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Poisoned Air, Shortened Lives:

$PM_{2.5}$ Exposure and Premature Mortality in Southern European Cities

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Abstract

This study examines the causal impact of $PM_{2.5}$ air pollution exposure on premature mortality in Southern European cities from 2010 to 2018. To address endogeneity, we leverage local variations in rainfall as a source of random variation in $PM_{2.5}$ exposure. Using the Two-Sample Two-Stage Least Squares (TS2SLS) estimator to reconcile monitoring station-level and city-level data, our findings reveal a statistically significant increase in premature mortality caused by $PM_{2.5}$. According to our preferred specification, a 1% increase in $PM_{2.5}$ causes a 1.13% rise in the under-65 mortality rate and a 1.41% rise in the infant mortality rate. The results are robust to alternative specifications. The most affected populations are those residing in urban areas (relative to suburban areas) and individuals living in cities located in richer regions (as opposed to poorer ones).

Keywords: air pollution, $PM_{2.5}$, cities, premature mortality, TS2SLS.

JEL classification: I18, Q53, Q58.

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1 Introduction

Air pollution poses a major public health risk, contributing to premature mortality and to a broad spectrum of critical illnesses, including cardiovascular and respiratory diseases, lung cancer, and increased susceptibility to infections (e.g., Pope & Dockery (2012)). Its impact is particularly severe in urban environments (e.g., Graff Zivin & Neidell (2013); He et al. (2019); Giaccherini et al. (2021)). Both short- and long-term exposure to air pollutants are associated with a range of adverse health outcomes, with vulnerable individuals (such as those with pre-existing health problems, children, the elderly, and the socioeconomically disadvantaged) disproportionately affected (e.g., Neidell (2004); Deryugina et al. (2019); DeCicca & Malak (2020); Giaccherini et al. (2021); Palma et al. (2022), among others).

Despite policy efforts to reduce emissions, evidenced by significant declines in key pollutants[1], challenges to further ameliorate air quality persist, especially in cities where pollution levels still exceed the limits recommended by international agencies[2]. In 2022, 96% of the EU urban population was exposed to particulate matter concentrations surpassing the thresholds defined by the World Health Organization (WHO) guidelines, with limits being breached in more than 100 cities across the continent (European Commission 2022).

Focusing on cities is important. Although urban areas represent just 4% of the EU territory, 75% of the EU population lives in cities, intensifying exposure risks. According to the European Environment Agency (2022*b*), in 2020, air pollution contributed to a

[1]See, e.g., EU Directives in European Parliament and Council (2008), (2010), (2016). The European Environmental Agency estimates, for instance, an improvement in air quality, with a 22% drop in particulate matter associated with a reduction in mortality by 45%. See European Environment Agency (2022*a*) and European Environment Agency (2024).

[2]The WHO (2021) thresholds are defined as follows: $PM_{2.5}$: 5 µg/m³ (annual), 15 µg/m³ (24-hour); PM_{10}: 15 µg/m³ (annual), 45 µg/m³ (24-hour); NO_2: 10 µg/m³ (annual), 25 µg/m³ (24-hour); SO_2: 40 µg/m³ (24-hour); CO: 4 µg/m³ (24-hour); O_3: 100 µg/m³ (8-hour), 60 µg/m³ (peak season).

substantial number of premature deaths across EU-27 Member States[3]. According to Khomenko et al. (2021), adherence to WHO air quality standards could prevent approximately 30 deaths per 100,000 urban residents annually.

In this paper, we examine the causal effect of exposure to $PM_{2.5}$ on premature mortality in European cities. Particulate matter (PM), particularly the fine particulate matter smaller than 2.5 micrometers ($PM_{2.5}$), is among the most harmful pollutants due to its ability to infiltrate deep into the lungs and bloodstream (e.g., Chay & Greenstone (2003); World Health Organization - WHO (2013); Pui et al. (2014); Fan et al. (2020)). Urban concentrations of $PM_{2.5}$ are especially high, with the most important sources represented by traffic, industrial activity, and heating systems[4].

The adverse health effects of air pollution are well-documented by a robust literature establishing causal links between exposure and health outcomes across demographic groups, regions, and periods. However, much of the existing literature has examined the health effects of pollutants different from fine particulate matter < 2.5, like Particulate Matter (PM_{10}), Nitrogen Dioxide (NO_2), Sulfur Dioxide (SO_2), Carbon Monoxide (CO) and Ozone (O_3). Early evidence from Chay et al. (2003) on the 1970 Clean Air Act shows reduced Total Suspended Particulate (TSP) but no clear link to adult or elderly mortality. On the contrary, studies, like, e.g., Chay & Greenstone (2003), Currie & Neidell (2005), Knittel et al. (2016), Cesur et al. (2017), find a link between infant mortality and pollutants such as TSP, CO, and PM. More recent research shifts the focus from mortality to acute effects, with, e.g., Neidell (2004) showing CO's impact on childhood asthma. Bauernschuster et al. (2017) exploit transit strikes in Germany, finding that 11-13% more

[3] $PM_{2.5}$ concentrations exceeding WHO (2021) guidelines accounted for 238,000 premature deaths, with NO_2 and acute O_3 exposure linked to 49,000 and 24,000 additional fatalities, respectively.

[4] In urban areas common pollutants also include Particulate Matter (PM_{10}), Nitrogen Dioxide (NO_2), Sulfur Dioxide (SO_2), Carbon Monoxide (CO), Ozone (O_3), and Volatile organic compounds (VOCs). See Dominici et al. (2014) and European Environment Agency (2021).

traffic, implies 14% increases in PM_{10}/NO_2, and 11% higher respiratory hospitalizations, especially in children. Similarly, Bauernschuster et al. (2017) and Schlenker & Walker (2016) link traffic and airport emissions to pediatric and asthma-related hospital admissions. Instrumental variable methods improve causal inference: Deryugina et al. (2019) use wind shifts to show $PM_{2.5}$ raises mortality among 25% of the U.S. elderly; to our knowledge, this is the only study that specifically analyzes the impact of fine particulate matter on mortality in the U.S. Also Palma et al. (2022) leverage strikes as exogenous shocks, finding that a one-standard-deviation increase in PM_{10} raises hospitalizations by 0.79 per 100,000, especially among vulnerable and younger groups. New research reveals specific vulnerabilities: Fan et al. (2023) identifies SO_2 as a cardiovascular mortality driver, especially in children under 5 years, whereas Cakaj et al. (2023) quantify growing ozone threats through a 1.3 per million inhabitant rise in annual premature deaths.

Our analysis is focused on the impact of $PM_{2.5}$ in Southern European cities during the period 2010-2018. We examine two distinct mortality outcomes: (1) premature mortality from circulatory and respiratory diseases among the under 65, and (2) infant mortality, i.e., deaths of live-born children under one year of age. We focus on these mortality indicators due to their well-established link to $PM_{2.5}$ exposure, as documented in prior studies (e.g., Loomis et al. (1999); Pope et al. (2009); Brook et al. (2010); Yixing et al. (2016); Heft-Neal et al. (2020); Ortigoza et al. (2021)). Cities represent a critical setting for examining the health impacts of air pollution. We focus on cities in Italy, Spain, Greece, Croatia, and Portugal - countries identified by the European Environment Agency (EEA) as experiencing some of the most severe air pollution problems in Europe, particularly concerning $PM_{2.5}$ concentrations.[5] We combine measures of air pollution from ground-

[5]The highest $PM_{2.5}$ levels in Europe are recorded in Eastern Europe and Northern Italy. In many Central and Eastern European countries, this stems from the widespread use of solid fuels (e.g., coal for residential heating, industrial processes, and power generation). The Po Valley (in Northern Italy) also represents a critical hotspot due to dense industrialization,

based monitoring stations with statistics on premature mortality. To identify the causal effect of exposure to $PM_{2.5}$, we leverage local variations in rainfall as a source of random variation in air pollution exposure.[6] Dry weather conditions, coupled with intense local economic activity, can sustain concentration levels above recommended limits for several days. Our identification strategy exploits yearly within-city variations in precipitation, assuming they are randomly distributed across cities and relies on the fact that, according to the literature (e.g., Shukla et al. (2008), Sundar et al. (2020), Tripathi et al. (2021)), rain can effectively *clean* the air from pollution. We conduct several robustness checks to rule out potential violations of the exclusion restrictions.

We employ the Two-Sample-Two-Stage-Least-Squares (TS2SLS) estimator proposed by Angrist & Krueger (1992) to properly consider station-level pollution data and city-level mortality outcomes, avoiding aggregation bias. This estimator allows for obtaining consistent instrumental variables estimation when the outcome and the instrument (but not the endogenous variable) are observed in one sample and the endogenous and the instrument (but not the outcome) are observed in a second different sample. In our case, we can observe the instrument, the endogenous, and the outcome in the sample at the city level. Still, we can observe only the instrument and the endogenous in the station-level sample.

Our results show a positive and statistically significant effect of $PM_{2.5}$ on both premature mortality indicators. We find that a one-unit increase in $PM_{2.5}$—corresponding to 7.5% of the average level of $PM_{2.5}$—implies an increase in the under 65 mortality rate and the

high population density, and geographical conditions that trap pollutants. This area consistently exceeds the EU $PM_{2.5}$ limit ($15\mu g/m^3$, 24-hour), European Environment Agency (2021). Due to insufficient mortality data for Eastern European cities, we prioritize Southern Europe in this study.

[6] More generally, weather conditions have been considered by the literature as a source of randomness. For instance, changes in wind direction have been employed as an instrument for SO_2 concentrations (Yang et al. 2017), while local weather conditions have been utilized for PM_{10} and CO (Knittel et al. 2016). Thermal inversions, likely to cause a temporary accumulation of certain pollutants, have also served as instruments for PM_{10}, CO, SO_2, and O_3 (Arceo et al. 2016). More recently, precipitations have been used as an instrument for PM_{10} exposure during the prenatal period (see Palma et al. (2022)). Following this literature, we employ precipitation as an instrumental variable for air pollution.

infant mortality rate of 0.2887 and 0.3134, corresponding to 8.49% and 10.57% of the average values. These marginal effects correspond to elasticities of approximately 1.13 (under 65 mortality rate) and 1.41 (infant mortality rate), indicating disproportionate sensitivity to air pollution, particularly among infants. Strong effects are also observed for PM_{10} and NO_2 exposure (4.9-6.0% increases and elasticities 1.19-1.51, respectively). Robustness checks using multiple weather-based instruments confirm these results, particularly for working-age populations. Spatial heterogeneity reveals urban areas face more severe mortality impacts relative to suburban areas, while a paradoxical socioeconomic gradient emerges: lower-poverty-risk cities exhibit higher pollution vulnerability, likely due to concentrated exposure.

We contribute to the existing literature in several ways. First, our analysis focuses on cities, a recognised pollution hotspot, and, unlike existing studies that typically examine single cities or countries (e.g., Deryugina et al. (2019)), we analyze a sample of southern European cities which contribute to enhancing the external validity of our findings. Second, we focus on under-65 mortality, which helps to avoid the common issue of 'mortality displacement' (or 'harvesting'), i.e., the fact that pollution-related deaths may reflect the acceleration of deaths that would have occurred shortly anyway, especially among the elderly or those with preexisting conditions. In addition, for comparison purposes, we examine infant mortality (0-1 years of age), a measure more commonly used in the literature (e.g., Chay & Greenstone (2003), Currie & Neidell (2005), Knittel et al. (2016), Cesur et al. (2017), DeCicca & Malak (2020)). Moreover, our mortality variables encompass deaths due to both respiratory and cardiovascular causes. Building on this, we extend the analysis beyond respiratory outcomes, commonly examined in earlier studies (e.g., Neidell (2004), Schlenker & Walker (2016), Giaccherini et al. (2021)). Third, we concentrate on

fine particulate matter ($PM_{2.5}$), the pollutant more closely linked to serious health issues and premature deaths due to its ability to infiltrate deep into the lungs and the bloodstreams. While most European studies have examined PM_{10} (e.g., Palma et al. (2022), Bauernschuster et al. (2017), Giaccherini et al. (2021)), research on $PM_{2.5}$—particularly in the U.S.—has largely focused on elderly populations (Arceo et al. (2016), Deryugina et al. (2019)). Fourth, in terms of methodology, we implement a TS2SLS approach to address potential measurement errors from aggregating station-level pollution and weather data to the city level. This method allows consistent estimation even when the endogenous regressor is only partially observed and helps to mitigate bias from discrepancies between local measurements and city-wide averages.

2 Data and samples

2.1 Data

We construct a geospatial data set by combining data on (i) premature mortality for cities in Southern European countries (Italy, Spain, Greece, Portugal, and Croatia) over the years 2010-2018 from Urban Audit (UA) with (ii) pollution and weather data from the Airbase and Climate Data Records (CDR) of the European Environmental Agency and the National Centers for Environmental Information, respectively.

Premature mortality. The UA database provides information on the quality of life in cities, considered a local administrative unit where the majority of the population lives in an urban center of at least 50,000 inhabitants. The dataset includes information on cities and their commuting zones (the so-called Functional Urban Areas). While the dataset collects comprehensive information across multiple domains (demography, housing, health,

labor market, education, environment, transport, and tourism), substantial missing values persist across variables. Our analysis focuses exclusively on the health components where coverage is sufficiently complete for our purposes. As of 2020, the UA dataset contains 980 "cities" and 49 "greater cities" in 31 European countries.[7] From these data, we gather two measures of premature mortality available at the city level: (i) mortality rate among individuals under 65 due to circulatory and respiratory diseases, measured as deaths in this category per 100,000 of the total population, and (ii) infant mortality rate, defined as the number of deaths of live-born children under one year of age per 1,000 live births.

Pollution and weather. The Airbase database of the European Environmental Agency (EEA) collects data on pollutants and includes validated concentration measures from monitoring stations in the majority of cities all over Europe. Our primary measure of pollution is fine particulate matter $PM_{2.5}$. In addition, we replicate the analysis using alternative airborne contaminants (e.g., PM_{10}, NO_2, or O_3) to assess whether our findings hold across different types of pollutants. Furthermore, the air monitoring stations in our dataset are geo-tagged with their location type (urban, suburban, or rural) enabling spatial analysis of pollution effects across different settlement densities[8].

Weather data, combining ground-based measurements and satellite data, are taken from the Climate Data Records (CDR), and are systematically collected and maintained by the National Centers for Environmental Information.[9]. Specifically, we consider total precipitation, temperature, and wind speed at a 10-meter elevation.

Pollution levels are provided as pre-aggregated annual concentrations, while weather data

[7]The 49 greater cities correspond to 160 cities and represent a city with a larger area or a combination of several cities.

[8]Urban areas are defined as continuously built-up regions where streets are predominantly lined with buildings, each having at least two floors, or large detached buildings with similar characteristics. These areas are mostly free of non-urbanised spaces, except for city parks. In contrast, suburban areas consist mainly of detached buildings, with a lower building density than urban areas. It's important to note that, in this context, suburban areas can exist independently of an urban centre, which differs from the common definition of a suburb as an outlying part of a city or town.

[9]For more details on the data, refer to https://www.ncei.noaa.gov/products/climate-data-records.

consist of annual precipitation averages, computed by aggregating daily records from monitoring stations, both georeferenced by latitude and longitude.

Additional data. We supplement our analysis with regional socioeconomic indicators from Eurostat[10]: GDP per capita (NUTS3, PPS-adjusted to EU27 averages), unemployment rates, and poverty risk (NUTS2). These variables account for disparities in pollution exposure and health vulnerability. Lower-SES populations face elevated risks due to both greater pollutant exposure and increased susceptibility from behavioral factors (e.g., smoking, limited health knowledge, e.g., DiNovi (2010)) and reduced capacity to mitigate environmental harms (e.g., Sexton (1997)).[11] Controlling for these factors isolates pollution effects while addressing structural inequities.

2.2 Samples

The full dataset combines data collected at two distinct levels: (i) city-level data and (ii) the monitoring station-level data. The city-level data contains information on premature mortality outcomes and economic indicators at a yearly frequency. The monitoring station-level data provides information about both air quality and weather conditions. However, monitoring stations may be located in different areas of the city, potentially failing to represent the same territorial cluster and leading to weather measurements that are not fully representative of $PM_{2.5}$ levels. Additionally, some stations may be located far from urban centres, making $PM_{2.5}$ measurements less representative of the city's premature mortality indicators. To address these issues we take the following steps: (i) we identify the weather monitoring station closest to each air quality monitoring station, en-

[10] According to Eurostat, the EU has developed a classification known as NUTS (Nomenclature of territorial units for statistics), which divides each EU country into 3 levels: NUTS 1 (major socio-economic regions), NUTS 2 (basic regions for regional policies), NUTS 3 (small regions for specific diagnoses). Taking Italy as an example, NUTS3 corresponds to provinces, while NUTS2 aligns with regions. See European Commission (2017).

[11] Wang et al. (2022) find higher $PM_{2.5}/NO_2$ exposure among high-SES individuals in China; however, this likely reflects urban density effects rather than reduced low-SES vulnerability.

suring a maximum (average) Euclidean distance of 12.5 km (5 km); (ii) we select weather and air quality monitoring station pairs located within a maximum (average) radius of 12.5 km (5 km) from the city centroid. This methodology enhances the spatial representativeness of our dataset concerning both air pollution and meteorological conditions. Notice that the selected maximum distance of 12.5 km is associated with a smaller average distance between station pairs and between station pairs and cities.

Our final dataset collects relevant information relative to 75 cities in Southern European countries (Croatia, Greece, Italy, Portugal, Spain) paired with pollution and weather conditions measured by 180 selected monitoring stations.[12] After incorporating additional covariates, we observe minimal sample attrition. Our regression samples consist of 429 city-year and 915 station-year observations for *premature mortality* and 482 city-year and 1,030 station-year observations for *infant mortality*.[13]

Table A1 shows descriptive statistics. Notably, the number of observations collected at the monitoring station level is higher than the number of observations at the city level, as a single city may have multiple pairs of monitoring stations for pollution and meteorological conditions. The sample consists of 57% of Spanish cities, 35% of Italian cities, 5.6% of Portuguese cities, 1.7% of Greek cities, and 0.7% of Croatian cities. It comprises 79% of stations classified as urban, 20% as suburban, and the remaining 1% as rural, based on their territorial location[14]. Considering our outcome measures, infant mortality is, on average, 2.97 every 1,000 live births. The under-65 mortality rate due to circulatory or respiratory diseases is equal to 3.4 every 100,000 individuals. Regarding meteorological

[12]The spatial distribution, average values, and variability of the two outcomes are presented in Figures A1 and A2 in the Appendix. Those of $PM_{2.5}$ levels and mean precipitation are respectively presented in Figures A3 and A4.

[13]The dataset is an unbalanced panel due to gaps in city-level mortality reporting and intermittent station operation. Thus, the observed city-years (482 for infant mortality) and station-years (1,030) are below the theoretical maximum over the years 2010-2018 (675 and 1,620, respectively).

[14]For a visual representation of both urban and suburban monitoring stations, refer to the figure A5 provided in the Appendix.

conditions, the average precipitation level is 0.05 mm/day, the average wind speed is 6.4 m/s, and the mean temperature is 15.5°C. Annual average air pollution levels in the sample reveal an average $PM_{2.5}$ concentration of 13.3 $\mu g/m^3$, PM_{10} at 24.5 $\mu g/m^3$, NO_2 at 27.2 $\mu g/m^3$, SO_2 at 4.5 $\mu g/m^3$, CO at 4.4 $\mu g/m^3$, and O_3 at 52.19 $\mu g/m^3$. For comparison, the 2021 WHO Air Quality Guidelines recommend the following limits: an annual average of 5 $\mu g/m^3$ for $PM_{2.5}$, 15 $\mu g/m^3$ for PM_{10}, and 10 $\mu g/m^3$ for NO_2; a 24-hour limit of 40 $\mu g/m^3$ for SO_2, a 24-hour limit of 4 $\mu g/m^3$ for CO and a maximum of 60 $\mu g/m^3$ for O_3. These statistics indicate that, on average, the levels of $PM_{2.5}$, PM_{10}, and NO_2 exceed the WHO-recommended thresholds, suggesting potential adverse health effects associated with air pollution exposure in the sampled cities. In contrast, the average concentrations of SO_2, CO, and O_3 remain within or close to the recommended limits.

3 Empirical strategy

We aim to estimate the causal effect of $PM_{2.5}$ on premature mortality in Southern European cities. We start by considering a standard OLS regression:

$$Y_{ct} = \alpha + \beta PM_{2.5ct} + \sum_{k=1}^{N_K} \iota_k X_{kct} + \sum_{j=1}^{N_J} \gamma_j X_{jrt} + \theta_c + \psi trend_t + \xi trend_t^2 + \epsilon_{ct}, \quad (1)$$

where Y_{ct} describes the outcomes of interest: (i) the under 65 mortality rate due to diseases of the circulatory or respiratory systems per 100,000 individuals recorded in city c and year t and (ii) the infant mortality rate per 1,000 live births recorded in city c and year t. Air pollution is measured using the level of $PM_{2.5}$ in micrograms per cubic meter ($\mu g/m^3$) in city c (i.e., the level measured by the air monitoring stations within a maximum (average) radius of 12.5 km (5 km) from the city centroid of city c) and year

t. X_{ct} represents a matrix of covariates at the city-year level. X_{rt} represents a matrix of covariates at the region-year level. θ_c are city fixed effects and $trend_t^i$, with $i = 1, 2$ is a quadratic time trend. ϵ_{ct} indicates the error term.

However, in this context, the coefficient of interest, β, which quantifies the effect of air pollution on health, can be biased if pollution exposure is non-randomly assigned across populations. For instance, a possible concern is given by the non-random spatial sorting of individuals and economic activities that could co-vary with both pollution levels and health determinants. As pointed out by, e.g., Neidell (2004) and Deryugina et al. (2019), individuals may engage in avoidance behaviors not only in response to smog alerts but also due to persistent exposure in highly polluted areas. These adaptive behavioral responses induce attenuation bias in conventional OLS estimates, resulting in a systematic downward bias in the estimated mortality effects of air pollution exposure. A possible solution is to adopt an IV strategy that leverages random variations in weather conditions as an instrument for air pollution. In this paper, following a large literature (e.g., Fontenla et al. (2019), Palma et al. (2022) - just to cite a few), we use yearly precipitations recorded in city c during year t as an instrumental variable. A valid instrument should satisfy two conditions: (i) relevance: rainfall should correlate with air pollution; (ii) exclusion restriction: rainfall should only affect mortality through its impact on pollution. As for relevance, rainfall facilitates the transportation of suspended particles from the atmosphere to the ground, which occurs via dry and wet deposition. As a raindrop falls through the atmosphere, it can attract tiny aerosol particles to its surface before hitting the ground[15]. Concerning the exclusion restriction, it must be noted that our identification strategy, by including city-specific fixed effects, relies on within-city yearly

[15]The process by which droplets and aerosols attract particles is called *coagulation*, a natural phenomenon that can clear the air of particle pollutants such as $PM_{2.5}$ (Ardon-Dryer et al. 2015). The effect of rainfall on pollution is broadly referred to as the *washing effect* (Guo et al. 2016) or *removal effect* (Zhao et al. 2020).

variations in precipitation levels, which we assume to be randomly distributed across cities and years.

A possible threat to identification in our model is represented by healthy individuals deciding to relocate to cities with greater yearly variations in precipitation levels. However, this behavior seems implausible for at least two reasons: (i) according to the literature, migration decisions across cities or countries are mainly driven by income or labour market opportunities (e.g., Borjas (1999)); (ii) even admitting a possible role for weather conditions, for the large majority of individuals it would not be fully rational to decide to move towards cities with more rain (and, thus, worse weather conditions). However, this mechanism may also arise spuriously if rain is positively correlated with income or if healthy individuals decide to move away from cities with high pollution levels and smaller yearly variations in precipitation levels. To assess this potential threat to identification, we test whether rainfall is correlated with income and examine patterns of residential relocation, as detailed in the robustness section 4.2.

If the above-mentioned conditions hold, we can estimate an IV model using the standard Two-Stage Least Squares (2SLS) estimator. The first stage regression is represented by the following equation:

$$PM_{2.5ct} = \alpha^1 + \lambda^1 Rain_{ct} + \sum_{k=1}^{N_K} \iota_k^1 X_{kct} + \sum_{j=1}^{N_J} \gamma_j^1 X_{jrt} + \theta_c^1 + \psi^1 trend_t + \xi^1 trend_t^2 + \epsilon_{ct}^1 \quad (2)$$

where $Rain_{ct}$ represents annual mean daily precipitation recorded in city c during year t and all the other covariates remain as previously defined and discussed in Equation 1.

The second stage regression is represented by the following equation:

$$Y_{ct} = \alpha^2 + \beta^2_{2SLS} PM_{2.5ct} + \sum_{k=1}^{N_K} \iota^2_k X_{kct} + \sum_{j=1}^{N_J} \gamma^2_j X_{jrt} + \theta^2_c + \psi^2 trend_t + \xi^2 trend^2_t + \epsilon^2_{ct}, \quad (3)$$

Now, β^2_{2SLS} captures the causal effect of $PM_{2.5}$ on mortality.

It must be noted that both the first and second-stage equations report variables measured at the city-year level. However, both pollution and other information about meteorological conditions, including our instrument based on precipitation, are recorded by monitoring stations located in different parts of the city. Ideally, one would like to exploit the station-year variability when estimating the first stage and the city-year variability for the second stage. This has two main advantages: (i) first-stage estimates rely on a larger sample size and, consequently, are more precise; (ii) not aggregating pollution and weather data at the city level, we avoid the risk of incurring in measurement errors due to estimating the correlation between precipitation and pollution measured by monitoring stations located in very distant areas, but close to the same city, or to relying on opposite measurements by different stations located close to the same city, that after aggregation could potentially lead to null variations. Nevertheless, in the latter scenario, we may observe an increase in pollution-related mortality at the city level, stemming from an area with lower variations in annual precipitation - and consequently higher pollution levels - if that area is more densely populated by individuals facing elevated health risks.

To avoid these problems, we use here the Two-Sample Two-Stage Least Squares estimator (TS2SLS) proposed by Angrist & Krueger (1992) according to which consistent instrumental variables estimation is possible when the outcome and the instrument (but not the endogenous variable) are observed in one sample and the endogenous and the instrument

(but not the outcome) are observed in a second different sample. In our case, we can observe the instrument, the endogenous and the outcome in the sample at the city level. Still, we can observe only the instrument and the endogenous in the station-level sample. In the case of exact identification, the conventional TS2SLS estimator is:

$$\hat{\beta}^2_{TS2SLS} = \left(\widehat{PM_{2.5_c}}' \widehat{PM_{2.5c}}\right)^{-1} \widehat{PM_{2.5_c}}' Y_c \tag{4}$$

where $\widehat{PM_{2.5c}} = Rain_c \left(Rain_s' Rain_s\right)^{-1} Rain_s' PM2.5_s$. As demonstrated by Inoue & Solon (2010), the TS2SLS estimator is preferred (i.e., more asymptotically efficient and robust when the two samples differ in their stratification schemes) to the two-sample instrumental variables (TSIV) version.

Unlike Equation 2 (estimated at the city-level c), the first stage equation is now estimated at the station-level s:

$$PM_{2.5st} = \alpha^1 + \gamma^1 Rain_{st} + \sum_{k=1}^{N_K} \iota_k^1 X_{kst} + \sum_{j=1}^{N_J} \gamma_j^1 X_{jrt} + \theta_s^1 + \psi^1 trend_t + \xi^1 trend_t^2 + \epsilon_{st}^1 \tag{5}$$

while the second stage equation remains defined as in Equation 3.

Lastly, to validate the consistency of our estimates, we consider four different specifications of the TS2SLS version of the model presented in Equations 3 and 5: specification (1), which includes in the first (second) stage equation station- (city-) specific fixed effects plus a quadratic time trend, in order to account for time invariant unobservable characteristics at the station (city) level, and non linear long-term mortality trends; specification (2), which introduces city-specific linear trends, in place of the basic linear trend included in specification 1, and maintains the common quadratic trend component, in order to account

for heterogeneous temporal dynamics across cities; specification (3), which adds, relative to specification (1), station-level covariates (i.e., the share of days with valid measurements along the year, the average wind speed and temperature measured by station s in city c during year t) and two spatial economic covariates (Gross domestic product GDP at current market prices measured at $p-NUTS3$, and unemployment rates measured at $r-NUTS2$, during year t); specification (4), which refines specification (3) by including city-specific linear trend while excluding the common linear time trend, allowing to account for flexible city-level temporal dynamics in mortality rates and pollution. Standard errors are clustered at the station (city) level in all the first and second stage equations.

4 Results

4.1 Baseline results

Table 1 presents our baseline estimates of the effect of $PM_{2.5}$ on the under 65 mortality rate, specifically for circulatory and respiratory diseases measured per 100,000 individuals in city c at time t, for all the four specifications described in Section 3. Panel A shows the OLS estimates, while Panel B displays the TS2SLS estimates.[16] OLS estimates (Panel A) show positive but not always statistically significant effects. TS2SLS estimates (Panel B) are always positive and highly statistically significant across all specifications. Coefficients range from 0.2660 (column 4) to 0.3346 (column 2). Our preferred specification is shown in column (3), which includes a linear time trend instead of a city-specific linear trend to prevent excessive absorption of $PM_{2.5}$ variation, thereby preserving the identification of its effect on premature mortality.[17] According to our estimates, an increase

[16] 2SLS estimates at the city level, including the first stage (equation 2) and the second stage (equation 3), for both under 65 mortality rate and infant mortality rate are reported in the Online Supplementary Material.

[17] While a city-specific trend, model (4), would account for unobserved local factors, it could also reduce the valuable variation in $PM_{2.5}$, compromising the precision of the estimates. Since the model already incorporates station fixed effects,

of one unit of $\mu g/m^3$ in $PM_{2.5}$ (corresponding to 7.5% of the average level) is associated with 0.2887 additional deaths per 100,000 inhabitants due to circulatory and respiratory diseases among individuals under 65. This corresponds to an 8.49% increase relative to the average mortality rate and implies an elasticity of approximately 1.13, indicating that a 1% increase in $PM_{2.5}$ exposure is associated with a 1.13% rise in mortality among the under-65 population.[18] The elasticity exceeding unity suggests that mortality rates are disproportionately sensitive to marginal changes in $PM_{2.5}$, underscoring the acute public health implications of its exposure. Notably, this estimate is 20 times larger than the OLS estimate of 0.0139, suggesting a substantial downward bias in the latter.

Table 2 shows estimates for the effect of $PM_{2.5}$ on the infant mortality rate per 1,000 live births. OLS estimates, also in this case, show coefficients that are not statistically significant (Panel A). The TS2SLS estimates (Panel B) yield positive coefficients, which are statistically significant at the 5% and 10% levels only in the first three specifications. In column (4), we do not find a statistically significant effect of $PM_{2.5}$ on the infant mortality rate per 1,000 live births. The magnitude of the coefficient associated with our preferred specification is of considerable size: in this case, a one $\mu g/m^3$ increase in $PM_{2.5}$ (corresponding to 7.5% of the average) implies an increase in the infant mortality rate of 0.3134 points, corresponding to 10.57% of the average infant mortality rate per 1,000 live births. The implied elasticity of 1.41 suggests that infant mortality is particularly responsive to $PM_{2.5}$ exposure, approximately 25% more elastic than the corresponding estimate for mortality among those under 65 (1.13). This gradient in exposure sensitivity by age group has important implications for the evaluation of mortality risk reductions

quadratic time trends, meteorological variables, monitoring station characteristics, and economic factors, the linear time trend provides an optimal balance, capturing common temporal trends without over-controlling for local differences already accounted for.

[18]The elasticity of 1.13 is computed as the ratio of the percentage change in mortality to the percentage change in pollution: (8.49%/7.5%).

in environmental policy.

Table 1: The impact of $PM_{2.5}$ on cardio-respiratory mortality rates under 65

	(1)	(2)	(3)	(4)
Panel A: OLS				
$PM_{2.5}$	0.0129	0.0394*	0.0139	0.0337
	(0.0233)	(0.0227)	(0.0241)	(0.0230)
Panel B: TS2SLS				
$PM_{2.5}$	0.3171***	0.3346**	0.2887***	0.2660**
	(0.1065)	(0.1331)	(0.1060)	(0.1149)
Mean of Y	3.4012	3.4012	3.4012	3.4012
SD of Y	1.1711	1.1711	1.1711	1.1711
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	180	180	180	180
Observations cities	429	429	429	429
Observations stations	915	915	915	915
Station FE	Yes	Yes	Yes	Yes
Linear trend	Yes	No	Yes	No
Quadratic trend	Yes	Yes	Yes	Yes
Station control variable	No	No	Yes	Yes
Weather control variables	No	No	Yes	Yes
City linear trend	No	Yes	No	Yes
GDP and Unemployment	No	No	Yes	Yes

Notes: The table presents baseline estimates of the effect of $PM_{2.5}$ on the mortality rate from circulatory and respiratory diseases (per 100,000 individuals aged <65 years) across four specifications (columns (1) - (4)). Panel A reports OLS estimates, while Panel B reports TS2SLS estimates. Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table 2: The impact of$PM_{2.5}$ on infant mortality rates

	(1)	(2)	(3)	(4)
Panel A: OLS				
$PM_{2.5}$	-0.0193	0.0392	-0.0205	0.02332
	(0.0415)	(0.0481)	(0.0429)	(0.0492)
Panel B: TS2SLS				
$PM_{2.5}$	0.3787**	0.4038*	0.3134*	0.2985
	(0.1803)	(0.2224)	(0.1795)	(0.1951)
Mean of Y	2.9665	2.9665	2.9665	2.9665
SD of Y	1.6519	1.6519	1.6519	1.6519
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	177	177	177	177
Observations cities	482	482	482	482
Observations stations	1,030	1,030	1,030	1,030
Station FE	Yes	Yes	Yes	Yes
Linear trend	Yes	No	Yes	No
Quadratic trend	Yes	Yes	Yes	Yes
Station control variable	No	No	Yes	Yes
Weather control variables	No	No	Yes	Yes
City linear trend	No	Yes	No	Yes
GDP and Unemployment	No	No	Yes	Yes

Notes: The table presents baseline estimates of the effect of $PM_{2.5}$ on the infant mortality rate per 1,000 live births across four specifications (columns (1) - (4)). Panel A reports OLS estimates, while Panel B reports TS2SLS estimates. Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

First stage estimates (Appendix Tables A2 and A3) show that rainfall is a strong predictor of $PM_{2.5}$ levels for both outcomes. The estimated coefficients are similar since the information at the station level differs for only 3 stations more in the infant mortality rate estimates. This is confirmed by the first-stage F-statistics, which vary from 29.6 (column 1) to 62.3 (column 2) and are well above 10 in every specification considered. According to our preferred estimates in column (3), a one unit of rain/day (i.e., 100 mm/day) increase in average rainfall is associated with a 0.1737-unit reduction in $PM_{2.5}$, suggesting

atmospheric washing-out effects. Table A4 shows reduced form estimates for the under-65 mortality rate (panel A) and the infant mortality rate (panel B). These estimates consistently support the view that rain significantly affects our outcome variables plausibly through the effect generated on $PM_{2.5}$.

4.2 Additional analyses

To assess the credibility of our identification strategy, we first address potential threats to the validity of the instrument, ensuring the exogeneity and the relevance of rainfall as an instrument for $PM_{2.5}$. Second, using specification (3), we conduct a set of robustness analyses aimed at testing the sensitivity of our main results to alternative pollutant exposures and instrumental variable configurations.

4.2.1 Instrument validity

First, we test whether rainfall is correlated with income, a potential confounder in the relationship between pollution and mortality. As shown in Table A5 Column (1), we find no significant correlation between GDP and rainfall, suggesting that rainfall does not operate through income to affect mortality. Furthermore, as shown in Column (2), current rainfall is not significantly predicted by precipitation levels in the two preceding years. This supports the assumption that rainfall is not systematically influenced by persistent local factors, thereby strengthening the evidence for the instrument's exogeneity.

Second, we examine whether healthier individuals are more likely to relocate from cities with higher pollution levels and lower variability in annual precipitation. We estimate an OLS regression model that includes city-specific time trends, as well as city and year fixed effects. Table A6 shows no significant correlation between migration decisions (im-

migration, emigration and their difference - standardised as rates per 1,000 municipal inhabitants) and current and lagged $PM_{2.5}$ levels, nor with current and lagged weather conditions (precipitation, wind speed, temperature), nor with current and lagged GDP. This evidence supports our exclusion restriction and the causal interpretation of our estimates on the impact of $PM_{2.5}$ on premature mortality.

4.2.2 Alternative exposures and instrumental strategies

While our primary focus remains on $PM_{2.5}$, we assess the robustness of our findings by extending the analysis to other pollutants - PM_{10}, NO_2, SO_2, CO, and O_3 - whose concentrations typically exhibit strong spatial and temporal correlations in urban environments[19]. According to previous studies, PM_{10}, which counts coarse (bigger) particles, and other gas pollutants such as NO_2, SO_2, O_3, and CO, particularly widespread in urban areas, are important contributors to the incidence of pediatric asthma globally (see Pandey et al. (2005), Giaccherini et al. (2021), Orellano et al. (2021), Anenberg et al. (2022)) and represent serious health risks, especially in terms of infant mortality (Currie & Neidell (2005)). This extension serves two key purposes. First, it allows us to test the sensitivity of our results and determine which pollutants have independent associations with health outcomes, a critical insight for targeted environmental regulation. Second, it helps clarify whether observed health effects stem specifically from $PM_{2.5}$ or result from exposure to a broader mix of pollutants. Additionally, the extended analysis accomplishes two further objectives: (1) it tests whether the observed scavenging effect of rainfall is consistent across pollutants with differing chemical compositions and atmospheric life-

[19] NO_2 exposure has been linked to approximately 13% of the global paediatric asthma burden, and up to 50% in the most populated 250 cities worldwide (e.g., Achakulwisut et al. (2019)). O_3 is a secondary pollutant formed when volatile organic compounds (VOCs) and nitrogen oxides react in sunlight. CO is a direct byproduct of combustion, primarily released by vehicles, industrial processes, and household appliances such as gas stoves and heaters. Major sources of air pollutants include fossil fuel combustion from power plants and industry, metal extraction, volcanic activity, and high-sulfur fuel use in transport and heavy machinery.

times, and (2) it helps identify potential heterogeneity in precipitation-driven pollution dynamics.

We report results using these additional pollutants in Table 3. We find significant causal effects of PM_{10} and NO_2 on mortality. While we do not find evidence of any significant effect when considering the other pollutants. Results in Panel A show that a one-unit increase in PM_{10} (corresponding to 4.1% of the average level) implies an increase in the mortality rate under 65 due to diseases of the circulatory or respiratory systems per 100,000 individuals of 0.1659 points (a 4.88% increase of the average mortality rate). In comparison, a one-unit increase in NO_2 (corresponding to 3.7% of the average level) implies an increase in mortality under 65 of 0.1525 points (equal to a 4.48% increase in the mortality rate). This corresponds to elasticities of 1.19 for PM_{10} and 1.21 for NO_2, both slightly exceeding the $PM_{2.5}$ elasticity of 1.13 for under-65 mortality, suggesting these pollutants may impose comparable marginal health burdens despite their lower average concentrations.

In Table 3, Panel B, we show the effect of air pollution on the infant mortality rate per 1,000 live births. Coefficients are positive and marginally statistically significant. A one-unit increase in PM_{10} (NO_2) is associated with a 6.04% (5.58%) increase in the average infant mortality rate per 1,000 live births. These effects imply elasticities of 1.47 for PM_{10} and 1.51 for NO_2, substantially (around 20%) higher than the corresponding elasticities for under-65 mortality (1.19 and 1.21, respectively). This gradient reinforces the pattern of greater infant vulnerability to air pollution observed in our primary $PM_{2.5}$ results. First-stage estimates, reported in the Online Supplementary Material, confirm that rainfall is a strong predictor of air pollution levels, but only for PM_{10} and NO_2. In Table 3, the F-statistic is always above 10 for these two pollutants.

Lastly, we extend our IV strategy to include, alongside precipitations, wind speed (used, among others, by Deryugina et al. (2019), Di Porto et al. (2021)) (2IV) and average temperature (implemented, among others, by Sager (2019), Filomena & Picchio (2024)) (3IV) as instrumental variables. This approach leverages exogenous weather-driven variation in $PM_{2.5}$ through distinct pathways: (1) precipitation's aerosol scavenging - as discussed, (2) wind speed's dual role in dispersion/resuspension, and (3) temperature's effects on emissions (e.g., heating demand) and atmospheric chemistry. Together, these instruments better isolate the $PM_{2.5}$ causal effect while addressing unobserved confounding. To validate our multi-instrument IV strategy, we implement the Sargan test of overidentifying restrictions.

Table A7 reports results for both under-65 and infant mortality outcomes. The estimates show cardio-respiratory mortality effects for individuals under 65 but not for infant mortality, consistent with the earlier marginally significant estimates. In more detail, in Panel A, we observe a statistically significant effect of air pollution on cardio-respiratory mortality among individuals under 65, while no effect is detected for infant mortality, both in the 2IV and in the 3IV specifications. The estimated effect ranges from 8.2% (2IV) to 8.7% (3IV) increases in the average mortality rate - corresponding to elasticities of 1.09 and 1.16, respectively - closely aligning with the results obtained using the baseline specification with one instrumental variable and other weather conditions as control variables. Panel B reports first-stage estimates, supporting the view that rainfalls and wind can reduce the concentration of pollutants, while no effects are detected for temperature. The Sargan test - reported in the bottom part of the Table - fails to reject the null hypothesis of valid overidentifying restrictions, confirming instruments' validity.

Table 3: The impact of other pollutants on mortality rates

	PM_{10}	NO_2	SO_2	CO	O_3
	(1)	(2)	(3)	(4)	(5)
Panel A: < 65 Cardio-Respiratory MR × 100,000					
Pollutant	0.1659**	0.1525***	-1.2305	-2.0942	-0.4929
	(0.0666)	(0.0574)	(0.9042)	(35.5495)	(0.3568)
Mean of Y	3.4012	3.4012	3.4012	3.4012	3.4012
SD of Y	1.1711	1.1711	1.1711	1.1711	1.1711
F-stat	15.3	11.6	1.2662	3.9064	0.5407
p-value F stat	1.05e-4	7.286e-4	0.2613	0.0488	0.4626
Number of cities	75	75	75	75	75
Number of pollution stations	180	180	166	180	178
Observations cities	429	429	429	429	429
Observations stations	914	915	818	913	890
Panel B: Infant MR × 1,000					
Pollutant	0.1791*	0.1655*	-1.2172	-2.2733	-0.4712
	(0.1074)	(0.0958)	(1.1138)	(38.6009)	(0.4256)
Mean of Y	2.9664	2.9664	2.9664	2.9664	2.9664
SD of Y	1.6519	1.6519	1.6519	1.6519	1.6519
F-stat	15.3	11.6	1.2662	3.9064	0.5407
p-value F stat	1.05e-4	7.286e-4	0.2613	0.0488	0.4626
Number of cities	75	75	75	75	75
Number of pollution stations	177	177	130	177	175
Observations cities	482	482	482	482	482
Observations stations	1,029	1,030	935	1,028	1,004

Notes: The table presents TS2SLS estimates of the effect of PM_{10} (1), NO_2 (2), SO_2 (3), CO (4) and O_3 (5) on the mortality rate from circulatory and respiratory diseases (per 100,000 individuals aged <65 years) (Panel A) and on the infant mortality rate per 1,000 live births (Panel B). The specification used is that of tables 1 and 2, column (3). Notice that the number of observations varies depending on the kind of pollutant explored. This is because not all the air-monitoring stations have registered pollutants continuously over the considered period. Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

4.3 Heterogeneity

This section investigates whether our baseline results are heterogeneous across two dimensions. First, we test whether the effect of $PM_{2.5}$ differs between urban and suburban areas. Second, we test whether the effect of pollution differs between the rich and the poor areas, classifying cities located in regions above or below the median of the distribution of the rate of individuals at risk of poverty within a given country.

Table 4 presents estimates of the heterogeneous effects of pollution on mortality across urban and suburban areas (columns 1 and 2, respectively), exploiting information about monitoring stations' location. In Panel A, the effect of $PM_{2.5}$ on the mortality rate under 65 due to diseases of the circulatory or respiratory systems per 100,000 individuals is significant only when considering monitoring stations located in urban, rather than suburban areas. This result likely suggests heightened vulnerability or exposure intensity in urban parts of the cities subject to higher concentrations of traffic-related pollutants or to the effect of heating systems.

In columns (3) and (4), we split our sample according to whether cities are located in regions where the proportion of individuals at risk of poverty is above or below the national median. This approach enables us to assess the effects of $PM_{2.5}$ exposure relative to economic conditions, offering a comparative perspective within the broader socioeconomic context. The estimates suggest that $PM_{2.5}$ has a significant effect on the mortality rate under 65 in cities located in richer regions. Individuals living in these regions have a lower risk of poverty at the cost of being exposed to higher levels of air pollution.

Looking at Panel B, which considers infant mortality rate per 1,000 live births, we find no statistically significant effects either splitting our sample of cities according to urban/suburban areas (columns 1 and 2), or according to the rate of individuals at risk of

poverty (columns 3 and 4).

Table 4: The heterogeneous effects of $PM_{2.5}$

	Metropolitan areas		**Risk of Poverty rate**	
	Urban	Suburban	Above	Below
	(1)	(2)	(3)	(4)
Panel A: < 65 Cardio-Respiratory MR × 100,000				
$PM_{2.5}$	0.3112***	0.2456	0.1249	0.2352***
	(0.1124)	(0.2133)	(0.9397)	(0.0736)
Mean of Y	3.4674	3.2797	3.7119	3.1373
SD of Y	1.1649	1.0134	1.1233	1.1481
F-stat	30.8	2.7732	6.1475	24.3
p-value F-stat	5.788e-8	0.0981	0.0141	1.662e-6
Number of cities	67	27	46	44
Number of pollution stations	102	38	98	96
Observations cities	359	148	197	232
Observations stations	714	195	433	459
Panel B: Infant MR × 1,000				
$PM_{2.5}$	0.2473	0.3165	1.5334	0.2047
	(0.1843)	(0.3724)	(2.6447)	(0.1319)
Mean of Y	2.9739	2.7638	3.1643	2.7719
SD of Y	1.6209	1.5632	1.6464	1.6373
F-stat	30.8	2.7732	6.1475	24.3
p-value F-stat	5.788e-8	0.0981	0.0141	1.662e-6
Number of cities	67	27	47	44
Number of pollution stations	138	38	99	93
Observations cities	404	169	239	243
Observations stations	798	226	512	490

Notes: The table presents TS2SLS estimates of $PM_{2.5}$'s effect on: (Panel A) circulatory/respiratory disease mortality (per 100,000 under-65 population) and (Panel B) infant mortality (per 1,000 live births). Results are stratified by: (1) urban vs. (2) suburban metropolitan areas, and by poverty risk: (3) above vs. (4) below median poverty rate. Urban areas are characterized by continuous built-up development, while suburban areas consist of largely built-up regions on the outskirts of urban centers. Cities are further categorized based on their economic status, with distinctions made between those above and below the poverty line (median). The specification used is that of tables 1 and 2, column (3). Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

5 Conclusions

This paper provides causal estimates of the impact of air pollution due to $PM_{2.5}$ on premature mortality in Southern European cities, focusing on two critical outcomes: (1) the under-65 mortality rate due to circulatory and respiratory diseases, and (2) the infant mortality rate. To address endogeneity concerns in pollution exposure and to account for outcome measures at the city-level and weather and pollution measures at the monitoring station-level, we employ the Two-Sample Two-Stage Least Squares (TS2SLS) estimator using yearly average total millimeters of daily rainfall as our instrument. Rainfall acts an exogenous source of variation in pollution levels, as it influences atmospheric dispersion while being uncorrelated with unobserved determinants of mortality. We concentrate on fine particulate matter ($PM_{2.5}$) as our primary pollution measure due to its well-documented role as a leading environmental risk factor for premature mortality and severe health conditions. Our findings contribute to the growing body of evidence on the adverse health effects of $PM_{2.5}$, particularly in urban settings where pollution concentrations are elevated due to traffic, heating systems, industrial activity, and other anthropogenic sources. We find a positive and statistically significant effect of $PM_{2.5}$ on both mortality indicators. According to our preferred specification, a 1% increase in $PM_{2.5}$ causes a 1.13% rise in the under-65 mortality rate and a 1.41% rise in the infant mortality rate.

Our empirical analysis further quantifies the adverse health effects of additional urban air pollutants beyond $PM_{2.5}$, specifically examining PM_{10} and nitrogen dioxide. The results demonstrate statistically significant increases in both mortality outcomes studied: pollutant exposure corresponds to elasticities of 1.19 (PM_{10}) and 1.21 (NO_2) for under-65 mortality from circulatory and respiratory diseases. More alarmingly, we find substan-

tially greater impacts on infant mortality, with elasticities of 1.47 for PM_{10} (consistent with findings by Palma et al. (2022)) and 1.51 for NO_2 exposure. The particularly strong effects on infant mortality highlight the heightened vulnerability of this group of the population to air quality degradation, warranting special policy consideration for maternal and child health protection in urban environmental planning.

In robustness checks, we expand our IV approach to include multiple weather-based instruments (adding wind and temperature to precipitation). Estimates for mortality under 65 years of age remain consistent with our baseline rainfall-only, while estimates for infant mortality lose significance. The convergence of results across these alternative specifications strengthens the credibility of our causal identification strategy, suggesting that our estimated pollution-mortality relationship is not sensitive to the particular choice of weather instrument. Notably, the robustness of these effects persists most strongly for working-age mortality outcomes, reinforcing our central findings regarding the vulnerability of this demographic group to air pollution exposure.

Our analysis reveals significant spatial and socioeconomic heterogeneity in the health impacts of $PM_{2.5}$ exposure. We find substantially stronger effects on under-65 mortality from circulatory and respiratory diseases in urban areas compared to suburban settings (elasticity of 1.2), likely reflecting higher pollution concentrations and greater population exposure in dense urban environments. Notably, the mortality impacts are particularly pronounced in cities located in regions with lower poverty risk (below the national median), suggesting an unexpected socioeconomic gradient in vulnerability because individuals living in these regions have a lower risk of poverty at the cost of being exposed to higher levels of air pollution. This pattern may stem from several factors: differences in population age structures, varying compositions of pollution sources with differential

toxicity, or distinct exposure patterns related to occupational and leisure activities. These findings highlight the complex interplay between environmental and socioeconomic factors in determining pollution-related health outcomes, emphasizing the need for spatially and demographically targeted policy interventions to mitigate the public health burden of air pollution. The results underscore the importance of considering both geographic context and local socioeconomic conditions when assessing the mortality impacts of $PM_{2.5}$ exposure and designing effective regulatory responses.

Overall, our results suggest that $PM_{2.5}$ is an important determinant of health. Adding to Deryugina et al. (2019), who focus on over-65 mortality rates, we use two measures associated with premature mortality, the mortality rate under 65 and the infant mortality rate. The policy implications of this work point in the direction of implementing further policies aimed at decreasing $PM_{2.5}$ levels across cities, especially in those areas where air pollution is more prevalent.

6 Appendix

Table A1: Summary statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
A.Outcomes					
<65 Cardio-Respiratory MR $\times$ 100,000 pop	429	3.4012	1.1711	0.4521	11.8839
Infant MR $\times$ 1,000 pop	482	2.9665	1.6519	0	9.4118
B.Pollution					
$PM_{2.5}$ $\mu g/m^3$	1,156	13.3422	5.6934	0.5029	41.25
$PM10$ $\mu g/m^3$	1,155	24.5582	7.6912	3	83
NO_2 $\mu g/m^3$	1,156	27.1635	13.8964	3.7593	103.1538
SO_2 $\mu g/m^3$	1,055	4.5293	3.5125	0.0046	25
CO $\mu g/m^3$	1,154	4.3622	37.2224	0.0176	644.7
O_3 $\mu g/m^3$	1,130	52.1915	10.1870	1.1	83.9874
C.Weather conditions					
Avg wind speed m/s	1,156	6.4193	2.9046	1.6677	14.7733
Avg temperature $°C$	1,156	16.5247	2.4535	10.0019	22.6763
Precipitations mm	1,156	0.0529	0.0450	0	0.23337
D.Station characteristics and coverage					
Distance $< 12.5km$	1,156	4.0291	3.2909	0.1827	12.3337
Data Coverage	1,156	80.3119	25.6096	0.01	100
Urban	1,156	0.7872	0.4095	0	1
Suburban	1,156	0.2076	0.4058	0	1
Rural	1,156	0.0052	0.0719	0	1
E.Economic variables					
Unemployment rate $NUTS2$	540	16.8193	7.865	4.9	34.8
GDP $NUTS3$	540	97.1630	25.3325	58	204
At risk of poverty $NUTS2$	540	0.5037	0.5005	0	1
F.Countries					
Croatia	540	0.0074	0.0858	0	1
Greece	540	0.0167	0.1281	0	1
Italy	540	0.35	0.4774	0	1
Portugal	540	0.0556	0.2293	0	1
Spain	540	0.5703	0.4955	0	1

Notes: The table presents summary statistics for key variables, grouped into six categories: (A) health outcomes - Infant Mortality Rate (ratio of infant deaths to live births per 1,000 population) and <65 Cardio-Respiratory Mortality Rate (ratio of deaths due to circulatory and respiratory diseases to the total population per 100,000 population), (B) pollution levels, (C) weather conditions, (D) station characteristics and coverage, (E) economic variables, and (F) country representation.

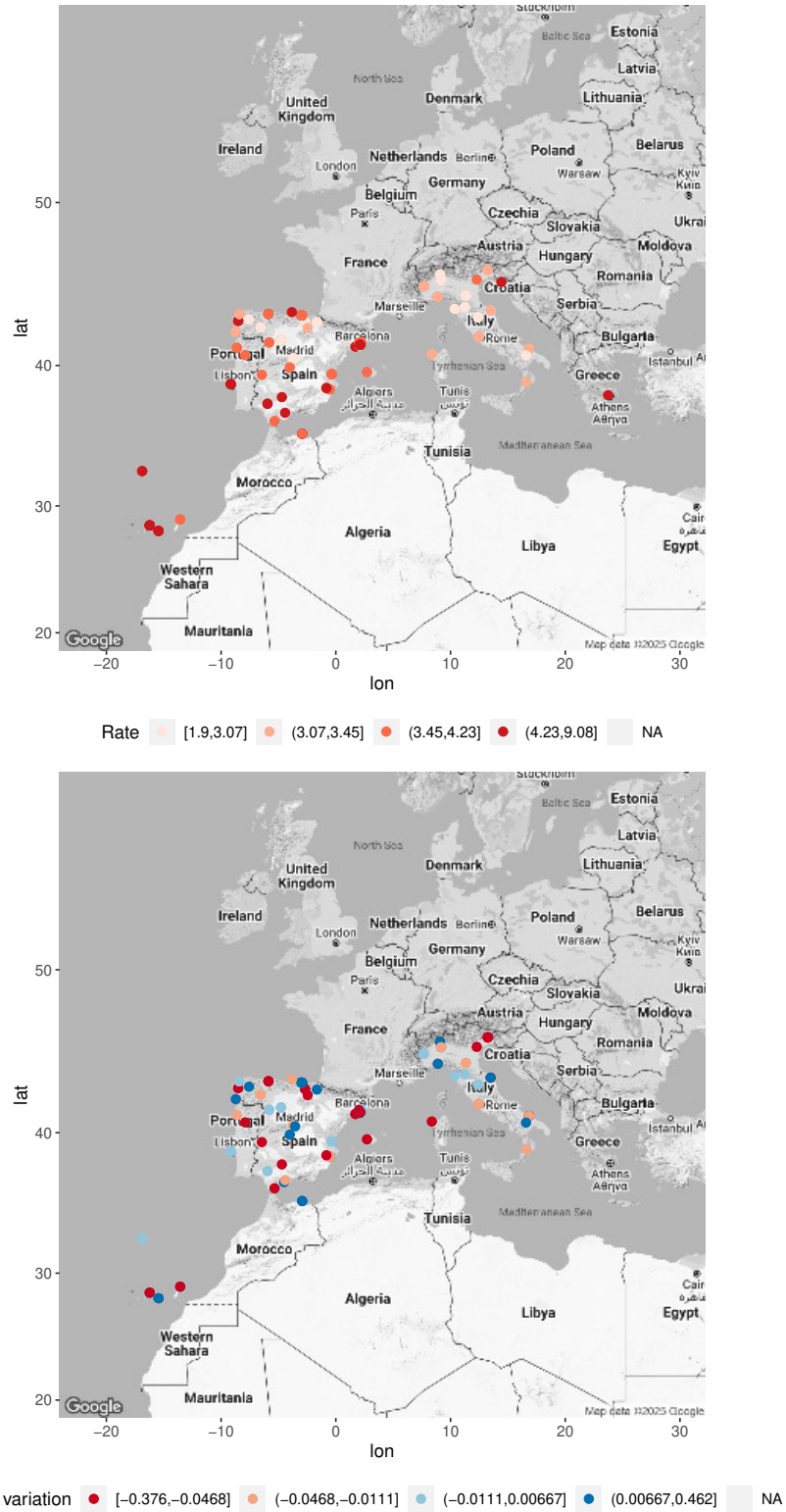

Figure A1: Mortality rate under 65 per 100,000 due to diseases of the circulatory or respiratory systems (upper panel) and average yearly % variation (lower panel)

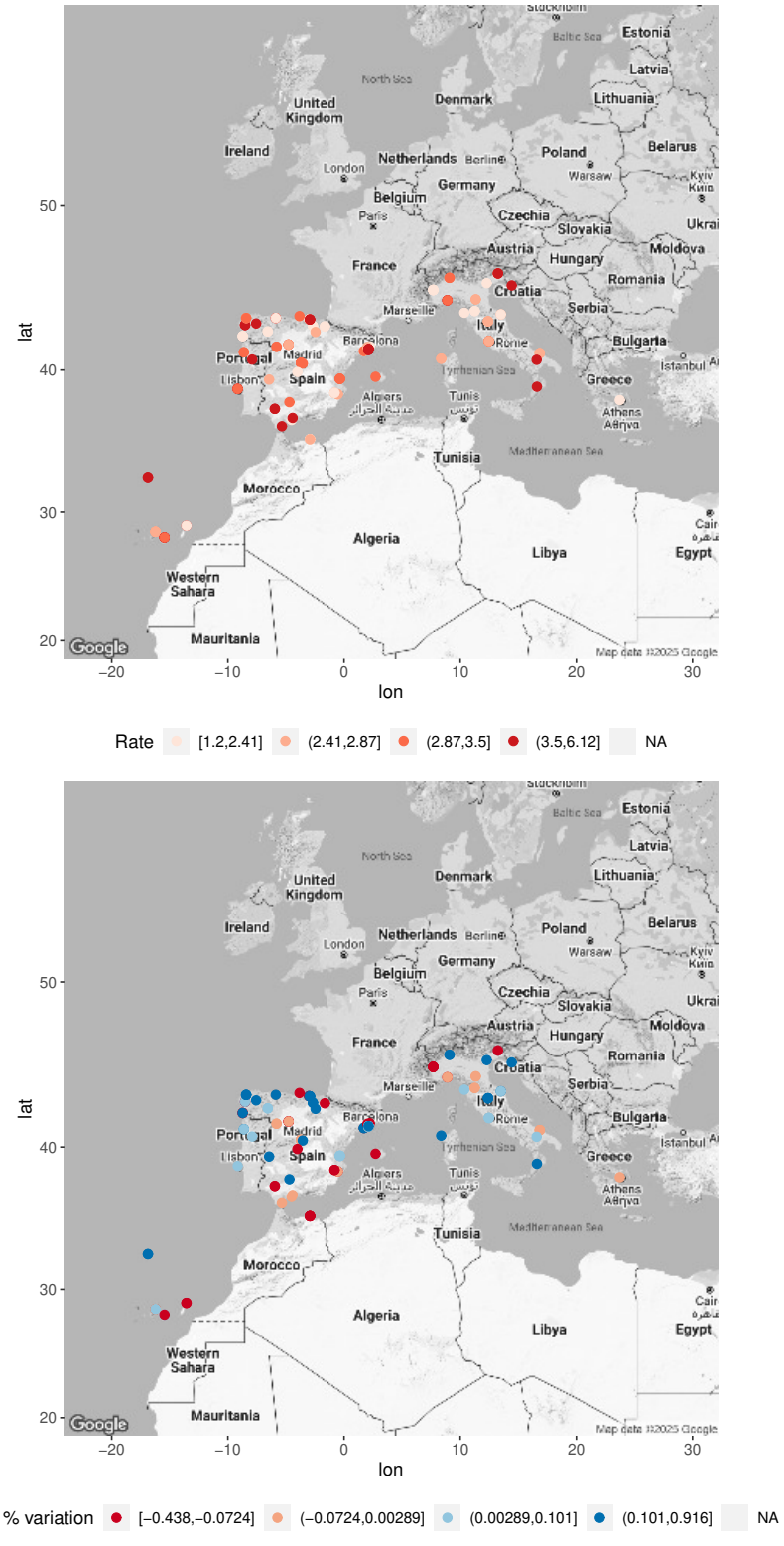

Figure A2: Infant mortality rate per 1,000 live births (upper panel) and average yearly % variation (lower panel)

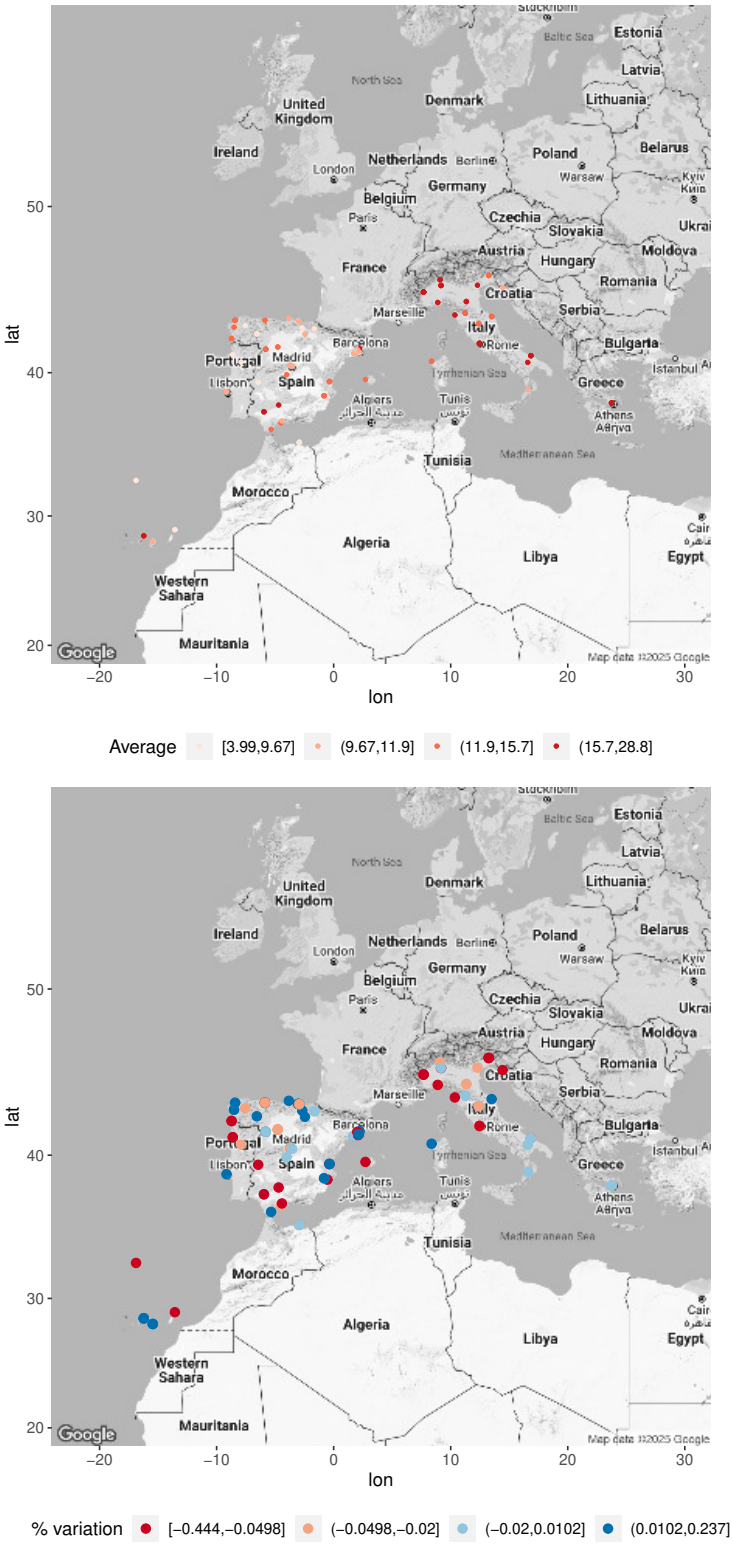

Figure A3: PM 2.5: average (upper panel) and average yearly % variation (lower panel)

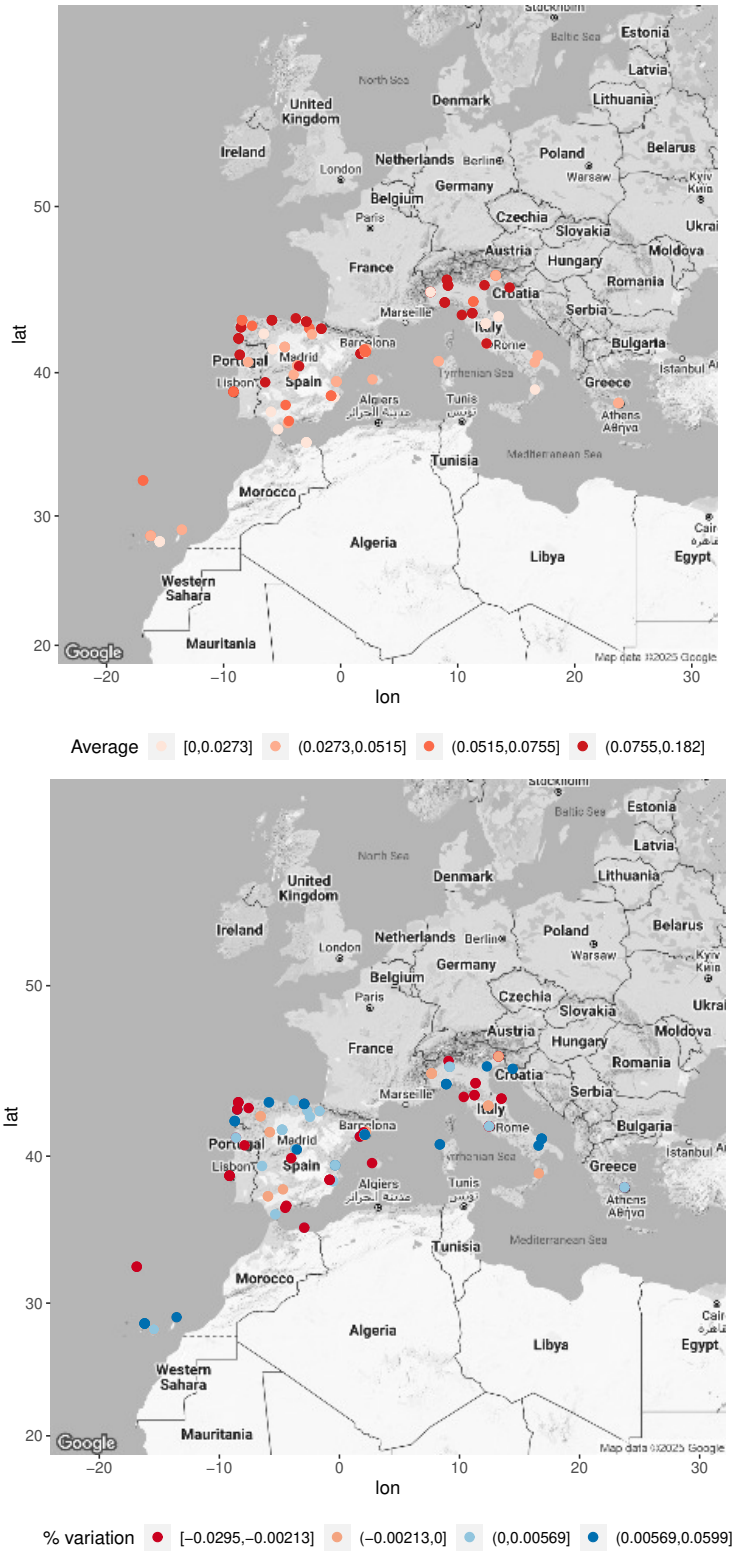

Figure A4: Mean precipitations: average (upper panel) and average yearly % variation (lower panel)

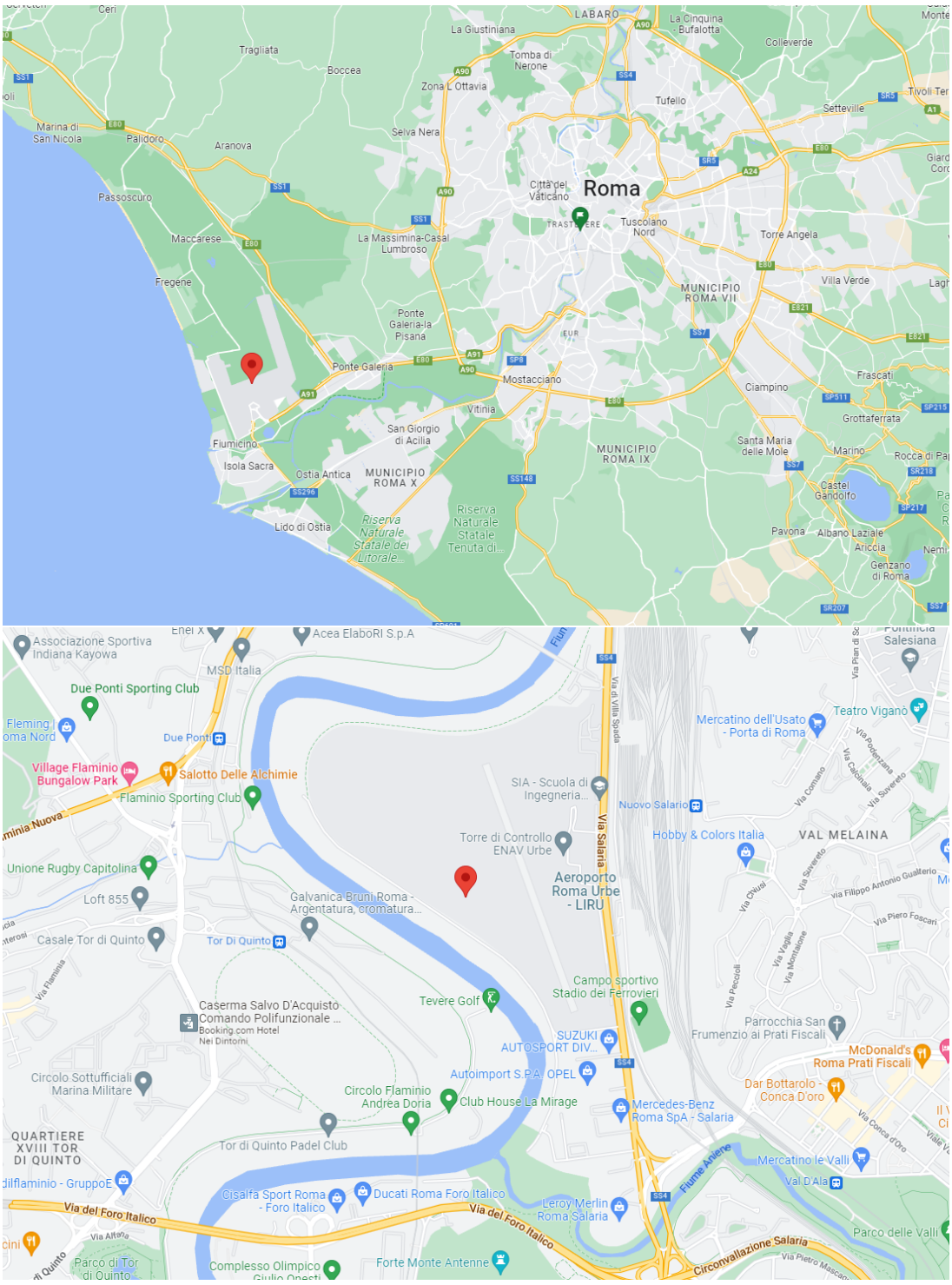

Figure A5: An illustration showing the location of a suburban (upper part) and an urban (lower part) air quality monitoring stations in Rome.

Table A2: First stage estimates for under-65 mortality rate

	(1)	(2)	(3)	(4)
Precipitations	-16.9855***	-15.8956***	-17.3727***	-18.3076***
	(2.5854)	(2.7713)	(2.5703)	(2.7887)
Mean of Y	13.3422	13.3422	13.3422	13.3422
SD of Y	5.6934	5.6934	5.6934	5.6934
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	180	180	180	180
Observations cities	429	429	429	429
Observations stations	915	915	915	915
Station FE	Yes	Yes	Yes	Yes
Linear trend	Yes	No	Yes	No
Quadratic trend	Yes	Yes	Yes	Yes
Station control variable	No	No	Yes	Yes
Weather control variables	No	No	Yes	Yes
City linear trend	No	Yes	No	Yes
GDP and Unemployment	No	No	Yes	Yes

Notes: The table presents first-stage estimates of precipitation on $PM_{2.5}$ for the mortality from circulatory and respiratory diseases (per 100,000 individuals aged <65 years) sample, across four specifications (columns (1) - (4)). Standard errors are clustered at the station level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A3: First stage estimates for infant mortality rate

	(1)	(2)	(3)	(4)
Precipitations	-16.9855***	-15.8956***	-17.3727***	-18.3076***
	(2.5854)	(2.7115)	(2.5703)	(2.7255)
Mean of Y	13.3422	13.3422	13.3422	13.3422
SD of Y	5.6934	5.6934	5.6934	5.6934
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	177	177	177	177
Observations cities	482	482	482	482
Observations stations	1,030	1,030	1,030	1,030
Station FE	Yes	Yes	Yes	Yes
Linear trend	Yes	No	Yes	No
Quadratic trend	Yes	Yes	Yes	Yes
Station control variable	No	No	Yes	Yes
Weather control variables	No	No	Yes	Yes
City linear trend	No	Yes	No	Yes
GDP and Unemployment	No	No	Yes	Yes

Notes: The table presents first-stage estimates of precipitation on $PM_{2.5}$ for the infant mortality rate per 1,000 live births sample, across four specifications (columns (1) - (4)). Standard errors are clustered at the station level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A4: Reduced form estimates

	(1)	(2)	(3)	(4)
Panel A: < 65 Cardio-Respiratory MR × 100,000				
Precipitations	-5.3862***	-5.3187***	-5.0155***	-4.8699**
	(1.6389)	(1.9234)	(1.7029)	(1.9828)
Mean of Y	3.4012	3.4012	3.4012	3.4012
SD of Y	1.1711	1.1711	1.1711	1.1711
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	180	180	180	180
Observations cities	429	429	429	429
Observations stations	915	915	915	915
Panel B: Infant MR × 1,000				
Precipitations	-6.4317**	-6.4181*	-5.4437*	-18.3076***
	(2.9217)	(3.3781)	(3.0244)	(3.4872)
Mean of Y	2.9665	2.9665	2.9665	2.9665
SD of Y	1.6519	1.6519	1.6519	1.6519
F-stat	29.6	62.6	31.1	53.4
p-value F-stat	8.968e-8	3.595e-14	4.58e-8	1.955e-12
Number of cities	75	75	75	75
Number of stations	177	177	177	177
Observations cities	482	482	482	482
Observations stations	1,030	1,030	1,030	1,030
Station FE	Yes	Yes	Yes	Yes
Linear trend	Yes	No	Yes	No
Quadratic trend	Yes	Yes	Yes	Yes
Station control variable	No	No	Yes	Yes
Weather control variables	No	No	Yes	Yes
City linear trend	No	Yes	No	Yes
GDP and Unemployment	No	No	Yes	Yes

Notes: The table presents reduced-form estimates of precipitation on the mortality rate for mortality from circulatory and respiratory diseases (per 100,000 individuals aged <65 years) (Panel A) and on the infant mortality rate per 1,000 live births (Panel B), across four specifications (columns (1) - (4)). Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A5: Rainfall correlations with GDP and lagged precipitations

	Precipitations	Precipitations
	(1)	(2)
GDP_t	0.0004	-
	(0.0005)	-
$Precipitations_{t-1}$	-	-0.1100-
	-	(0.0802)
$Precipitations_{t-2}$	-	0.0696
	-	(0.0726)
Observations	540	405
Number of cities	76	76
Mean of Y	0.0591	0.0571
SD of Y	0.0484	0.0478
Station FE	Yes	Yes
Linear trend	Yes	Yes
Quadratic trend	Yes	Yes
Station control variable	Yes	Yes
Weather control variables	Yes	Yes
City linear trend	No	No
GDP and Unemployment	Yes	Yes

Notes: The table presents OLS estimates of current GDP (t) on current precipitation (t) in column (1) and the OLS estimates of yearly lagged precipitation ($t-1$, $t-2$) on current precipitation (t) in column (2). The linear regressions follow preferred specification 3. The higher observation count relative to the main specification stems from excluding mortality outcomes, allowing broader analysis of precipitation, its lags, and GDP. Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A6: Correlations between migration to and from cities with weather conditions

	Immigration	Emigration	Net inflow
	(1)	(2)	(3)
$PM_{2.5}$	0.1806	-0.0778	0.2584
	(0.1957)	(0.0991)	(0.2272)
$PM_{2.5,t-1}$	-0.2159	-0.2359	0.0199
	(0.1736)	(0.1844)	(0.2162)
$Precipitations$	0.3139	0.1225	0.1913
	(0.7634)	(0.5649)	(0.3962)
$Precipitations_{t-1}$	0.6196	-0.1076	0.7271
	(0.4801)	(0.4995)	(0.4779)
$Windspeed$	-1.0966	-0.0502	-1.0464
	(0.8934)	(1.2187)	(0.7869)
$Windspeed_{t-1}$	-0.6413	0.1344	-0.7757
	(1.1808)	(0.3575)	(1.2679)
$Temperature$	-0.0556	0.5557	-0.61139
	(0.5616)	(0.4965)	(0.8216)
$Temperature_{t-1}$	0.0773	0.2157	-0.1384
	(0.6242)	(0.3793)	(0.6123)
GDP	-9.91e-06	9.02e-06	-0.0001
	(0.0001)	(8.53e-06)	(0.0001)
GDP_{t-1}	-0.0004	-0.0001	-0.0002
	(0.0002)	(0.0003)	(0.0002)
Constant	1282.92***	243.33	1039.59***
	(382.3498)	(325.84)	(282.88)
City FE	Yes	Yes	Yes
Year FE	Yes	Yes	Yes
City time trends	Yes	Yes	Yes
Observations	520	520	520
Number of cities	104	104	104
Mean of Y	21.9115	20.9551	0.9564
SD of Y	7.7940	6.03599	5.2366

Notes: The table presents OLS estimates of one year lagged ($t-1$) and contemporaneous (t): $PM_{2.5}$, *precipitation*, $Wind$ *speed*, *temperature* and GDP on current (t) Immigration (1), Emigration (2) and Net inflow (3). Outcome variables are standardised as rates per 1,000 municipal inhabitants. The linear regressions in columns (1)-(3) cover Italy only due to limited data availability for other countries. Standard errors are clustered at the city level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A7: Multiple instruments: rainfall, windspeed (2IV) and temperature (3IV)

	2IV		**3IV**	
	Cardio-Respiratory	Infant	Cardio-Respiratory	Infant
	MR $\times 100,000$	MR $\times 1,000$	MR $\times 100,000$	MR $\times 1,000$
	(1)	(2)	(3)	(4)
Panel A: TS2SLS				
$PM_{2.5}$	0.279***	0.1269	0.2969***	0.1806
	(0.0949)	(0.1585)	(0.0937)	(0.1562)
Mean of Y	3.4012	2.9665	3.4012	2.9665
SD of Y	1.1711	1.6519	1.1711	1.6519
Panel B: First stage				
Precipitations	-17.3727***	-17.3727***	-17.3727***	-17.3727***
	(2.5703)	(2.5703)	(2.5703)	(2.5703)
Wind speed	-0.4946***	-0.4946***	-0.4946***	-0.4946***
	(0.1369)	(0.1369)	(0.1369)	(0.1369)
Temperature	-	-	-0.0512	-0.0512
	-	-	(0.0933)	(0.0933)
Mean of Y	13.3422	13.3422	13.3422	13.3422
SD of Y	5.6934	5.6934	5.6934	5.6934
Sargan-stat	0.0176	5.6617	1.4517	8.5917
p-value Sargan	0.8945	0.01734	0.4839	0.0136
Number of cities	75	75	75	75
Number of stations	180	177	180	177
Observations cities	429	482	429	482
Observations stations	915	1,030	915	1,030

Notes: The table presents in Panel A, TS2SLS estimates of $PM_{2.5}$'s effect on mortality rates using multiple instrumental variables. Columns 1 and 3 show results for mortality from circulatory and respiratory diseases (per 100,000 individuals aged <65 years), while columns 2 and 4 report infant mortality (per 1,000 live births). Columns 1-2 use precipitation and wind speed as IVs; columns 3-4 add temperature. Panel B reports the corresponding first-stage estimates. The specification used is that of tables 1 and 2, column (3). Standard errors are clustered at the city level for TS2SLS estimates and at the station level in the first stage. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A8: First stage estimates for heterogeneity analysis

	Metropolitan areas		**Risk of Poverty rate**	
	Urban	Suburban	Above	Below
	(1)	(2)	(3)	(4)
Panel A: < 65 Cardio-Respiratory MR × 100,000				
Precipitations	-17.3336***	-18.0809***	-3.2843	-28.8275***
	(2.8580)	(5.7518)	(3.7547)	(3.2823)
Mean of Y	14.0865	12.7678	11.5960	15.9169
SD of Y	6.1844	4.6395	4.5773	6.3016
F-stat	30.8	2.7732	6.1475	24.3
p-value F-stat	5.788e-8	0.0981	0.0141	1.662e-6
Number of cities	67	27	46	44
Number of pollution stations	102	38	98	96
Observations cities	359	148	197	232
Observations stations	714	195	433	459
Panel B: Infant MR × 1,000				
Precipitations	-17.3336***	-18.0809***	-3.2843	-28.8275***
	(2.8580)	(5.7518)	(3.7547)	(3.2823)
Mean of Y	13.3693	12.5434	11.2802	15.1922
SD of Y	5.7819	4.4699	4.5920	5.7756
F-stat	30.8	2.7732	6.1475	24.3
p-value F-stat	5.788e-8	0.0981	0.0141	1.662e-6
Number of cities	67	27	47	44
Number of pollution stations	138	38	99	93
Observations cities	404	169	239	243
Observations stations	798	226	512	490

Notes: The table presents first-stage estimates of precipitation on $PM_{2.5}$ for mortality rate from circulatory and respiratory diseases (per 100,000 individuals aged <65 years) sample (Panel A) and for the infant mortality rate per 1,000 live births sample (Panel B), stratifying by: (1) urban vs. (2) suburban metropolitan areas, and by poverty risk: (3) above vs. (4) below median poverty rate. Urban areas are characterised by continuous built-up development, while suburban areas consist of largely built-up regions on the outskirts of urban centres. Cities are further categorised based on their economic status, with distinctions made between those above and below the poverty line (median). The specification used is that of tables 1 and 2, column (3). Standard errors are clustered at the station level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

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