When Air Gets Cleaner: The Health Impact of Industrial Pollution Abatement in Italy

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Abstract

We study the causal effect of PM2.5 exposure on respiratory morbidity by combining municipality-level administrative data on hospital admissions and in-hospital mortality in Italy (2013–2019) with high-resolution pollution measures. Focusing on municipalities hosting industrial plants, we use local wind direction as an instrumental variable to address endogeneity in pollution levels. Higher PM2.5 concentrations significantly increase emergency hospitalizations for COPD, pneumonia, and respiratory failure, with stronger effects among mild and moderate cases, indicating that pollution primarily triggers acute exacerbations. We then assess how the Industrial Emissions Directive (IED) altered this relationship, results show a marked decline in the pollution–health marginal effect after the IED's implementation, especially for COPD, pneumonia, and respiratory failure. Overall, the findings underscore the health burden of industrial emissions and the effectiveness of regulatory interventions in reducing pollution-related respiratory morbidity.

Keywords: air pollution, industrial emissions, health outcomes.
JEL classification: H75, J13, Q53, Q58.

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Introduction

Air pollution remains one of the most significant environmental threats to human health worldwide. Although European air quality has improved substantially over recent decades, exposure to fine particulate matter ($PM_{2.5}$) continues to generate considerable morbidity and mortality, particularly through respiratory and cardiovascular pathways. A vast literature documents these associations, yet credibly identifying the causal effects of pollution on health remains challenging. Pollution levels are jointly determined by economic activity, weather conditions, population density, commuting patterns, and behavioral responses, raising concerns of omitted variables and reverse causality that bias standard observational estimates (Zivin and Neidell, 2009; Moretti and Neidell, 2011; Arceo, Hanna and Oliva, 2016; Deschenes et al., 2017). Moreover, issues such as mortality displacement ("harvesting") further complicate the interpretation of short-run health impacts.

This paper provides new causal evidence on the health effects of $PM_{2.5}$ exposure in Italy between 2013 and 2019, focusing specifically on municipalities that host at least one industrial plant. Industrial facilities—defined according to the European Environment Agency's Industrial Reporting Database and regulated under the European Pollutant Release and Transfer Register (E-PRTR) and the Industrial Emissions Directive (IED)—represent major sources of air emissions and thus create substantial spatial heterogeneity in ambient pollution. Restricting the analysis to municipalities with plants allows us to study populations most exposed to industrial pollution, and to evaluate regulatory impacts in a policy-relevant subset of Italian territory. Our empirical strategy exploits month-to-month variation in local wind direction to generate plausibly exogenous fluctuations in pollution exposure. Following the logic of Deryugina et al. (2019) and Zivin et al. (2023), winds transport pollutants from industrial sources toward downwind municipalities, creating high-frequency variation that is unrelated to contemporaneous economic or behavioral conditions. By combining detailed meteorological data, rich geolocated $PM_{2.5}$ information, and comprehensive administrative hospital records—including emergency respiratory admissions and in-hospital mortality—we implement an instrumental-variable design that isolates causal changes in exposure.

Italy offers an ideal empirical setting for three reasons. First, industrial activity is highly geographically concentrated, particularly in Northern regions such as Lombardy, Veneto, and Emilia–Romagna, generating sharp spatial gradients in emissions. Second, the country's complex geography—including the Po Valley basin, coastal regions, and mountainous areas—amplifies spatial variation in pollutant dispersion. Third, Italy's nationwide Hospital Discharge Registry (SDO) provides near-universe coverage of emergency admissions and mortality, with sufficiently detailed diagnostics to distinguish mild, moderate, and severe respiratory cases. The combination of granular health data, detailed environmental information, and natural variation from wind direction enables a

clean quasi-experimental design.

Beyond estimating the baseline response of respiratory health to $PM_{2.5}$ exposure, a central contribution of this paper is to evaluate whether these effects changed following the implementation of the Industrial Emissions Directive (IED). The IED, adopted in 2010 and fully enforced in Italy from 2015–2016 after a delayed transposition, imposed stricter emission limits and mandated the use of Best Available Techniques (BATs) for industrial facilities—particularly Large Combustion Plants (LCPs) with thermal capacity above 50 MW. While emissions from European large combustion and industrial plants dropped sharply after 2015, causal evidence on the health consequences of these regulatory changes remains scarce. To measure regulatory exposure, we exploit detailed information on each facility's main industrial activity. Only installations operating in sectors explicitly covered by the Industrial Emissions Directive—such as energy production, metal industries, mineral processing, chemicals, waste management, and other heavy industrial activities—are fully subject to its emission standards, whereas plants in lighter industrial sectors fall outside the scope of the Directive or face substantially weaker requirements. We classify municipalities as "treated" if they host at least one regulated facility. Interacting this treatment indicator with a post-2016 dummy allows us to study whether the health effects of pollution attenuated in municipalities exposed to stricter regulatory standards.

Our paper contributes to three main strands of literature. First, it adds to work using quasi-experimental variation to identify the health impacts of air pollution (Chay and Greenstone, 2003; Currie and Neidell, 2005; Arceo et al. 2016; Deryugina et al. 2019; Zivin et al. 2023). We implement, for the first time in Italy, a wind-based IV design focused on industrial municipalities. Second, we contribute to the literature on the environmental and health effects of industrial regulation, complementing studies on the effectiveness of European emission standards (EEA 2023). Our estimates show how the baseline health impacts of PM2.5 evolved after the IED's full enforcement. Third, we add to research examining heterogeneity across disease severity, showing that PM2.5 increases mild and moderate emergency admissions substantially more than severe cases—consistent with pollution acting primarily as an acute trigger of exacerbations (Schlenker and Walker, 2016; Anderson 2020). Overall, this study provides the first causal evidence linking industrial pollution, wind-driven exposure, and respiratory health in Italy, while also quantifying how these relationships changed following major regulatory tightening under the IED. The findings highlight the important health benefits of improved industrial emission standards and contribute to the ongoing debate on the effectiveness of European environmental policy.

Institutional Background

The Industrial Emissions Directive (IED). The Industrial Emissions Directive (IED, Directive 2010/75/EU) is the cornerstone of the European Union's regulatory framework for controlling pollution from large industrial installations. Adopted in 2010, it consolidated and replaced seven previous directives—including the 2001 Large Combustion Plant (LCP) Directive—with the objective of reducing harmful emissions into air, water, and soil, while ensuring a high level of environmental and human health protection.

The IED applies to industrial installations covered by Annex I, which lists activities with the highest pollution potential. These include energy industries, metal production, mineral processing, chemical manufacturing, waste management, and other large-scale industrial operations. Covered facilities must obtain an integrated environmental permit and comply with emission limit values (ELVs) based on *Best Available Techniques* (BAT) and associated emission levels (BAT-AELs), as defined in legally binding BAT Reference Documents (BREFs).

A central feature of the IED is the requirement that all installations adopt BATs and respect BAT-AELs within four years of the publication of updated BREF documents. This mechanism ensures progressive tightening of environmental standards as technology improves. For Large Combustption Plants (LCPs)—installations with a rated thermal input of at least 50 MW—the directive imposed particularly stringent limits on sulfur dioxide (SO_2), nitrogen oxides (NO_x), and particulate matter emissions, contributing substantially to the EU-wide decline in industrial air pollution.

Implementation in the European Union and Italy. EU Member States were required to transpose the IED into national legislation by January 2013. However, several countries, including Italy, experienced delays. The European Commission opened an infringement procedure against Italy in 2013 for failure to transpose the directive on time. The IED was eventually implemented through Legislative Decree No. 46 of 4 March 2014, which entered into force on 11 April 2014.

Although formal transposition occurred in 2014, full enforcement of the directive — particularly for LCPs — took effect only in 2015–2016, when BAT conclusions for the sector became binding. This timing is consistent with both regulatory documents and evidence presented by the European Environment Agency (EEA), which reports substantial declines in LCP emissions after 2015. Between 2004 and 2022, emissions from European LCPs fell dramatically: SO_2 and dust emissions declined by roughly 92%, while NO_x emissions dropped by about 70%. Two key turning points contributed to these reductions: (i) the 2007–2009 modernization wave triggered by the LCP Directive and (ii) the post-2015 tightening under the IED.

Italy's industrial structure—with high spatial concentration and a large share of older

combustion plants—makes the country particularly sensitive to regulatory stringency. The 2015–2016 enforcement phase therefore represents a natural break suitable for empirical analysis.

Definition of Industrial Plants and Treatment Status. We identify industrial plants following the definition adopted by the European Environment Agency Industrial Reporting Database[1]. In this framework, an industrial plant corresponds to a geographically defined production unit that is required to report emissions under the European Pollutant Release and Transfer Register (E-PRTR) and, when applicable, is subject to the Industrial Emissions Directive (IED). Plants constitute the administrative level at which environmental releases are monitored, and they serve as parent entities for installations regulated under specific IED chapters (e.g. large combustion plants, waste incineration and co-incineration).

A crucial step for the empirical design is to determine which plants are effectively regulated by the IED. For this purpose, we rely on the variable `EPRTRAnnexIMainActivityCode`, which classifies facilities according to the IED Annex I activity categories. Only activities falling under categories 1 to 6 are subject to the directive's emission limits and monitoring requirements:

- Category 1: Energy industries;
- Category 2: Production and processing of metals;
- Category 3: Mineral industry;
- Category 4: Chemical industry;
- Category 5: Waste management;
- Category 6: Other regulated industrial activities (e.g., pulp and paper, food processing, intensive livestock).

Activities classified in categories 7–9 fall outside the IED regulatory scope or are only lightly regulated. Since our dataset includes facilities with activity codes ranging from 1 to 9, this classification provides meaningful variation in regulatory exposure.

We therefore define a facility as treated if its activity code belongs to categories 1–6:

$$Treated_s = \begin{cases} 1 & \text{if } EPRTRAnnexIMainActivityCode_s \in \{1,2,3,4,5,6\}, \\ 0 & \text{otherwise.} \end{cases}$$

[1] https://www.eea.europa.eu/en/datahub/datahubitem-view/9405f714-8015-4b5b-a63c-280b82861b3d

At the municipality level, we classify a location as treated if it hosts at least one such regulated plant. The post-reform period begins in 2016, corresponding to the year in which the IED became fully enforceable for existing installations.

Industrial plants are highly unevenly distributed across Italy, and this spatial heterogeneity motivates our focus on municipalities directly affected by industrial emissions. Figure 1 illustrates the geographic distribution of municipalities hosting at least one industrial plant in our dataset. The pattern mirrors Italy's broader industrial geography: a dense concentration of regulated facilities in the Northern regions—particularly Lombardy, Veneto, and Emilia–Romagna—alongside additional clusters in Central and Southern regions. Municipalities in the South and the islands contain far fewer plants, and these tend to be isolated rather than forming contiguous industrial zones.

This spatial structure underscores why focusing on municipalities with industrial facilities is essential for studying both pollution exposure and the potential impacts of the IED.

Figure 1: Municipalities hosting at least one industrial plant (2013–2018).

Data

We construct a novel municipality-level dataset by integrating multiple data sources: (i) administrative Hospital Discharge Records (HDRs) from the Italian Ministry of Health; (ii) pollution exposure measures from the Copernicus Atmosphere Monitoring Service (CAMS); and (iii) information on industrial plants from the Industrial Reporting Database. Our analysis covers the period 2013–2018 and includes approximately 1240 municipalities and 1230 hospitals.

Health Data. We use administrative *Hospital Discharge Records* from the Italian Ministry of Health covering 2013–2019. The dataset includes information on all emergency hospital admissions and in-hospital deaths across public and private hospitals nationwide. Each record contains detailed diagnostic codes based on the International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM), which is currently the standard coding system in Italy for hospital diagnoses and procedures (Giordani et al., 2024).

The HDR database not only includes basic demographic information such as patient age, sex, and region of residence, but also clinical details like the primary and secondary diagnoses, lengths of hospital stay, and discharge status. It is a valuable source for longitudinal health outcome analysis, as it captures the complexity of hospitalizations including comorbidities and treatment episodes (European Health Information Portal, 2023).

Due to its administrative nature, the HDR is routinely collected and subject to standardized coding practices regulated by ministerial decrees, ensuring consistency in diagnosis classification across regions. This makes the HDR a reliable tool for research on respiratory and other health outcomes linked to environmental exposures.

We have grouped respiratory conditions into four principal categories, derived from the ICD-9-CM chapter [2] on respiratory diseases (codes 460–519), with the objective to isolate key conditions commonly affected by air pollution exposure:

- **Chronic Obstructive Pulmonary Disease (COPD)**: Codes 490–496, encompassing bronchitis and chronic bronchitis, emphysema, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction, not elsewhere classified, which generally refers to chronic airflow obstruction consistent with COPD but not otherwise specified.

- **Asthma**: Code 493, representing bronchial asthma as a distinct obstructive airway disease.

[2] https://www.salute.gov.it/new/it/tema/assistenza-ospedaliera/il-manuale-icd9cm/

- **Pneumonia**: Code 486, indicating unspecified pneumonia cases, a major acute respiratory infection.

- **Respiratory failure**: Codes 518.81–518.84, covering acute and chronic respiratory failure conditions with clinical severity implications.

We further stratify cases into *mild*, *moderate*, and *severe* groups based on secondary diagnoses and comorbidities indicative of disease exacerbation and complexity. This hierarchical severity classification allows us to differentiate health outcomes according to the clinical burden and underlying patient health status.

Mortality is defined as in-hospital deaths where either a primary or any secondary diagnosis corresponds to one of the above respiratory categories, ensuring comprehensive capture of fatal respiratory events within hospitalization episodes.

The four panels in Figure 1 reveal a clear and consistent seasonal pattern in respiratory hospitalizations. All outcomes display a pronounced U-shaped profile, with admissions peaking during the winter months, declining steadily through spring, reaching a minimum during July–September, and rising again in the autumn.

Air Pollution and Meteorology. Monthly $PM_{2.5}$ concentration data used in this study are drawn from the Copernicus Atmosphere Monitoring Service (CAMS) at a municipality level. Since our analysis focuses on Italian municipalities hosting at least one industrial plant, $PM_{2.5}$ exposure is measured only for this subset of municipalities.

To control for meteorological influences, we incorporate monthly covariates including temperature, precipitation, radiation, evapotranspiration, and vapor pressure, all derived from the ERA5-Land reanalysis dataset at a municipality level. These meteorological variables were selected to accurately capture environmental conditions potentially affecting pollutant levels and health outcomes.

Wind Direction Variable. A major challenge in estimating the health effects of $PM_{2.5}$ is that pollution levels are not randomly assigned but instead co-move with economic activity, weather conditions, mobility patterns, and demographic factors. To address this endogeneity, we follow the strategy of Zivin et al. (2023) and Deryugina et al. (2019) and exploit exogenous monthly variation in wind direction. The identifying idea is that, conditional on local meteorological conditions, shifts in prevailing wind direction alter the origin of incoming air masses—and thus local pollution concentrations—without directly affecting health outcomes.

The wind direction variable is constructed at the municipality–month level. Meteorological data used to compute the variable, which includes zonal (u) and meridional (v) wind components, are obtained from the Copernicus Climate Data Store (CDS) while geographic coordinates for Italian municipalities come from the Istituto Geografico Militare

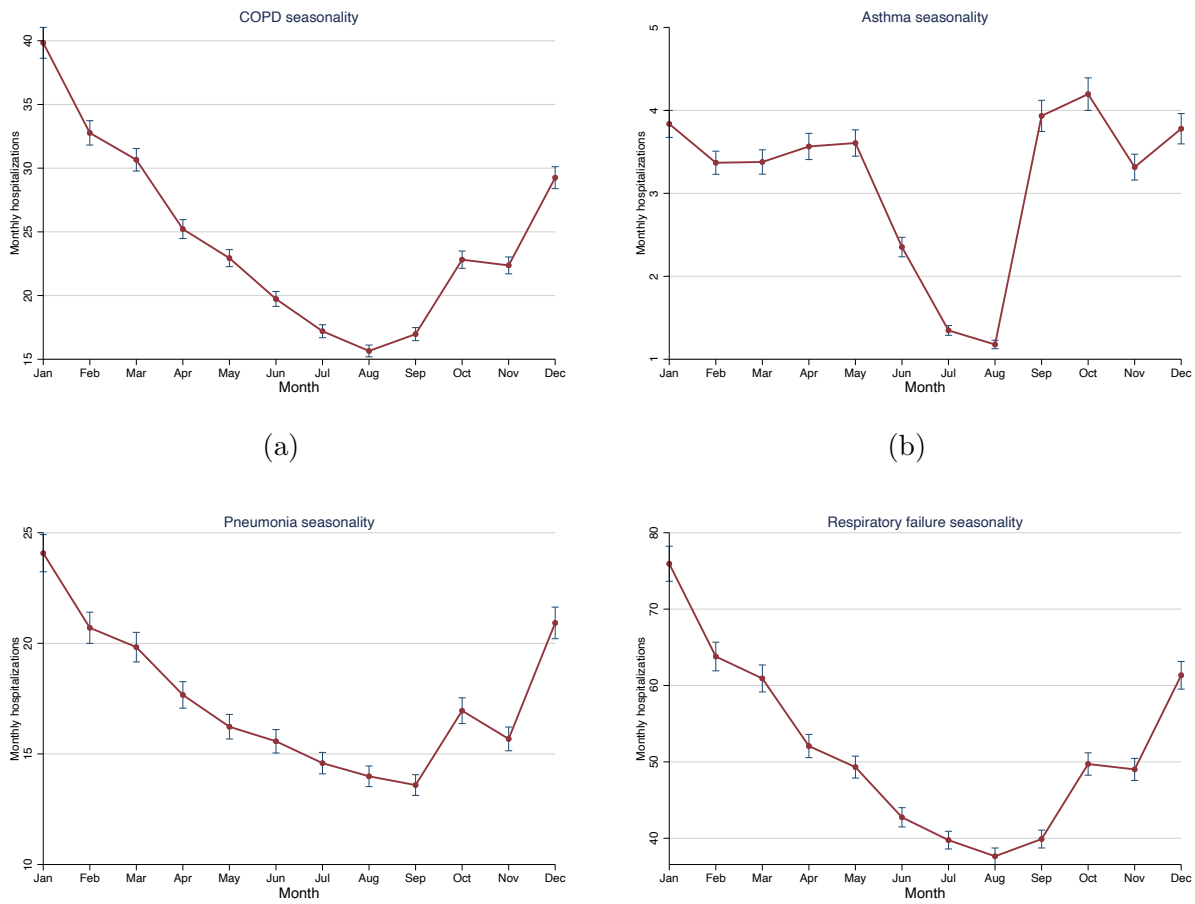

Figure 2: Seasonality of monthly hospitalizations for respiratory outcomes. Panel (a) reports COPD seasonality, Panel (b) asthma, Panel (c) pneumonia, and Panel (d) Respiratory failure.

(IGM).

Wind direction is computed using the two-dimensional arctangent of the negated wind components, which yields the direction from which the wind originates rather than the direction it is blowing toward. Specifically, for each municipality i and month t:

$$\text{Wind\_dir}_{it} = 180/\pi * \arctan 2(-u_{it}, -v_{it}),$$

producing values in the range $(-\pi, \pi)$. These were then converted into compass degrees (0°–360°) to facilitate quadrant classification.

Each monthly observation was assigned to one of four broad compass quadrants:

- **NE**: $[45°, 135°)$
- **SE**: $[135°, 225°)$
- **SW**: $[225°, 315°)$

- **NW**: $[315°, 360°)$ and $[0°, 45°)$.

We then computed wind speed for each municipality i and month t:

$$\text{Wind\_speed}_{it} = \sqrt{u_{it}^2 + v_{it}^2}.$$

This variable will be used as a control.

Empirical Strategy

Our empirical analysis focuses on Italian municipalities hosting at least one industrial plant regulated under the European Pollutant Release and Transfer Register (E–PRTR) and the Industrial Emissions Directive (IED). In these municipalities, industrial emissions represent a major source of local ambient $PM_{2.5}$, making them a relevant environment for studying the causal health impacts of particulate exposure.

Because pollution levels may be endogenous, we estimate a two-stage least squares (2SLS) model that uses monthly variation in wind direction as an instrument for local $PM_{2.5}$ exposure. The identifying idea, following Zivin et al. (2023) and Deryugina et al. (2019), is to exploit the exogenous variation in pollution exposure induced by changes in wind directions. Changes in prevailing wind direction shift the origin of incoming air masses in a manner that is orthogonal to local health shocks but strongly predictive of measured pollution. In other words, the wind blowing from a particular direction moves pollution within the municipality, but also brings in pollution from outside. Thus, stronger or more persistent winds from a given industrial quadrant increase local exposure to emissions originating from that direction, thereby generating exogenous variation in $PM_{2.5}$ concentrations.

Baseline 2SLS specification. For each municipality i and quadrant q, we construct a pollution shock that captures how polluted the air tends to be when winds originate from quadrant q. Following equation (3) in Zivin et al. (2023), we compute:

$$\widetilde{PM}_{iq} \;=\; \frac{1}{N_{iq}} \sum_{t \in q} PM_{it} \;-\; \frac{1}{N_i} \sum_{t} PM_{it}, \tag{1}$$

where the first term is the average $PM_{2.5}$ concentration in municipality i during months when winds came from quadrant q, and the second term is the municipality's overall mean $PM_{2.5}$. This measure captures whether air masses arriving from direction q historically bring cleaner or more polluted air into municipality i. Because our analysis focuses on municipalities hosting at least one industrial plant, this quantity reflects persistent spatial asymmetries in pollution dispersion relative to fixed emission sources.

In the second step, we interact these pollution shocks with the monthly wind direction to create a set of time-varying instruments Z_{iqt}. Each instrument equals the historical shock $\widetilde{PM}_{iq}$ only in months when the prevailing wind in municipality i comes from quadrant q:

$$Z_{iqt} = \begin{cases} \widetilde{PM}_{iq}, & \text{if } WindDir_{it} = q, \\ 0, & \text{otherwise.} \end{cases}$$

Thus, the instrument varies over time with prevailing wind direction, while its magnitude is determined by the municipality-specific historical relationship between winds and pollution.

The resulting instruments enter the first-stage relationship between wind-driven variation and $PM_{2.5}$ as follow:

$$PM_{2.5,it} = \pi_0 + \pi_1 Ziq_{it}^{NE} + \pi_2 Ziq_{it}^{SE} + \pi_3 Ziq_{it}^{SW} + \pi_4 Ziq_{it}^{NW} + X_{it}\theta + \delta_i + \lambda_t + u_{it}, \quad (2)$$

where X_{it} includes monthly meteorological controls (average temperature, precipitation, evapotranspiration, solar radiation, and vapor pressure). We include municipality $\times$ year-quarter fixed effects (δ_i) to absorb time-invariant and seasonal differences across locations, and year $\times$ month fixed effects (λ_t) to flexibly control for national time trends.

The predicted values $\widehat{PM}_{2.5,it}$ are then used in the second stage:

$$Y_{it} = \alpha + \beta \widehat{PM}_{2.5,it} + X_{it}\gamma + \delta_i + \lambda_t + \varepsilon_{it}, \quad (3)$$

where Y_{it} is the number of respiratory hospital admissions or in-hospital deaths in municipality i and month t. Identification relies on within-municipality monthly changes in wind direction that alter exposure to industrial emissions but do not directly affect health outcomes. Then, to capture also heterogeneity in clinical severity, each specification is estimated separately for mild, moderate, and severe hospitalizations, as well as for the corresponding in-hospital mortality outcomes. The econometric structure of the model remains identical across severity groups; what varies is simply the definition of the dependent variable Y_{it}.

Post-IED specification. To study how the health effects of particulate exposure evolved following the implementation of the Industrial Emissions Directive (IED), we augment the baseline model with an interaction between $PM_{2.5}$ and an indicator for the post-IED period. This analysis is motivated by the fact that the IED substantially tightened emission standards for large combustion plants (LCPs), waste incinerators, and a broad set of industrial installations regulated under Annex I of the directive. If the directive effectively reduced industrial emissions, then the marginal health impact of $PM_{2.5}$ exposure

should weaken after its enforcement because a given increase in pollution would reflect cleaner industrial processes, lower toxic content, or fewer extreme pollution episodes.

We define the post-IED period as beginning in 2016, corresponding to the point at which the directive became fully operational for existing plants.

The post-IED exposure is defined as the interaction

$$\text{postIED}_{it} = \text{treated}_f \times \text{post}_t,$$

where $treated_f$ are the facilities with the activity code (EPRTRAnnexIMainActivityCode) belonging to categories 1-6, and $post_t$ is a post-2016 dummy. The variable varies across municipalities depending on the industrial composition of local plants and over time depending on the regulatory calendar. This term captures how the causal effect of $PM_{2.5}$ changes once IED standards become binding for regulated facilities.

The model with the post-IED indicator becomes:

$$Y_{it} = \alpha + \beta_1 \widehat{PM}_{2.5,it} + \beta_2 \widehat{PM}_{2.5,it} \times \text{postIED}_{it} + X_{it}\gamma + \delta_i + \lambda_t + \varepsilon_{it}, \tag{4}$$

where $\widehat{PM}_{2.5,it}$ and $\widehat{PM}_{2.5,it} \times \text{postIED}_{it}$ are the fitted values from the two first-stage equations considering the wind direction instruments. Equation (4) identifies two causal parameters:

- β_1: the pre-IED causal effect of $PM_{2.5}$ on outcome Y_{it},
- β_2: the change in the pollution effect after IED enforcement.

A negative β_2 indicates that industrial emission standards introduced by the IED reduce the marginal health damages of particulate exposure.

Results

Main Estimates. Table 1 reports the baseline effects of $PM_{2.5}$ exposure on monthly respiratory hospitalizations and in-hospital mortality. Panel A presents OLS estimates, while Panel B shows the corresponding IV results using the wind-direction instruments. In all cases, the IV coefficients are substantially larger than the OLS ones, a pattern consistent with attenuation bias in the OLS model due to classical measurement error in $PM_{2.5}$ and with downward bias arising from reverse causality between respiratory demand and local emissions. The first-stage F-statistic (around 295) confirms that the wind-direction instruments are highly relevant.

Across outcomes, higher particulate concentrations significantly increase emergency hospitalizations for COPD, pneumonia, and respiratory failure, as well as deaths associated with these conditions. The magnitudes imply that even relatively modest month-

to-month changes in $PM_{2.5}$ lead to sizeable fluctuations in hospital admissions, which is consistent with the short-run triggering role of air pollution documented in Deryugina et al. (2019), Heissel et al. (2023), and Zivin et al. (2023). These conditions — in particular COPD and respiratory failure — exhibit strong sensitivity to acute environmental shocks, as high levels of inflammation and impaired lung function make patients more vulnerable to transient reductions in ambient air quality.

By contrast, the estimated coefficient for asthma is negative and statistically significant. This should not be interpreted as evidence that asthma risk falls when pollution rises, but rather as a consequence of how rare and noisy asthma hospitalizations are in our data. Even in a sample of urgent admissions, asthma accounts on average for fewer than three hospitalizations per municipality-month, so a small absolute change in case counts translates into a relatively large change in the regression coefficient. In addition, diagnostic coding may reclassify episodes with overlapping symptoms — for example, an acute event in a patient with both asthma and COPD or pneumonia. In such cases, pollution-driven exacerbations that clinically involve asthma may be recorded in the COPD or pneumonia categories rather than under asthma itself (Currie and Neidell, 2005; Schlenker and Walker, 2016). Taken together, the low baseline incidence, high relative noise, and potential diagnostic substitution across respiratory categories plausibly explain the negative asthma coefficient, without contradicting the strong positive effects found for other respiratory outcomes.

Panel C reports the first-stage coefficients linking wind direction to $PM_{2.5}$. All four instruments (NE, SE, SW, NW quadrants) display positive and precisely estimated effects. This is expected in settings where wind direction shifts pollutant inflow but average concentrations are always higher when winds bring air from industrial zones relative to the municipal centroid. A similar pattern is found in the U.S. literature studying large point sources, such as coal-fired plants (Deryugina et al. 2019), where winds blowing from the direction of industrial facilities uniformly increase local pollution regardless of the specific quadrant. In our context, industrial plants are spatially concentrated around municipalities in the sample; thus, winds from any direction tend to raise $PM_{2.5}$ levels relative to calmer conditions, generating uniformly positive first-stage coefficients.

Overall, the IV estimates provide strong evidence that $PM_{2.5}$ remains a major driver of respiratory morbidity and mortality in Italian municipalities hosting industrial plants. The magnitude and precision of the effects support a causal interpretation and highlight the importance of addressing industrial emissions as a central component of public health policy.

Table 1: Effects of $PM_{2.5}$ on health outcomes

	COPD	Pneumonia	Asthma	Resp_failure	Resp	D_COPD	D_pneumonia	D_asthma	D_resp_failure	D_resp
Panel A: OLS estimates										
$Pm_{2.5}$	0.148***	0.048**	-0.026***	0.264***	0.353***	0.025***	-0.010***	0.001***	0.009	0.017
	(0.028)	(0.020)	(0.008)	(0.043)	(0.063)	(0.004)	(0.004)	(0.000)	(0.012)	(0.014)
Panel B: IV-2SLS estimates										
$Pm_{2.5}$	1.168***	0.320***	-0.146***	1.218***	2.067***	0.076***	0.072***	0.002*	0.143*	0.245***
	(0.175)	(0.090)	(0.052)	(0.259)	(0.384)	(0.025)	(0.021)	(0.001)	(0.077)	(0.089)
Panel C: First-stage estimates										
ZiqNE	0.294***	0.294***	0.294***	0.294***	0.294***	0.294***	0.294***	0.294***	0.294***	0.294***
	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)
ZiqSE	0.220***	0.220***	0.220***	0.220***	0.220***	0.220***	0.220***	0.220***	0.220***	0.220***
	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)
ZiqSW	0.456***	0.456***	0.456***	0.456***	0.456***	0.456***	0.456***	0.456***	0.456***	0.456***
	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)
ZiqNW	0.255***	0.255***	0.255***	0.255***	0.255***	0.255***	0.255***	0.255***	0.255***	0.255***
	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.017)
Observations					99,034					
First-stage F-stat					295.2184					
Mean of Y	24.637	17.493	3.157	51.880	86.068	1.428	1.797	0.011	10.814	13.122
SD of Y	35.818	28.280	7.005	73.276	116.192	2.547	3.467	0.115	15.380	18.395

Notes: Robust SE in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. Models include City×Year_Quarter and Year_Month fixed effects; standard errors clustered at the City level.

Heterogeneity by hospitalization severity. Table 2 explores how the health effects of $PM_{2.5}$ vary across the severity of hospital admissions. A clear gradient emerges. The largest and most precisely estimated effects appear for mild and moderate respiratory cases, whereas the coefficients for severe cases are substantially smaller in magnitude. This pattern is consistent with the clinical interpretation of respiratory disease progression. Mild and moderate cases—such as COPD exacerbations, acute episodes of wheezing, or moderate pneumonia—are highly sensitive to short-run fluctuations in ambient pollution. Acute exposure to particulate matter can trigger inflammation, airway constriction, or infection-related complications within hours or days, generating a rapid increase in emergency admissions. Similar dynamics are documented in Deryugina et al. (2019) and Schlenker and Walker (2016), who show that short-term pollution spikes disproportionately affect conditions characterized by acute exacerbations. In contrast, severe hospitalizations often reflect chronic disease progression, multimorbidity, or underlying frailty. These cases may be less responsive to short-run pollution shocks, either because their baseline risk is already elevated or because clinical deterioration occurs over longer time horizons (e.g., chronic respiratory failure, long-standing COPD, or severe pneumonia). This attenuated responsiveness mirrors evidence in Zivin et al. (2023), who show that acute pollution shocks primarily affect marginal patients—those whose health status is most likely to shift at the margin in response to environmental stressors. Overall, the severity-stratified results suggest that $PM_{2.5}$ acts primarily as a trigger for acute respiratory episodes rather than as an immediate driver of severe, chronic deterioration. This interpretation underscores the importance of pollution control policies in preventing acute healthcare surges, particularly in industrial municipalities where exposure levels and vulnerability may be higher.

Table 2: IV Effects of $PM_{2.5}$ on Health Outcomes by Severity

	COPD	Pneumonia	Asthma	Resp_failure	Resp	D_COPD	D_pneumonia	D_asthma	D_resp_failure	D_resp
Panel A: Mild cases										
$pm_{2.5,m}$	0.196***	0.032.	-0.210***	0.721***	0.739***	0.006**	0.005*	0.000.	0.119*	0.131*
	(0.032)	(0.030)	(0.042)	(0.190)	(0.212)	(0.003)	(0.003)	(0.000)	(0.070)	(0.071)
Mean of Y	2.908	3.755	1.839	39.427	47.928	0.051	0.143	0.000	9.352	9.546
SD of Y	5.696	7.238	4.636	55.515	64.769	0.278	0.517	0.019	13.350	13.543
Panel B: Moderate cases										
$Pm_{2.5,m}$	0.901***	0.247***	0.042***	.	1.189***	0.066***	0.058***	0.000.	.	0.125***
	(0.154)	(0.065)	(0.015)	(.)	(0.191)	(0.023)	(0.017)	(0.001)	(.)	(0.030)
Mean of Y	20.041	11.796	0.873	.	32.709	1.228	1.187	0.004	.	2.419
SD of Y	29.539	18.902	1.997	.	46.456	2.273	2.386	0.068	.	4.076
Panel C: Severe cases										
$Pm_{2.5,m}$	0.071***	0.041*	-0.005.	0.497***	0.604***	0.004.	0.009.	0.002*	0.024.	0.039.
	(0.018)	(0.025)	(0.014)	(0.104)	(0.121)	(0.005)	(0.011)	(0.001)	(0.022)	(0.028)
Mean of Y	1.688	1.943	0.783	12.453	16.867	0.149	0.468	0.006	1.461	2.085
SD of Y	3.292	4.526	1.790	19.094	24.913	0.486	1.168	0.091	2.559	3.500
Observations					99,034					
First-stage F-stat (IV)					295.2184					

Notes: Robust SE in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. All IV models include FE City#Year_Quarter and Year_Month; SE clustered at City. Instruments: Ziq{NE,SE,SW,NW}.

Results post-IED

Table 3 evaluates whether the health impact of particulate exposure changed after the full enforcement of the Industrial Emissions Directive (IED). Across all outcomes, the interaction term $PM_{2.5} \times post\_IED$ is negative, sizeable, and statistically significant in both OLS and IV estimates. This pattern indicates that the marginal effect of $PM_{2.5}$ on respiratory health became substantially smaller after 2016, consistent with the IED reducing the toxicity and intensity of industrial emissions.

Quantitatively, the IV coefficients point to a marked moderation of pollution-related harms. For example, the effect of $PM_{2.5}$ on COPD admissions declines from 1.28 pre-IED to approximately $1.28 - 0.82 \approx 0.46$ in the post-IED period. A similar proportional reduction is observed for pneumonia, respiratory failure, total respiratory admissions, and in-hospital deaths. Mortality outcomes display particularly pronounced attenuation, suggesting that emission standards affected both exposure levels and the composition of pollutants emitted by industrial facilities.

This attenuation is consistent with the regulatory mechanisms introduced by the IED, which mandated the adoption of Best Available Techniques (BATs) and stricter emission ceilings for combustion-intensive sectors (Annex I categories 1–6). Evidence from other European contexts supports similar improvements: Brehm (2022) documents sharp declines in particulate and sulfur emissions following LCP regulation in Germany, while Gehrsitz (2017) reports meaningful reductions in ambient pollution after industrial emission standards were tightened in the UK. Our findings complement this literature by showing that regulatory improvements translate into measurable reductions in pollution-related health damages.

Importantly, the negative interaction does not imply that $PM_{2.5}$ ceased to matter after 2016. Even in the post-regulation period, estimated coefficients remain positive and significant, consistent with medical evidence that health effects persist even at relatively low concentrations (Liu et al., 2019; Deryugina et al., 2019). Instead, the results indicate that the IED substantially mitigated—but did not eliminate—the health burden generated by industrial emissions in municipalities hosting regulated plants.

A noteworthy feature of the first stage is that, once interacted with the post-IED indicator, some wind-direction instruments become negative. This pattern is mechanically consistent with the construction of the instruments: Ziq_{it} captures the differential pollution load associated with air masses arriving from quadrant q, and the interaction $Ziq_{it} \times post\_IED$ reflects how this directional contribution changes after emission standards tightened. Because the IED reduced pollution more strongly for winds blowing from directions aligned with major industrial sources, the post-IED differential often flips sign, generating negative coefficients in the interacted first stage. This does not undermine instrument validity; rather, it confirms that the IED altered the spatial distribution of

emissions in a manner fully consistent with the policy's intended effects.

Overall, the consistent reductions across all outcomes reinforce the regulatory effectiveness of the IED and highlight the public-health relevance of continued emission monitoring and enforcement in industrial municipalities.

Table 3: Effects of $PM_{2.5}$ on respiratory outcomes with post-IED interaction

	COPD	Pneumonia	Asthma	Resp_failure	Resp	D_COPD	D_pneumonia	D_asthma	D_resp_failure	D_resp
Panel A: OLS estimates										
$pm_{2.5,m}$	0.171***	0.055***	-0.020**	0.296***	0.404***	0.026***	-0.010**	0.002***	0.018.	0.028*
	(0.031)	(0.021)	(0.008)	(0.045)	(0.067)	(0.004)	(0.004)	(0.000)	(0.014)	(0.015)
$pm_{2.5,m}$×post_IED	-0.097***	-0.029.	-0.021**	-0.132**	-0.211***	-0.004.	-0.002.	-0.001***	-0.035.	-0.047**
	(0.033)	(0.029)	(0.010)	(0.055)	(0.076)	(0.007)	(0.007)	(0.000)	(0.021)	(0.023)
Panel B: IV-2SLS estimates										
$pm_{2.5,m}$	1.280***	0.393***	-0.122**	1.413***	2.433***	0.085***	0.074***	0.003**	0.198***	0.307***
	(0.182)	(0.090)	(0.051)	(0.263)	(0.396)	(0.026)	(0.020)	(0.001)	(0.075)	(0.087)
$pm_{2.5,m}$×post_IED	-0.818***	-0.582***	-0.119**	-1.727***	-2.987***	-0.073*	-0.021.	-0.004**	-0.537***	-0.607***
	(0.253)	(0.171)	(0.059)	(0.436)	(0.662)	(0.042)	(0.048)	(0.002)	(0.153)	(0.170)
Panel C: First-stage (depvar = $pm_{2.5,m}$)										
ZiqNE	0.296***	0.296***	0.296***	0.296***	0.296***	0.296***	0.296***	0.296***	0.296***	0.296***
	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)	(0.026)
ZiqSE	0.212***	0.212***	0.212***	0.212***	0.212***	0.212***	0.212***	0.212***	0.212***	0.212***
	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)	(0.031)
ZiqSW	0.476***	0.476***	0.476***	0.476***	0.476***	0.476***	0.476***	0.476***	0.476***	0.476***
	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)	(0.023)
ZiqNW	0.266***	0.266***	0.266***	0.266***	0.266***	0.266***	0.266***	0.266***	0.266***	0.266***
	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)	(0.018)
Panel C: First-stage (depvar = $pm_{2.5,m}$×post_IED)										
ZiqNE	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***	-0.114***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)
ZiqSE	0.051***	0.051***	0.051***	0.051***	0.051***	0.051***	0.051***	0.051***	0.051***	0.051***
	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)	(0.009)
ZiqSW	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***	-0.040***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
ZiqNW	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
Observations					99,034					
First-stage F ($pm_{2.5,m}$)					154.0804					
First-stage F ($pm_{2.5,m}$×post_IED)					129.4009					
Mean of Y	24.637	17.493	3.157	51.880	86.068	1.428	1.797	0.011	10.814	13.122
SD of Y	35.818	28.280	7.005	73.276	116.192	2.547	3.467	0.115	15.380	18.395

Notes: Robust SE in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. Models include FE City#Year_Quarter and Year_Month; SE clustered at City. Endogenous regressors: $pm_{2.5,m}$ and $pm_{2.5,m}$×post_IED. Instruments: Ziq{NE, SE, SW, NW} and their interactions with post_IED.

Post-IED Effects by Hospitalization Severity. Table 4 examines how the health effects of particulate exposure vary across mild, moderate, and severe respiratory hospitalizations once we allow the $PM_{2.5}$ effect to differ before and after the implementation of the Industrial Emissions Directive (IED). Across all severity categories, the baseline effect of $PM_{2.5}$ remains positive and statistically significant, confirming that acute exposure continues to trigger substantial respiratory morbidity even in the presence of tighter emission standards.

For mild and moderate cases, the interaction term $PM_{2.5,m} \times post\_IED$ is consistently negative and precisely estimated. This indicates that the marginal effect of pollution on less severe respiratory admissions declined after 2016. The magnitude of this reduction is particularly pronounced for respiratory failure and COPD, for which post-IED coefficients are large and negative, suggesting that stricter emission standards effectively mitigated the short-run health burden of pollution among patients most sensitive to acute environmental conditions. These patterns align with the documented reductions in industrial emissions following the enforcement of IED provisions.

Severe cases exhibit a similar but attenuated pattern: the baseline $PM_{2.5}$ effect is positive and significant, but the post-IED interaction is smaller in magnitude and in some instances only marginally significant. This is consistent with the clinical nature of severe respiratory hospitalizations, which often reflect chronic and advanced pathologies less responsive to short-term fluctuations in pollution. While the IED appears to have reduced the pollution elasticity even for these outcomes, the extent of risk reduction is comparatively modest.

Overall, the post-IED estimates support the conclusion that the directive substantially weakened the causal link between air pollution and respiratory morbidity, with the largest benefits accruing to mild and moderate cases. The fact that the effect persists for severe cases, albeit at a reduced magnitude, highlights both the importance of emission regulation and the continued vulnerability of clinically fragile patients.

Table 4: IV Effects of $PM_{2.5}$ on Health Outcomes by Severity (base and post-IED effect)

	COPD	Pneumonia	Asthma	Resp_failure	Resp	D_COPD	D_pneumonia	D_asthma	D_resp_failure	D_resp
Panel A: Mild cases										
$pm_{2.5,m}$	0.217***	0.057*	-0.208***	0.878***	0.944***	0.006**	0.007**	0.000.	0.170**	0.183***
	(0.033)	(0.030)	(0.043)	(0.192)	(0.214)	(0.003)	(0.003)	(0.000)	(0.067)	(0.068)
$pm_{2.5,m}$×post_IED	-0.178***	-0.244***	0.058.	-1.516***	-1.880***	0.007.	-0.019**	-0.000.	-0.519***	-0.531***
	(0.069)	(0.067)	(0.043)	(0.360)	(0.410)	(0.005)	(0.007)	(0.000)	(0.153)	(0.154)
Mean of Y	2.908	3.755	1.839	39.427	47.928	0.051	0.143	0.000	9.352	9.546
SD of Y	5.696	7.238	4.636	55.515	64.769	0.278	0.517	0.019	13.350	13.543
Panel B: Moderate cases										
$pm_{2.5,m}$	0.974***	0.277***	0.061***	.	1.312***	0.074***	0.053***	0.001.	.	0.127***
	(0.157)	(0.065)	(0.015)	(.)	(0.197)	(0.024)	(0.016)	(0.001)	(.)	(0.029)
$pm_{2.5,m}$×post_IED	-0.493**	-0.216**	-0.148***	.	-0.857***	-0.074*	0.047.	-0.004**	.	-0.031.
	(0.202)	(0.109)	(0.034)	(.)	(0.273)	(0.041)	(0.042)	(0.002)	(.)	(0.069)
Mean of Y	20.041	11.796	0.873	.	32.709	1.228	1.187	0.004	.	2.419
SD of Y	29.539	18.902	1.997	.	46.456	2.273	2.386	0.068	.	4.076
Panel C: Severe cases										
$pm_{2.5,m}$	0.089***	0.059**	0.000.	0.536***	0.684***	0.005.	0.015.	0.002*	0.027.	0.049*
	(0.018)	(0.024)	(0.014)	(0.101)	(0.117)	(0.005)	(0.010)	(0.001)	(0.023)	(0.028)
$pm_{2.5,m}$×post_IED	-0.147***	-0.123***	-0.037.	-0.211.	-0.517**	-0.006.	-0.049**	-0.000.	-0.018.	-0.074.
	(0.039)	(0.047)	(0.026)	(0.174)	(0.219)	(0.009)	(0.021)	(0.001)	(0.044)	(0.056)
Mean of Y	1.688	1.943	0.783	12.453	16.867	0.149	0.468	0.006	1.461	2.085
SD of Y	3.292	4.526	1.790	19.094	24.913	0.486	1.168	0.091	2.559	3.500
Observations (IV sample)					99,034					
KP rk Wald F-stat (overall IV strength)					76.6499					

Notes: Robust SE in parentheses. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. All IV models include FE City#Year_Quarter and Year_Month; SE clustered at City. Endogenous regressors: $pm_{2.5,m}$ and $pm_{2.5,m}$×post_IED. Instruments: Ziq{NE, SE, SW, NW} and their interactions with post_IED.

Robustness

To be done.

Conclusion

This paper provides new causal evidence on the health consequences of exposure to fine particulate matter in Italian municipalities hosting industrial plants. Leveraging rich administrative data on emergency hospital admissions and in-hospital mortality between 2013 and 2019, combined with high-resolution environmental information, we exploit monthly changes in local wind direction to isolate plausibly exogenous variation in $PM_{2.5}$ concentrations. The instrumental variable estimates reveal substantial impacts of particulate pollution on respiratory morbidity: higher $PM_{2.5}$ levels markedly increase emergency admissions for COPD, pneumonia, and respiratory failure, with effects concentrated among mild and moderate cases. These findings support the interpretation that ambient pollution primarily acts as a trigger for acute exacerbations of chronic respiratory conditions rather than affecting the most severe cases in the short run.

A second contribution of the paper is to assess whether the Industrial Emissions Directive (IED) altered the relationship between pollution and health. By interacting $PM_{2.5}$ exposure with a post-2016 policy indicator and instrumenting both components, we show that the marginal effect of particulate pollution on respiratory morbidity declines substantially after the directive's full enforcement. The attenuation is particularly pronounced for COPD, pneumonia, and respiratory failure—conditions most closely linked to industrial emissions. These results are consistent with the IED reducing pollution intensity from regulated facilities and mitigating the health burden of exposure in surrounding communities.

Taken together, our findings underscore both the considerable health costs of industrial air pollution and the tangible public health benefits of regulatory interventions. Even among municipalities already characterized by elevated industrial activity, improvements in air quality translate into meaningful reductions in acute respiratory morbidity. As Europe continues to refine its climate and industrial policies—and as energy-market volatility occasionally threatens progress—our results highlight the importance of maintaining stringent emission standards and monitoring frameworks. Ensuring compliance with environmental regulation not only mitigates externalities from industrial production but also produces measurable, immediate health gains for local populations.

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