipitations	-0.1695*** (0.0496)	-0.0048 (0.0513)	-0.0192 (0.0447)	-0.0026 (0.0298)	0.0722* (0.0396)	0.0135 (0.0181)
Precipitations ²	0.0331 (0.0347)	-0.0218 (0.0305)	-0.0471 (0.0306)	0.0308 (0.0206)	-0.0371 (0.0252)	0.0066 (0.0158)
Temperature	0.0012 (0.0033)	0.0004 (0.0030)	0.0036 (0.0028)	0.0075*** (0.0018)	0.0029 (0.0024)	-0.0002 (0.0010)
Temperature ²	-0.0005 (0.0004)	-0.0005 (0.0004)	0.0002 (0.0004)	-0.0003 (0.0002)	-0.0006* (0.0003)	0.0002 (0.0001)
Wind strength	0.0121 (0.0099)	0.0078 (0.0092)	-0.0003 (0.0077)	0.0079 (0.0048)	0.0049 (0.0069)	-0.0048* (0.0027)
Wind strength ²	-0.0008 (0.0010)	-0.0010 (0.0010)	-0.0007 (0.0008)	-0.0002 (0.0005)	0.0002 (0.0007)	-0.000004 (0.0003)
Humidity	0.0062*** (0.0017)	0.0002 (0.0016)	-0.0027** (0.0013)	-0.0005 (0.0009)	-0.0004 (0.0013)	0.0001 (0.0005)
Humidity ²	0.0001* (0.00005)	-0.00001 (0.00004)	-0.00002 (0.00004)	0.000003 (0.00003)	-0.00002 (0.00003)	-0.00003** (0.00001)
Sunlight	-0.0011*** (0.0004)	-0.0001 (0.0004)	-0.0007* (0.0004)	-0.0007*** (0.0002)	-0.0009*** (0.0003)	-0.0001 (0.0001)
Observations	[6,135]	[6,135]	[6,135]	[6,135]	[6,135]	[6,135]
Instruments	35	35	35	35	35	35

This Table presents the main results of post-clustered-lasso IV models with weather controls. Significance: *p<0.1; **p<0.05; ***p<0.01

8 Online Appendix

Table B.1: Instruments list.

	Specification	Dimensions
Planetary Boundary Layer		74
- Height	Daily average, 6 moments of the day	- 7
- Height Inverse	Daily average, 6 moments of the day	- 7
	Per urban area, 6 moments of the day	- 60
Thermal inversions		14
- #Hours	Daily average, and for 6 moments of the day	- 7
- Strength	Daily average, and for 6 moments of the day	- 7
“Layered” Variables		240
- Zonal wind	Daily Average for 60 altitude layers	- 60
- Meridional wind	Daily Average for 60 altitude layers	- 60
- Total strength	Daily Average for 60 altitude layers	- 60
- Altitude Pressure levels	Daily Average for 60 altitude layers	- 60
Total		328

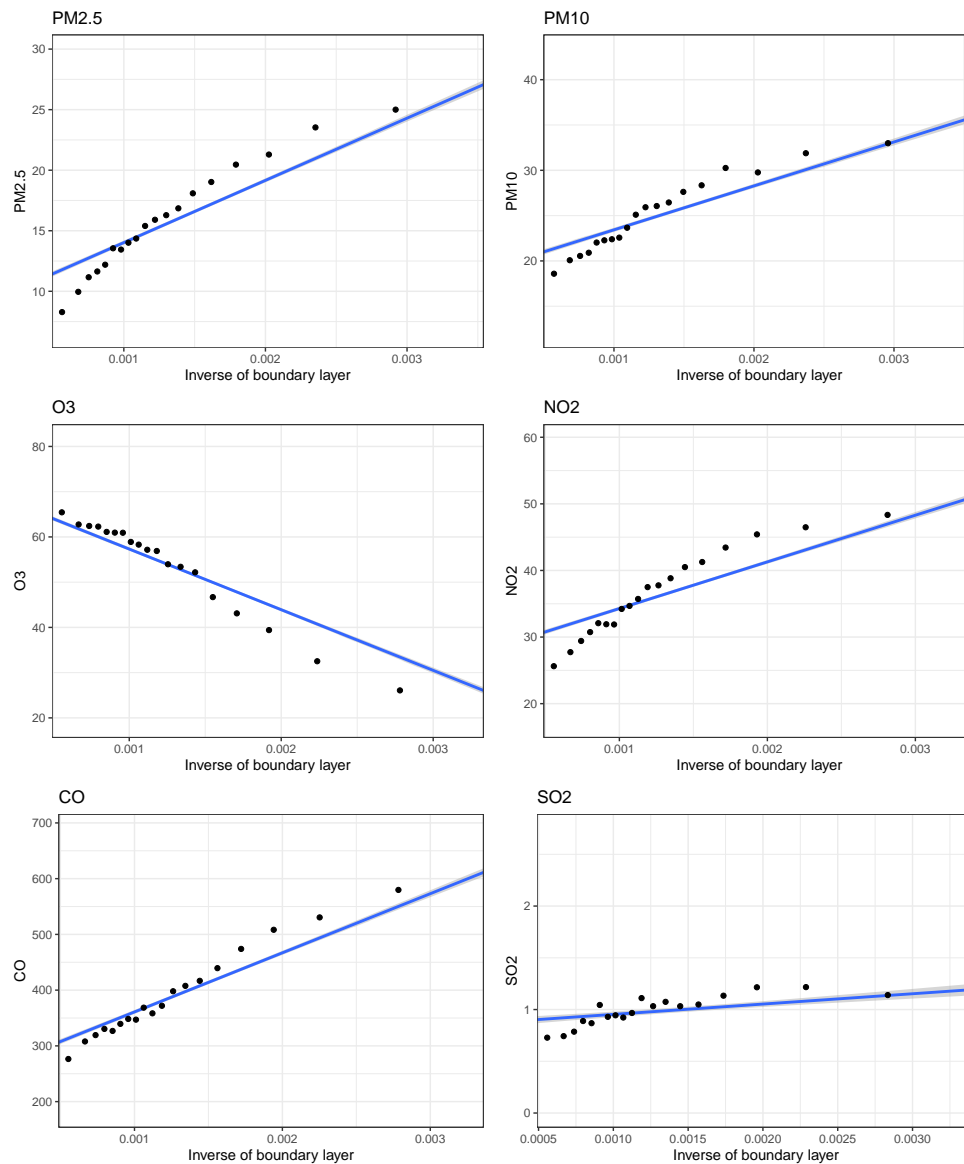


Figure B.1: Pollutant concentrations and Inverse Planetary Boundary Layer Height: Raw Correlations.

Note: On top of the regression line, for each quantile from p5 to p95 of unusual inverse boundary layer height, are represented the mean of unusual pollutant concentration.

Table B.2: Causal effect of Air Pollutants on Health Outcomes. Robustness checks to the specification of the Initial Set of Instruments.

	Emergency admissions									
	Respiratory Diseases					Cardiovascular Diseases				
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
PM2.5	0.0009 (0.0024)	0.0009 (0.0024)	0.0004 (0.0028)	0.0009 (0.0023)	0.0022 (0.0023)	0.0008 (0.0021)	0.0007 (0.0021)	-0.0005 (0.0021)	0.0008 (0.0021)	0.0019 (0.0021)
CO	0.0002 (0.0003)	0.0002 (0.0003)	0.0003 (0.0003)	0.0002 (0.0003)	0.0002 (0.0003)	0.0007*** (0.0003)	0.0006** (0.0002)	0.0008*** (0.0003)	0.0007*** (0.0003)	0.0006** (0.0003)
O3	0.0056*** (0.0019)	0.0057*** (0.0019)	0.0051** (0.0025)	0.0056*** (0.0019)	0.0050** (0.0020)	0.0020 (0.0018)	0.0014 (0.0018)	0.0006 (0.0019)	0.0019 (0.0019)	0.0019 (0.0020)
NO2	0.0026 (0.0027)	0.0027 (0.0027)	-0.0016 (0.0042)	0.0024 (0.0027)	0.0024 (0.0026)	-0.0028 (0.0026)	-0.0021 (0.0026)	-0.0057 (0.0035)	-0.0027 (0.0026)	-0.0031 (0.0026)
SO2	0.0957*** (0.0350)	0.0882*** (0.0335)	0.2000*** (0.0721)	0.0951*** (0.0348)	0.0678* (0.0361)	-0.0250 (0.0342)	-0.0310 (0.0334)	0.0315 (0.0531)	-0.0240 (0.0345)	-0.0349 (0.0346)
<i>Instruments</i>	Ref	Ref + City x TI	Ref - City x IPBLH	Ref + Humidity	Ref - Alt. Pressure	Ref	Ref + City x TI	Ref - City x IPBLH	Ref + Humidity	Ref - Alt. Pressure
Full Set	328	388	268	388	268	328	388	268	388	268
Selected Set	35	38	24	36	32	35	38	24	36	32
<i>Obs.</i>	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135
	Mortality with Respiratory Causes					Mortality with Cardiovascular Causes				
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
PM2.5	0.0019* (0.0011)	0.0017 (0.0011)	0.0022* (0.0012)	0.0019* (0.0011)	0.0018* (0.0011)	0.0042*** (0.0016)	0.0043*** (0.0016)	0.0047*** (0.0017)	0.0043*** (0.0016)	0.0043*** (0.0016)
CO	0.0001 (0.0001)	0.00004 (0.0001)	0.00002 (0.0001)	0.0001 (0.0001)	0.0002 (0.0001)	0.0001 (0.0002)	0.0001 (0.0002)	0.0001 (0.0002)	0.0002 (0.0002)	0.0002 (0.0002)
O3	-0.0007 (0.0010)	-0.0009 (0.0010)	-0.0004 (0.0011)	-0.0007 (0.0010)	-0.0003 (0.0010)	0.0012 (0.0015)	0.0012 (0.0014)	0.0016 (0.0015)	0.0013 (0.0015)	0.0016 (0.0016)
NO2	0.0004 (0.0014)	0.0006 (0.0014)	0.0015 (0.0018)	0.0004 (0.0014)	0.00004 (0.0014)	0.0007 (0.0019)	0.0013 (0.0019)	0.0011 (0.0024)	0.0007 (0.0019)	0.00004 (0.0019)
SO2	0.0381** (0.0159)	0.0351** (0.0153)	0.0192 (0.0271)	0.0382** (0.0159)	0.0325** (0.0160)	0.0196 (0.0238)	0.0099 (0.0229)	0.0141 (0.0399)	0.0177 (0.0241)	0.0164 (0.0244)
<i>Instruments</i>	Ref	Ref + City x TI	Ref - City x IPBLH	Ref + Humidity	Ref - Alt. Pressure	Ref	Ref + City x TI	Ref - City x IPBLH	Ref + Humidity	Ref - Alt. Pressure
Full Set	328	388	268	388	268	328	388	268	388	268
Selected	35	38	24	36	32	35	38	24	36	32
<i>Obs.</i>	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135	6,135

This Table presents the results of post-clustered-lasso IV models. We vary the initial set of instruments to compare with the reference presented in column (1). In column (2), we add the interactions between city and thermal inversions by moment of the day (6 moments of the day times 10 cities). In columns (3), we withdraw IBLH's same 60 interactions. In column (4), we add humidity measured in 60 altitude layers. In column (4), we withdraw altitude pressure variables measured in 60 altitude layers. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.3: Causal effect of Air Pollutants on Health Outcomes: Robustness Checks by Adding Successively Weather Controls.

	Emergency admissions									
	Respiratory Diseases					Cardiovascular Diseases				
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
PM2.5	0.0011 (0.0025)	0.0011 (0.0025)	0.0007 (0.0025)	0.0009 (0.0024)	0.0009 (0.0024)	0.0007 (0.0021)	0.0008 (0.0021)	0.0005 (0.0021)	0.0005 (0.0020)	0.0008 (0.0021)
CO	0.0001 (0.0003)	0.0002 (0.0003)	0.0002 (0.0003)	0.0002 (0.0003)	0.0002 (0.0003)	0.0006** (0.0003)	0.0006** (0.0003)	0.0007** (0.0003)	0.0007** (0.0003)	0.0007*** (0.0003)
O3	0.0056*** (0.0018)	0.0058*** (0.0019)	0.0058*** (0.0019)	0.0054*** (0.0019)	0.0056*** (0.0019)	0.0023 (0.0017)	0.0023 (0.0017)	0.0023 (0.0018)	0.0021 (0.0018)	0.0020 (0.0018)
NO2	0.0015 (0.0022)	0.0017 (0.0023)	0.0010 (0.0022)	0.0024 (0.0027)	0.0026 (0.0027)	-0.0030 (0.0023)	-0.0034 (0.0022)	-0.0037* (0.0022)	-0.0030 (0.0026)	-0.0028 (0.0026)
SO2	0.0834** (0.0334)	0.0998*** (0.0341)	0.1024*** (0.0348)	0.0972*** (0.0351)	0.0957*** (0.0350)	-0.0298 (0.0336)	-0.0198 (0.0336)	-0.0228 (0.0339)	-0.0244 (0.0340)	-0.0250 (0.0342)
Temperature	1	1	1	1	Ref.	1	1	1	1	Ref.
Humidity	1	1	1	1	Ref.	1	1	1	1	Ref.
Sunlight	0	1	1	1	Ref.	0	1	1	1	Ref.
Precipitations	0	0	1	1	Ref.	0	0	1	1	Ref.
Wind Strength	0	0	0	1	Ref.	0	0	0	1	Ref.
Observations	6,236	6,177	6,156	6,135	6,135	6,236	6,177	6,156	6,135	6,135
	Mortality with Respiratory Causes					Mortality with Cardiovascular Causes				
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
PM2.5	0.0014 (0.0011)	0.0016 (0.0012)	0.0018 (0.0012)	0.0019* (0.0011)	0.0019* (0.0011)	0.0031* (0.0016)	0.0032* (0.0017)	0.0034** (0.0017)	0.0039** (0.0016)	0.0042*** (0.0016)
CO	0.0001 (0.0001)	0.0001 (0.0001)	0.0001 (0.0001)	0.0001 (0.0001)	0.0001 (0.0001)	0.0002 (0.0002)	0.0002 (0.0002)	0.0002 (0.0002)	0.0002 (0.0002)	0.0001 (0.0002)
O3	-0.0003 (0.0009)	-0.0001 (0.0009)	-0.0002 (0.0010)	-0.0008 (0.0010)	-0.0007 (0.0010)	0.0019 (0.0013)	0.0020 (0.0013)	0.0019 (0.0014)	0.0014 (0.0014)	0.0012 (0.0015)
NO2	-0.0010 (0.0012)	-0.0005 (0.0012)	-0.0005 (0.0012)	0.0004 (0.0014)	0.0004 (0.0014)	-0.0002 (0.0016)	0.0006 (0.0016)	0.0006 (0.0016)	0.0008 (0.0019)	0.0007 (0.0019)
SO2	0.0368** (0.0153)	0.0396** (0.0159)	0.0405** (0.0158)	0.0374** (0.0158)	0.0381** (0.0159)	0.0174 (0.0230)	0.0147 (0.0235)	0.0161 (0.0235)	0.0188 (0.0238)	0.0196 (0.0238)
Temperature	1	1	1	1	Ref.	1	1	1	1	Ref.
Humidity	1	1	1	1	Ref.	1	1	1	1	Ref.
Sunlight	0	1	1	1	Ref.	0	1	1	1	Ref.
Precipitations	0	0	1	1	Ref.	0	0	1	1	Ref.
Wind Strength	0	0	0	1	Ref.	0	0	0	1	Ref.
Observations	6,236	6,177	6,156	6,135	6,135	6,236	6,177	6,156	6,135	6,135

This Table presents the results of post-clustered-lasso IV models. We vary the initial set of linear weather controls by adding controls one by one to compare with the reference presented in column (1). All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant clustered-lasso selection is performed, conditional on various weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations with the same weather controls. Standard errors are clustered at the month-year-urban area level. Significance: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table B.4: Causal effect of Air Pollutants Daily-Minimum and Daily-Maximum. Post-Lasso IVs.

Panel A: <i>Daily Maximum</i>				
	<i>Emergency Admissions</i>		<i>Mortality</i>	
	Respiratory	Cardiovascular	Respiratory	Cardiovascular
PM2.5	−0.0006 (0.0016)	0.0005 (0.0014)	0.0017** (0.0007)	0.0032*** (0.0010)
CO	0.0002 (0.0002)	0.0001 (0.0002)	0.00004 (0.0001)	−0.000004 (0.0001)
O3	0.0035** (0.0016)	−0.0012 (0.0016)	−0.0003 (0.0007)	0.0005 (0.0011)
NO2	−0.0025 (0.0025)	0.0012 (0.0022)	−0.0001 (0.0011)	−0.00001 (0.0016)
SO2	0.0215** (0.0097)	−0.0081 (0.0090)	0.0062 (0.0044)	0.0051 (0.0068)
Observations	[6,135]	[6,135]	[6,135]	[6,135]
Instruments	31	31	31	31
Panel B: <i>Daily Minimum</i>				
	<i>Emergency Admissions</i>		<i>Mortality</i>	
	Respiratory	Cardiovascular	Respiratory	Cardiovascular
PM2.5	−0.0062 (0.0043)	−0.0004 (0.0039)	−0.0003 (0.0020)	0.0035 (0.0029)
CO	0.0004 (0.0005)	0.0012** (0.0005)	−0.00004 (0.0003)	−0.0004 (0.0004)
O3	−0.0015 (0.0023)	−0.0004 (0.0021)	0.0002 (0.0012)	0.0009 (0.0016)
NO2	0.0016 (0.0056)	−0.0074 (0.0051)	0.0061** (0.0028)	0.0061 (0.0038)
SO2	0.1422 (0.1352)	−0.0156 (0.1270)	0.0757 (0.0605)	0.0698 (0.0772)
Observations	[6,135]	[6,135]	[6,135]	[6,135]
Instruments	38	38	38	38

This Table presents the results of post-clustered-lasso IV models. Significance: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$

Table B.5: Mortality From at Least One Respiratory Cause. From Single-pollutant to Multi-pollutant Models, Sample and Instruments Selection.

<i>Mortality rate (Respiratory)</i>				
<i>Models with 1 or 5 pollutants</i>				
	- 1 -	- 1 -	- 1 -	- 5 -
PM2.5	0.0026*** (0.0005)	0.0032*** (0.0008)	0.0035*** (0.0008)	0.0019* (0.0011)
CO	0.0002*** (0.00005)	0.0004*** (0.0001)	0.0003*** (0.0001)	0.0001 (0.0001)
O3	-0.0016*** (0.0005)	-0.0021*** (0.0007)	-0.0022*** (0.0008)	-0.0007 (0.0010)
NO2	0.0035*** (0.0006)	0.0036*** (0.0011)	0.0042*** (0.0011)	0.0004 (0.0014)
SO2	0.0289*** (0.0075)	0.0680*** (0.0179)	0.0718*** (0.0178)	0.0381** (0.0159)
Sample (Ap) or (B)				
Estimation	(Ap)	(B)	(B)	(B)
Instruments Selection	(Ap)	(Ap)	(B)	(B)
Model features				
Distinct IVs	5	5	5	1
Observations	14109 to 16095	6135	6135	6135
Instruments	19;17;17;20;9	19;17;17;20;9	15;3;10;14;3	35

Notes: Except for the last column, each coefficient is derived from a separate single-pollutant model. The last column corresponds to a multi-pollutant model. Samples (Ap) are pollutant-specific: each sample corresponds to the sample where the given pollutant concentration is not missing. Sample (B) is the sample where none of the five pollutants is missing. First, we partial out fixed effects: all variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed on the instrument selection sample, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations on the estimation sample. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.6: Mortality From at Least One Cardiovascular Cause. From Single-pollutant to Multi-pollutant Models, Sample and Instruments Selection.

<i>Mortality rate (Cardiovascular)</i>				
<i>Models with 1 or 5 pollutants</i>				
	- 1 -	- 1 -	- 1 -	- 5 -
PM2.5	0.0029*** (0.0007)	0.0048*** (0.0011)	0.0045*** (0.0011)	0.0042*** (0.0016)
CO	0.0002*** (0.0001)	0.0004*** (0.0001)	0.0004*** (0.0001)	0.0001 (0.0002)
O3	-0.0006 (0.0007)	-0.0017* (0.0010)	-0.0016 (0.0012)	0.0012 (0.0015)
NO2	0.0030*** (0.0009)	0.0055*** (0.0014)	0.0055*** (0.0016)	0.0007 (0.0019)
SO2	0.0366*** (0.0105)	0.0775*** (0.0258)	0.0659** (0.0262)	0.0196 (0.0238)
Sample (Ap) or (B)				
Estimation	(Ap)	(B)	(B)	(B)
Instruments Selection	(Ap)	(Ap)	(B)	(B)
Model features				
Distinct IVs	5	5	5	1
Observations	14109 to 16095	6135	6135	6135
Instruments	19;17;17;20;9	19;17;17;20;9	15;3;10;14;3	35

Notes: Except for the last column, each coefficient is derived from a separate single-pollutant model. The last column corresponds to a multi-pollutant model. Samples (Ap) are pollutant-specific: each sample corresponds to the sample where the given pollutant concentration is not missing. Sample (B) is the sample where none of the five pollutants is missing. First, we partial out fixed effects: all variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed on the instrument selection sample, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations on the estimation sample. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.7: Emergency admissions for respiratory diseases. From single-pollutant to multi-pollutant models: sample and instruments selection.

<i>Emergency admissions for respiratory diseases</i>				
<i>Models with 1 or 5 pollutants</i>				
	- 1 -	- 1 -	- 1 -	- 5 -
PM2.5	0.0005 (0.0010)	0.0012 (0.0018)	0.0007 (0.0018)	0.0009 (0.0024)
CO	0.0002** (0.0001)	0.0004** (0.0002)	0.0003 (0.0002)	0.0002 (0.0003)
O3	0.0035*** (0.0010)	0.0031** (0.0014)	0.0038** (0.0015)	0.0056*** (0.0019)
NO2	0.0033** (0.0015)	0.0048** (0.0019)	0.0063*** (0.0021)	0.0026 (0.0027)
SO2	0.0644*** (0.0188)	0.0873** (0.0382)	0.0840** (0.0385)	0.0957*** (0.0350)
Sample (Ap) or (B)				
Estimation	(Ap)	(B)	(B)	(B)
Instruments Selection	(Ap)	(Ap)	(B)	(B)
Model features				
Distinct IVs	5	5	5	1
Observations	14109 to 16095	6135	6135	6135
Instruments	19;17;17;20;9	19;17;17;20;9	15;3;10;14;3	35

Notes: Except for the last column, each coefficient is derived from a separate single-pollutant model. The last column corresponds to a multi-pollutant model. Samples (Ap) are pollutant-specific: each sample corresponds to the sample where the given pollutant concentration is not missing. Sample (B) is the sample where none of the five pollutants is missing. First, we partial out fixed effects: all variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed on the instrument selection sample, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations on the estimation sample. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.8: Emergency admissions for cardiovascular diseases. From single-pollutant to multi-pollutant models: sample and instruments selection.

Pollutant in the model:	<i>Emergency admissions for cardiovascular diseases</i>			
	(1)	(1)	(1)	(5)
PM2.5	0.0001 (0.0008)	0.0019 (0.0016)	0.0018 (0.0015)	0.0008 (0.0021)
CO	0.0002** (0.0001)	0.0004** (0.0002)	0.0004* (0.0002)	0.0007*** (0.0003)
O3	0.0009 (0.0009)	0.0002 (0.0015)	0.0003 (0.0016)	0.0020 (0.0018)
NO2	0.0023* (0.0012)	0.0030 (0.0019)	0.0040** (0.0020)	−0.0028 (0.0026)
SO2	0.0055 (0.0143)	−0.0127 (0.0339)	−0.0242 (0.0379)	−0.0250 (0.0342)
Sample (Ap) or (B)				
Estimation	(Ap)	(B)	(B)	(B)
Instruments Selection	(Ap)	(Ap)	(B)	(B)
Model features				
Distinct IVs	5	5	5	1
Observations	14109 to 16095	6135	6135	6135
Instruments	19;17;17;20;9	19;17;17;20;9	15;3;10;14;3	35

Notes: Except for the last column, each coefficient is derived from a separate single-pollutant model. The last column corresponds to a multi-pollutant model. Samples (Ap) are pollutant-specific: each sample corresponds to the sample where the given pollutant concentration is not missing. Sample (B) is the sample where none of the five pollutants is missing. First, we partial out fixed effects: all variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed on the instrument selection sample, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations on the estimation sample. Standard errors are clustered at the month-year-urban area level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.9: Causal effect of Air Pollutants on Emergency Admissions Outcomes By Age Group. Post-Lasso IVs models.

<i>Emergency Admissions for Respiratory Diseases</i>										
	(0-4)	(5-14)	(15-59)	(60-79)	(≥ 80)	(0-4)	(5-14)	(15-59)	(60-79)	(≥ 80)
O3	0.027 p = 0.111	0.001 p = 0.738	0.002 p = 0.150	0.012** p = 0.016	0.015 p = 0.397	0.021* p = 0.071	0.003** p = 0.046	0.001* p = 0.054	0.006** p = 0.032	0.021** p = 0.030
SO2	0.702** p = 0.025	0.036 p = 0.513	0.001 p = 0.977	0.069 p = 0.472	0.783** p = 0.015	0.428** p = 0.028	0.062** p = 0.046	0.017 p = 0.147	0.063 p = 0.227	0.574*** p = 0.002
PM2.5	-0.013 p = 0.545	-0.003 p = 0.329	0.001 p = 0.705	0.009* p = 0.085	0.001 p = 0.950					
CO	-0.001 p = 0.578	0.0005 p = 0.254	0.00002 p = 0.899	0.001 p = 0.382	0.002 p = 0.335					
NO2	0.055** p = 0.017	0.0001 p = 0.984	0.002 p = 0.315	-0.004 p = 0.561	-0.031 p = 0.217					
Observations	6,135	6,135	6,135	6,135	6,135	11,416	11,416	11,416	11,416	11,416
Mean Dep. Var	5.5	0.5	0.5	1.9	7.3	5.5	0.5	0.5	1.9	7.3
Instruments	35	35	35	35	35	26	26	26	26	26
<i>Emergency Admissions for Cardiovascular Diseases</i>										
	(0-14)	(15-59)	(60-79)	(≥ 80)		(0-14)	(15-59)	(60-79)	(≥ 80)	
CO	-0.00004 p = 0.816	0.0002 p = 0.115	0.001 p = 0.113	0.007* p = 0.055		0.00004 p = 0.481	0.00004 p = 0.457	-0.0003 p = 0.416	0.004*** p = 0.002	
PM2.5	0.001 p = 0.680	-0.001 p = 0.648	-0.009 p = 0.189	0.037 p = 0.182						
O3	-0.001 p = 0.335	0.0003 p = 0.813	0.0003 p = 0.964	0.032 p = 0.168						
NO2	-0.001 p = 0.527	-0.001 p = 0.625	-0.002 p = 0.818	-0.035 p = 0.288						
SO2	0.002 p = 0.951	-0.018 p = 0.427	0.021 p = 0.843	-0.107 p = 0.802						
Observations	6,135	6,135	6,135	6,135		14,109	14,109	14,109	14,109	
Mean Dep. Var	0.1	0.6	3.5	13.2		0.1	0.6	3.5	13.2	
Instruments	35	35	35	35		17	17	17	17	

Note: Each column correspond to a multi-pollutants post-clustered-lasso IV-regression. Before all regressions, we partial out fixed effects. All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations. Standard errors are clustered at the month-year-city level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.10: Causal effect of Air Pollutants on Mortality By Age Group. Post-Lasso IVs models.

	<i>Emergency Admissions for the Mortality Rate</i>							
	(0-14)	(15-59)	(60-79)	(≥ 80)	(0-14)	(15-59)	(60-79)	(≥ 80)
PM2.5	0.0002 p = 0.899	0.001 p = 0.526	0.013* p = 0.056	0.047 p = 0.149	0.0004 p = 0.564	0.001 p = 0.163	0.005 p = 0.306	0.052*** p = 0.007
SO2	0.012 p = 0.535	0.019 p = 0.360	0.025 p = 0.830	1.020** p = 0.050	0.006 p = 0.598	0.002 p = 0.893	0.083 p = 0.195	0.411 p = 0.153
CO	0.0001 p = 0.536	0.00002 p = 0.888	-0.0001 p = 0.900	0.003 p = 0.414				
O3	-0.0004 p = 0.764	-0.001 p = 0.352	-0.003 p = 0.633	0.012 p = 0.684				
NO2	-0.003* p = 0.089	-0.002 p = 0.349	0.006 p = 0.521	-0.002 p = 0.966				
Observations	6,135	6,135	6,135	6,135	11,487	11,487	11,487	11,487
Mean Dep. Var	0.1	0.5	4	22.1	0.1	0.5	4	22.1
Instruments	35	35	35	35	27	27	27	27

Note: Each column correspond to a multi-pollutants post-clustered-lasso IV-regression. Before all regressions, we partial out fixed effects. All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. A first step of per-pollutant lasso selection is performed, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations. Standard errors are clustered at the month-year-city level. Significance: *p<0.1; **p<0.05; ***p<0.01

Table B.11: Lagged effects. Post-lasso IVs.

	Respiratory Emergencies		Cardiovascular Emergencies		Cardiovascular Mortality		Respiratory Mortality	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
O3 (t+1)		0.0003 (0.0023)						
O3 (t)	0.0041** (0.0019)	0.0015 (0.0022)						
O3 (t-1)	0.0008 (0.0019)	-0.0003 (0.0011)						
O3 (t-2)	0.0008 (0.0017)							
CO (t+1)				-0.0001 (0.0002)				
CO (t)	0.0006** (0.0003)		0.0002** (0.0001)	0.0003* (0.0002)				
CO (t-1)	0.0002 (0.0003)		0.00003 (0.0001)	0.00001 (0.0001)				
CO (t-2)	0.0002 (0.0003)		-0.00002 (0.0001)					
PM2.5 (t+1)						-0.0001 (0.0020)		
PM2.5 (t)					0.0037** (0.0015)	0.0042* (0.0024)		
PM2.5 (t-1)					-0.0006 (0.0016)	-0.0011 (0.0019)		
PM2.5 (t-2)					-0.0007 (0.0012)	-0.0002 (0.0014)		
SO2 (t+1)		0.0367 (0.0469)				-0.0078 (0.0238)		0.0035 (0.0155)
SO2 (t)	0.0463* (0.0277)	0.0378 (0.0328)			0.0032 (0.0183)	0.0053 (0.0203)	0.0211** (0.0101)	0.0203* (0.0119)
SO2 (t-1)	-0.0012 (0.0258)	0.0010 (0.0184)			0.0053 (0.0159)	0.0126 (0.0183)	0.0058 (0.0095)	0.0054 (0.0102)
SO2 (t-2)	0.0052 (0.0285)				0.0067 (0.0145)	-0.0028 (0.0149)	-0.0002 (0.0090)	-0.0015 (0.0092)
NO2 (t-1)								
NO2 (t)	-0.0007 (0.0036)							
NO2 (t-1)	-0.0011 (0.0036)							
NO2 (t-2)	-0.0021 (0.0027)							
Observations	4,144	3,652	11,676	11,672	6,885	5,783	10,963	10,036

Before all regressions, we partial out fixed effects. All variables are first regressed on month-year and day-of-the-week fixed effects both interacted with urban area fixed effects and then replaced by the corresponding residuals. Compared to the contemporaneous IV equation, the set of instruments is the same, but is inflated with all instruments' lags, before selection. A first step of per-pollutant lasso selection is performed, conditional on weather variables which are forced into the model (no selection), selected instruments are then pooled and enter a regular IV estimations. Standard errors are clustered at the city-level. *p<0.1; **p<0.05; ***p<0.01

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G2015/08	Q. LAFFÈTER - M. PAK Élasticités des recettes fiscales au cycle économique : étude de trois impôts sur la période 1979-2013 en France	B. CAMPAGNE - A. POISSONNIER Structural reforms in DSGE models: a case for sensitivity analyses	G2016/06	B. CAMPAGNE - A. POISSONNIER Structural reforms in DSGE models: a case for sensitivity analyses	C.-M. CHEVALIER - A. LUCIANI Computerization, labor productivity and employment: impacts across industries vary with technological level	G2019/06	J.-P. CLING - S. EGHBAL-TEHERANI - M. ORZONI - C. PLATEAU The Differences between EU Countries for Sustainable Development Indicators: It is (mainly) the Economy!
G2015/09	J.-M. DAUSSIN-BENICHOU, S. IDMACHICHE, A. LEDUC et E. POULIQUEN Les déterminants de l'attractivité de la fonction publique de l'État	Y. DUBOIS et M. KOUBI Relèvement de l'âge de départ à la retraite : quel impact sur l'activité des séniors de la réforme des retraites de 2010 ?	G2016/07	Y. DUBOIS et M. KOUBI Relèvement de l'âge de départ à la retraite : quel impact sur l'activité des séniors de la réforme des retraites de 2010 ?	R. MONIN - M. SUAREZ CASTILLO L'effet du CICE sur les prix : une double analyse sur données sectorielles et individuelles	G2019/07	P. CHONÉ - L. WILNER Competition on Unobserved Attributes: The Case of the Hospital Industry
G2015/10	P. AUBERT La modulation du montant de pension selon la durée de carrière et l'âge de la retraite : quelles disparités entre assurés ?	A. NAOUAS - M. ORAND - I. SLIMANI HOULTI Les entreprises employant des salariés au Smic : quelles caractéristiques et quelle rentabilité ?	G2016/08	A. NAOUAS - M. ORAND - I. SLIMANI HOULTI Les entreprises employant des salariés au Smic : quelles caractéristiques et quelle rentabilité ?	R. LARDEUX Who Understands The French Income Tax? Bunching Where Tax Liabilities Start		
G2015/11	V. DORTET-BERNADET - M. SICSIC Effet des aides publiques sur l'emploi en R&D dans les petites entreprises	T. BLANCHET - Y. DUBOIS - A. MARINO - M. ROGER Patrimoine privé et retraite en France	G2016/09	T. BLANCHET - Y. DUBOIS - A. MARINO - M. ROGER Patrimoine privé et retraite en France	M. PAK - A. POISSONNIER Accounting for technology, trade and final		
G2016/01	M. PAK - A. POISSONNIER Accounting for technology, trade and final	M. PAK - A. POISSONNIER Accounting for technology, trade and final	G2016/10	M. PAK - A. POISSONNIER Accounting for technology, trade and final			
G2016/02	M. PAK - A. POISSONNIER Accounting for technology, trade and final	M. PAK - A. POISSONNIER Accounting for technology, trade and final	G2016/11	M. PAK - A. POISSONNIER Accounting for technology, trade and final			

G2017/01	D. FOUGERE - E. GAUTIER - S. ROUX Understanding Wage Floor Setting in Industry-Level Agreements: Evidence from France	G2017/01	D. FOUGERE - E. GAUTIER - S. ROUX Understanding Wage Floor Setting in Industry-Level Agreements: Evidence from France	consumption in employment: an Input-Output decomposition	G2018/05	C.-M. CHEVALIER Financial constraints of innovative firms and sectoral growth
G2017/02	Y. DUBOIS - M. KOUBI Règles d'indexation des pensions et sensibilité des dépenses de retraites à la croissance économique et aux chocs démographiques	G2017/02	Y. DUBOIS - M. KOUBI Règles d'indexation des pensions et sensibilité des dépenses de retraites à la croissance économique et aux chocs démographiques	Y. DUBOIS - M. KOUBI Règles d'indexation des pensions et sensibilité des dépenses de retraites à la croissance économique et aux chocs démographiques	G2018/06	R. S.-H. LEE - M. PAK Pro-competitive effects of globalisation on prices, productivity and markups: Evidence in the Euro Area
G2017/03	A. CAZENAVE-LACROUITZ - F. GODET L'espérance de vie en retraite sans incapacité sévère des générations nées entre 1960 et 1990 : une projection à partir du modèle Desimie	G2017/03	A. CAZENAVE-LACROUITZ - F. GODET L'espérance de vie en retraite sans incapacité sévère des générations nées entre 1960 et 1990 : une projection à partir du modèle Desimie	A. CAZENAVE-LACROUITZ - F. GODET L'espérance de vie en retraite sans incapacité sévère des générations nées entre 1960 et 1990 : une projection à partir du modèle Desimie	G2018/07	C.-M. CHEVALIER Consumption inequality in France between 1995 and 2011
G2017/04	J. BARDAJ - B. CAMPAGNE - M.-B. KHDER - Q. LAFFÈTER - O. SIMON (Insee)	G2017/04	J. BARDAJ - B. CAMPAGNE - M.-B. KHDER - Q. LAFFÈTER - O. SIMON (Insee)	J. BARDAJ - B. CAMPAGNE - M.-B. KHDER - Q. LAFFÈTER - O. SIMON (Insee)	G2018/08	A. BAUER - B. GARBINTI - S. GEORGES-KOT Financial Constraints and Self-Employment in France, 1945-2014
G2017/05	A.-S. DUFERNEZ - C. ELEZAAR - P. LEBLANC - E. MASSON - H. PARTOUCHE (DG-Trésor)	G2017/05	A.-S. DUFERNEZ - C. ELEZAAR - P. LEBLANC - E. MASSON - H. PARTOUCHE (DG-Trésor)	A.-S. DUFERNEZ - C. ELEZAAR - P. LEBLANC - E. MASSON - H. PARTOUCHE (DG-Trésor)	G2018/09	P. BEAUMONT - A. LUCIANI Prime à l'embauche dans les PME : évaluation à partir des déclarations d'embauche
G2017/06	A. CAZENAVE-LACROUITZ - A. GODZINSKI Effects of the one-day waiting period for sick leave on health-related absences in the French central civil service	G2017/06	A. CAZENAVE-LACROUITZ - A. GODZINSKI Effects of the one-day waiting period for sick leave on health-related absences in the French central civil service	A. CAZENAVE-LACROUITZ - A. GODZINSKI Effects of the one-day waiting period for sick leave on health-related absences in the French central civil service	G2018/10	C. BELLÉGO - V. DORTET-BERNADET - M. TEPAUT Comparaison de deux dispositifs d'aide à la R&D collaborative public-privé
G2017/07	P. CHARNNOZ - M. ORAND Qualification, progrès technique et marchés du travail locaux en France, 1990-2011	G2017/07	P. CHARNNOZ - M. ORAND Qualification, progrès technique et marchés du travail locaux en France, 1990-2011	P. CHARNNOZ - M. ORAND Qualification, progrès technique et marchés du travail locaux en France, 1990-2011	G2018/11	R. MONIN - M. SUAREZ CASTILLO Replication et rapprochement des travaux d'évaluation de l'effet du CICE sur l'emploi en 2013 et 2014
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G2017/09	C.-M. CHEVALIER - R. LARDEUX Homeownership and labor market outcomes: disentangling externality and composition effects	G2017/09	C.-M. CHEVALIER - R. LARDEUX Homeownership and labor market outcomes: disentangling externality and composition effects	C.-M. CHEVALIER - R. LARDEUX Homeownership and labor market outcomes: disentangling externality and composition effects	G2019/01	M. ANDRÉ - A.-L. BIOTTEAU Effets de moyen terme d'une hausse de TVA sur le niveau de vie et les inégalités : une approche par microsimulation
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