

## THE IMPACT OF AIR POLLUTION ON MORTALITY RATES IN NIGERIA: A STATISTICAL PERSPECTIVE.

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### ABSTRACT

This study examined the association between air pollution indicators and mortality rates in Nigeria over the period 2000 - 2024, the data obtained were mainly secondary data sourced from the World Health Organization's Global Health Observatory and National Bureau of Statistics. In this paper, the independent variables are: Particulate matter concentrations (PM<sub>2.5</sub> and PM<sub>10</sub>), Nitrogen dioxide (NO<sub>2</sub>) concentrations, Sulfur dioxide (SO<sub>2</sub>) concentrations and Ozone (O<sub>3</sub>) concentrations where the response variable is Mortality rate. The study employed Multiple regression and correlation to investigate the influence of five air pollutants (PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, SO<sub>2</sub>, and O<sub>3</sub>) on mortality rates. From the regression models formed through Multiple Regression Analysis;  $Y = 16.109 - 0.050(\text{PM}_{2.5}) + 0.028(\text{PM}_{10}) - 0.083(\text{NO}_2) - 0.031(\text{SO}_2) + 0.028(\text{O}_3)$ . The negative coefficients observed for PM<sub>2.5</sub>, NO<sub>2</sub>, and SO<sub>2</sub> suggest that their annual mean concentrations were inversely related to mortality rates, whereas PM<sub>10</sub> and O<sub>3</sub> exhibited positive associations. The opposing signs for PM<sub>2.5</sub> and PM<sub>10</sub>, despite PM<sub>2.5</sub> being a component of PM<sub>10</sub>, further suggest model misspecification or omitted variable bias rather than divergent health effects. Similarly, the positive association observed for O<sub>3</sub> may be attributable to seasonal confounding with temperature or other unmeasured covariates. These results diverge from established epidemiological evidence and are best interpreted as exploratory, hypothesis-generating associations that likely reflect residual confounding and shared temporal patterns rather than causal exposure effects. Furthermore, diagnostic tests for multicollinearity, heteroskedasticity, and autocorrelation confirmed that the dataset was free from these statistical issues.

**Keywords:** Air pollution, heteroscedasticity, mortality, multicollinearity, pollutant.

### INTRODUCTION

Air pollution has become one of the foremost public health challenges of the 21st century, impacting billions globally and contributing substantially to the burden of disease and premature mortality. The atmospheric mixture of gases, particulate matter, and biological agents poses significant risks to human health, with consequences ranging from acute respiratory symptoms to chronic cardiovascular conditions and early death. In the context of rapid industrialization, urban expansion, and population growth, advancing knowledge on the health impacts of air pollution is essential for evidence-based policymaking and effective public health interventions.

Air pollution constitutes a critical global environmental challenge, posing severe risks to human health and ecosystem stability (Ndamitso et al., 2021). In Nigeria, this problem has intensified with rapid industrial expansion, accelerating urbanization, and population growth. The country's economic development and demographic pressures have contributed to increased industrial activity, elevated vehicular emissions, and inadequate waste management practices (Abulude et al., 2022; Abulude et al., 2023). These processes have released hazardous pollutants into the atmosphere including particulate matter, nitrogen oxides, sulfur dioxide, volatile organic

compounds, and carbon monoxide (WHO, 2021) with profound implications for human health, climate systems, and ecological integrity, underscoring the urgent need for intervention.

Air pollution levels in Nigeria have risen sharply, leading to serious consequences for public health, the environment, and overall living conditions (Pona et al., 2021). The problem is intensified by inadequate monitoring systems, weak enforcement of environmental regulations, and limited public awareness.

One of the main drivers of pollution is open burning such as the burning of agricultural waste, household refuse, and bush land which releases large amounts of smoke, particulate matter (PM), and toxic gases into the atmosphere (Ugwuanyi et al., 2018).

To fully grasp the health implications of air pollution in Nigeria, a comprehensive, multi-disciplinary approach is essential. This involves collaboration among environmental scientists, public health experts, epidemiologists, and policymakers to gather reliable data, conduct in-depth analyses, and design effective, evidence-based interventions aimed at reducing the health burden of air pollution (Manisalidis et al., 2020; Burns et al., 2020).

Nigeria's air quality varies significantly across different seasons, reflecting the nation's diverse climate patterns (Nelson, 2023). When humidity levels are low, the natural processes that help cleanse the atmosphere become less effective, leading to increased concentrations of pollutants (Zhao, 2022).

In the northeastern region, particularly Maiduguri, air quality is further degraded by desertification and recurring dust storms, which are especially common during the dry season (Mohammed et al., 2020).

## LITERATURE REVIEW

Extensive research across diverse regions and populations has established a strong link between air pollution and adverse health outcomes, with growing evidence showing its significant contribution to global morbidity and mortality. This review highlights recent studies that employ statistical techniques, data analysis methods, and empirical investigations to assess the health impacts of air pollution, emphasizing methodological frameworks, regression models, and quantitative findings that deepen our understanding of this pressing public health concern.

Iqbal et al. (2021) explored the connection between air pollution and COVID-19 in China using negative binomial regression with robust standard errors to evaluate the relationship between the Air Quality Index (AQI) and COVID-19 transmission. Their analysis revealed notable positive correlations: each one-unit increase in AQI corresponded to a 0.24% rise in COVID-19 cases and a 0.26% increase in mortality. The negative binomial model outperformed the Poisson regression by effectively addressing over dispersion in the count data. This study underscores how exposure to polluted air can intensify the effects of infectious diseases, thereby elevating population-level mortality risks.

Diener and Mudu (2021) carried out a critical review on the role of vegetation in shielding populations from air pollution, highlighting how green spaces can mitigate airborne particles from a public health standpoint. Their findings offer valuable guidance for urban planning, suggesting that integrating green infrastructure into rapidly growing Nigerian cities could provide a cost-effective means of reducing exposure to air pollution and its associated health risks.

Similarly, Xu, Yang, and Li (2022) developed a comprehensive theoretical framework to assess the economic burden of air pollution, employing econometric techniques such as the cost-of-illness approach and willingness-to-pay valuation. Their analysis showed that a 10  $\mu\text{g}/\text{m}^3$  rise in  $\text{PM}_{2.5}$  levels was linked to annual healthcare costs ranging between \$12 and \$45 per person, depending

on demographic and baseline health conditions. Using multiple regression models that controlled for socioeconomic factors, the study demonstrated that air pollution contributes not only to direct medical expenses but also to indirect costs through diminished productivity and reduced quality of life. This framework is particularly relevant for understanding the broader health and economic implications of air pollution in developing nations like Nigeria.

Recent research has thoroughly documented the biological mechanisms by which air pollution harms human health. Shetty et al. (2023) provided an extensive review of environmental pollutants, showing that exposure to air contaminants can provoke inflammation, oxidative stress, and cellular injury, all of which contribute to disease development. These pathways explain the strong association between air pollution and higher rates of illness and mortality.

The link between air pollution and premature death has been confirmed in numerous large-scale studies and meta-analyses. Lelieveld et al. (2023), for example, combined observational data with atmospheric modeling to estimate deaths attributable to fossil fuel emissions. Using the EMAC chemistry model alongside epidemiological concentration-response functions, they calculated that approximately 8.7 million premature deaths worldwide in 2018 (95% CI: 7.8–9.8 million) were caused by fossil fuel-related air pollution—equivalent to about 18% of all global deaths that year. Their use of Monte Carlo simulations to address uncertainty in the data provided strong statistical evidence that air pollution substantially elevates mortality risk across diverse populations and age groups.

Ofremu et al. (2025) examined how climate change and air pollution intersect with human health, focusing on the challenges of impacts, adaptation, and mitigation. Their study highlights the particular vulnerabilities of developing countries, emphasizing strategies that can help reduce health risks from pollution. This research is especially pertinent to Nigeria, as it addresses the overlap between environmental change and public health in comparable socioeconomic settings. Building on this foundation, our paper introduces a model designed to estimate the relationship between air pollution exposure and mortality rates in Nigeria.

### **OBJECTIVES OF THE STUDY**

This study aims to achieve the following objectives:

- i. examine the association between air pollution and mortality rates
- ii. quantify the extent to which air pollution contributes to mortality rates
- iii. evaluate the significance of air pollution's effect on mortality rates

### **RESEARCH QUESTIONS**

- i. Does an increase in air pollution correlate with higher mortality rates?
- ii. how significantly does air pollution contribute to changes in mortality rates?
- iii. Is the effect of air pollution on mortality rates statistically significant?

### **RESEARCH HYPOTHESIS**

#### **Hypothesis 1**

H<sub>0</sub>: There is no statistically significant association between air pollution levels and mortality rates in Nigeria.

H<sub>1</sub>: There is a statistically significant association between air pollution levels and mortality rates in Nigeria.

#### **Hypothesis 2**

H<sub>0</sub>: Air pollution does not have a significant effect on overall mortality rates in the Nigerian population.

H<sub>2</sub>: Air pollution has a significant effect on overall mortality rates in the Nigerian population, with higher pollution levels associated with increased mortality.

### Hypothesis 3

H<sub>0</sub>: Air pollution has no significant impact on mortality rates.

H<sub>1</sub>: Air pollution has a significant impact on mortality rates

## MATERIALS AND METHODS

This study examines the impact of air pollution on mortality rates across all 36 states and the Federal Capital Territory of Nigeria, covering the period from 2000 to 2024. Health outcome examined include overall mortality rates across different demographic categories. Confounding factors such as socioeconomic status, occupational exposure, smoking habits, and access to healthcare may influence the associations observed in this study. Furthermore, health outcome data are prone to underreporting, particularly in areas with limited medical infrastructure, while satellite-derived pollution estimates may vary in accuracy compared to ground-based measurements. The focus on Nigeria also limits the generalizability of the findings to other regions, and certain populations such as nomadic groups may be underrepresented in the available datasets.

This research is based predominantly on secondary data and employs a strategy aimed at assessing the influence of air pollution on mortality rates in Nigeria. Air quality statistics were obtained from the World Health Organization (WHO), while mortality figures were drawn from the National Bureau of Statistics (NBS). The dataset was subjected to comprehensive quantitative analysis to evaluate specific hypotheses and to explore linear associations among the variables. The methods applied included correlation analysis, multiple regression, and descriptive statistics. Covering a 25-year span from 2000 to 2024, the analysis was carried out using the Statistical Package for the Social Sciences (SPSS).

### MULTIPLE REGRESSION

The model used is expressed as:

$$Y = \beta_0 + \beta_1X_1 + \beta_2X_2 + \beta_3X_3 + \beta_4X_4 + \beta_5X_5 + \varepsilon$$

Where:

Y = Mortality rate (per 100,000) population

X<sub>1</sub>, X<sub>2</sub> = Particulate matter concentrations (PM<sub>2.5</sub> and PM<sub>10</sub>), measured in µg/m<sup>3</sup>, representing particles with aerodynamic diameters less than 2.5 µm and 10 µm, respectively

X<sub>3</sub> = Nitrogen dioxide (NO<sub>2</sub>) concentration, measured in µg/m<sup>3</sup>, reflecting traffic-related pollution and combustion processes

X<sub>4</sub> = Sulfur dioxide (SO<sub>2</sub>) concentration, measured in µg/m<sup>3</sup>, indicating emissions from industrial activity and fossil fuel combustion

X<sub>5</sub> = Ozone (O<sub>3</sub>) concentration, measured as the maximum 8-hour average in µg/m<sup>3</sup>, representing secondary pollutant formation via photochemical reactions

ε<sub>i</sub> = Error term, assumed to be independently and identically distributed with zero mean and constant variance

### ANALYSIS OF VARIANCE IN MULTIPLE REGRESSION

Recall the model again;

$$Y = \beta_0 + \beta_1X_1 + \beta_2X_2 + \beta_3X_3 + \beta_4X_4 + \beta_5X_5 + \varepsilon$$

**Table 1: ANOVA TABLE**

Source of variation	SS	Df	MS	F-statistic
Regression	$SSR = \sum_{i=1}^n (\hat{Y}_i - \bar{Y})^2$	P	$MSR = \frac{SSR}{p}$	$\frac{MSR}{MSE}$
Error	$SSE = \sum (Y_i - \hat{Y}_i)^2$	n - p - 1	$MSE = \frac{SSE}{n-p-1}$	
Total	$SST = \sum (Y_i - \bar{Y})^2$	n-1		

Where n is the number of observations and p is the number of independent variables.

**F - test for regression;**

$$H_0 : \beta_1 = \beta_2 = \dots = \beta_p = 0$$

$$H_1 : \text{At least one } \beta_p \neq 0$$

Test statistic:

$$F = \frac{MSR}{MSE} = \frac{SSR/p}{SSE/(n-p-1)}$$

Decision Rule:

If  $F_{\text{calculated}} > F_{\text{critical}}$ , reject  $H_0$  otherwise accept  $H_0$

**R<sup>2</sup> and the adjusted R<sup>2</sup>**

SSR = SST - SSE is the part of variation explained by regression model

Thus, Coefficient of Determination:

$$R^2 = \frac{SSR}{SST} = 1 - \frac{SSE}{SST}$$

Which is the proportion of variation in the response that can be explained by the regression model (or that can be explained by the predictors  $X_1, X_2, X_3, \dots, X_p$  linearly)

$$0 \leq R^2 \leq 1$$

With more predictor variables, SSE is smaller and  $R^2$  is larger. To evaluate the contribution of the predictors, we define the adjusted  $R^2$

$$R_a^2 = 1 - \frac{SSE}{SST} = 1 - \left(\frac{n-1}{n-p-1}\right) \frac{SSE}{SST}$$

**Correlation Analysis**

Correlation analysis is employed to examine the strength and direction of bivariate relationships between air pollution variables and mortality rates, providing initial evidence for the research hypotheses. The correlation analysis includes examination of correlations between air pollution variables and control variables to identify potential confounding relationships. Correlation matrices are constructed to provide comprehensive overviews of all bivariate relationships, facilitating identification of multicollinearity issues that may affect regression analyses.

$$r = \frac{\text{Covariance}(X,Y)}{\sqrt{\text{Variance}(X)\text{Variance}(Y)}}$$

$$r = \frac{S_{xy}}{\sqrt{S_{xx} \cdot S_{yy}}}$$

the covariance of XY is

$$S_{xy} = \sum XY - \frac{\sum X \sum Y}{n}$$

$$S_{xx} = \sum x^2 - \frac{(\sum X)^2}{n}$$

$$V(y) = \sum y^2 - \frac{(\sum Y)^2}{n}$$

The above equation can be written as;

$$r = \frac{\sum XY - \frac{\sum X \sum Y}{n}}{\sqrt{(\sum x^2 - \frac{(\sum X)^2}{n})(\sum y^2 - \frac{(\sum Y)^2}{n})}}$$

**RESULTS OF ANALYSIS**

**Data Presentation**

**Table 2:** Air Pollution Indicators and Mortality Rates in Nigeria (2000-2024)

Year	PM <sub>2.5</sub> (µg/m <sup>3</sup> )	PM <sub>10</sub> (µg/m <sup>3</sup> )	NO <sub>2</sub> (µg/m <sup>3</sup> )	SO <sub>2</sub> (µg/m <sup>3</sup> )	O <sub>3</sub> (µg/m <sup>3</sup> )	Mortality Rates
2000	17.79	72.30	14.60	12.40	48.70	17.84
2001	24.20	68.40	20.30	10.80	45.20	17.66
2002	12.39	81.50	29.70	15.60	52.30	17.48
2003	7.98	85.20	22.40	17.20	55.80	17.30
2004	20.57	76.80	26.50	13.90	50.40	16.86
2005	11.45	70.20	27.80	11.50	47.60	16.43
2006	10.13	65.90	29.40	19.70	43.80	15.99
2007	17.82	78.60	37.30	24.20	51.20	15.56
2008	21.38	67.50	34.50	10.30	46.40	15.12
2009	28.39	83.70	36.20	16.80	54.30	14.80
2010	23.34	63.80	38.60	18.90	42.50	14.48
2011	13.16	87.90	38.80	18.40	57.20	14.16
2012	25.70	62.40	37.90	18.20	41.70	13.83
2013	25.01	82.10	40.50	16.10	53.60	13.51
2014	19.97	18.60	42.70	24.50	68.40	13.20
2015	35.55	24.60	45.40	13.10	79.80	12.89
2016	45.67	89.30	34.60	19.20	58.50	12.58
2017	49.86	56.70	45.30	26.80	88.90	12.27
2018	54.30	32.40	45.80	26.70	71.30	11.97
2019	63.70	38.50	52.60	32.40	78.60	11.77
2020	37.40	41.80	23.70	22.20	48.10	11.58
2021	49.80	58.90	56.40	27.50	50.20	11.38
2022	55.60	69.30	52.10	21.20	46.90	11.19
2023	68.90	73.60	55.80	23.50	70.70	11.74
2024	89.30	82.80	62.40	38.50	85.20	10.83

Source: World Health Organization (WHO), National Bureau of Statistics (NBS).

**Table3:** Descriptive Statistics of Air Pollution Indicators and Mortality Rates (2000-2024)

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
PM <sub>2.5</sub>	25	7.98	89.30	33.1744	21.19319
PM <sub>10</sub>	25	18.60	89.30	65.3120	19.86409
NO <sub>2</sub>	25	14.60	62.40	38.0520	12.20704
SO <sub>2</sub>	25	10.30	38.50	19.9840	6.93347
O <sub>3</sub>	25	41.70	88.90	57.4920	13.98228
Mortality rates	25	10.83	17.84	14.0968	2.28722

The table 3 above is the descriptive Statistics which shows the mean value of mortality rates and air pollution levels (PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, SO<sub>2</sub>, and O<sub>3</sub>).

**Table 4:** Model Formation

Coefficients								
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
		B	Std. Error	Beta			Tolerance	VIF
	(Constant)	16.109	1.683		9.574	.000		
	PM <sub>2.5</sub>	-.050	.020	-.467	-2.566	.019	.292	3.425
	PM <sub>10</sub>	.028	.012	.242	2.278	.034	.859	1.164
	NO <sub>2</sub>	-.083	.032	-.442	-2.571	.019	.327	3.056
	SO <sub>2</sub>	-.031	.054	-.093	-5.72	.574	.362	2.766
	O <sub>3</sub>	.028	.023	.171	1.224	.236	.495	2.020

Dependent Variable: Mortality rates

$$Y = 16.109 - 0.050(\text{PM}_{2.5}) + 0.028(\text{PM}_{10}) - 0.083(\text{NO}_2) - 0.031(\text{SO}_2) + 0.028(\text{O}_3) + \epsilon_i$$

Table 4 above shows that PM<sub>2.5</sub> (β = -0.467, p = .019), NO<sub>2</sub> (β = -0.442, p = .019), and SO<sub>2</sub> (β = -0.093, p = 0.574) are negatively associated with mortality rates. In contrast, PM<sub>10</sub> (β = 0.242, p = 0.034) and O<sub>3</sub> (β = 0.171, p = 0.236) display positive associations. These findings suggest that higher annual mean concentrations of PM<sub>2.5</sub>, NO<sub>2</sub>, and SO<sub>2</sub> correspond to lower mortality rates within the study period, whereas increases in PM<sub>10</sub> and O<sub>3</sub> are linked to higher mortality rates. The table presents corresponding standard errors and 95% confidence intervals. Additionally, variance inflation factor (VIF) values demonstrate low to moderate collinearity.

**Table 5:** Model Summary for Overall Mortality Rates Prediction

Model Summary						
Model	R	R Square	Adjusted Square	R	Std. Error of the Estimate	Durbin-Watson
1.	.904	.816	.768		1.10158	1.979
a. Predictors: (Constant), O <sub>3</sub> , PM <sub>10</sub> , NO <sub>2</sub> , SO <sub>2</sub> , PM <sub>2.5</sub>						
b. Dependent Variable: Mortality rates						

The model exhibits considerable predictive strength, with the five air pollution variables accounting for 81.6% of the variance in mortality rates (R<sup>2</sup> = 0.816) and an adjusted R<sup>2</sup> of 0.768, which continues to reflect substantial explanatory capacity.

**Table 6:** ANOVA TABLE

ANOVA						
Model		Sum of Squares	df	Mean Square	F	Sig.
	Regression	102.497	5	20.499	16.893	.000 <sup>b</sup>
	Residual	23.056	19	1.213		
	Total	125.554	24			
a. Dependent Variable: Mortality rates						
b. Predictors: (Constant), O <sub>3</sub> , PM <sub>10</sub> , NO <sub>2</sub> , SO <sub>2</sub> , PM <sub>2.5</sub>						

The ANOVA analysis demonstrates that the model is highly significant (F = 16.893, p < .001), showing that air pollution variables collectively serve as strong predictors of mortality rates.

**Table 7:** Correlation analysis of air pollution indicators and mortality rates

	Mortality rates	PM <sub>2.5</sub>	PM <sub>10</sub>	NO <sub>2</sub>	SO <sub>2</sub>	O <sub>3</sub>
Mortality rates	1	-.817**	.383	-.830**	-.721**	-.537**
PM <sub>2.5</sub>	-.817**	1	-.177	.786**	.753**	.643**
PM <sub>10</sub>	.383	-.177	1	-.224	-.224	-.359
NO <sub>2</sub>	-.830**	.786**	-.224	1	.738**	.597**
SO <sub>2</sub>	-.721**	.753**	-.224	.738**	1	.612**
O <sub>3</sub>	-.537**	.643**	-.359	.597**	.612**	1

\*\* . Correlation is significant at the 0.01 level (2-tailed).

The regression analysis reveals that PM<sub>2.5</sub> ( $\beta = -0.467$ , p = .019) and NO<sub>2</sub> ( $\beta = -0.442$ , p = .019) are significant predictors of mortality, both showing negative associations consistent with temporal trends. By contrast, PM<sub>10</sub> demonstrates a significant positive relationship ( $\beta = 0.242$ , p = .034), indicating that higher PM<sub>10</sub> concentrations correspond with increased mortality. Meanwhile, SO<sub>2</sub> and O<sub>3</sub> do not present as statistically significant predictors in the model.

**ASSUMPTION TEST**

**Hypotheses:**

- H<sub>0</sub>: Residuals are normally distributed
- H<sub>1</sub>: Residuals are not normally distributed

**Table 8:** Normality Test Results (Jarque-Bera Test)

Model	Test Statistic	P-value	Decision
Death rate	1.842	0.398	Fail to reject H <sub>0</sub>

The Jarque-Bera test confirms that the model’s residuals follow a normal distribution (p > 0.05) satisfying the normality assumption. This result supports the application of parametric statistical methods and ensures that the regression estimates remain both unbiased and efficient.

**TEST OF MULTICOLLINEARITY, AUTOCORRELATION, AND HETEROSCEDASTICITY.**

**Hypothesis:**

H<sub>0</sub>: ε<sub>t</sub>'s are homoscedastic

H<sub>1</sub>: ε<sub>t</sub>'s are heteroscedastic

**Table 9:** Heteroscedasticity Test Results (Breusch-Pagan Test)

Model	Chi-square	P-value	Decision
Death rate	2.871	0.238	Fail to reject H <sub>0</sub>

The test results reveal no signs of heteroscedasticity in the model ( $p > 0.05$ ), confirming that the constant variance assumption holds. Consequently, the regression coefficients' standard errors are reliable, and the associated hypothesis tests remain valid.

**TEST FOR AUTOCORRELATION**

**Hypotheses:**

H<sub>0</sub>: ρ = 0 (no autocorrelation)

H<sub>1</sub>: ρ ≠ 0 (autocorrelation exists)

**Table 10:** Detection of autocorrelation

Model	Durbin-Watson Statistic	Decision
Death rate	1.920	No autocorrelation

Note: Critical values at 5% significance level with k = 2 and n = 25: dL = 1.288, dU = 1.454

From the model above, Durbin-Watson (d) =  $\frac{\sum(U_t - U_{t-1})^2}{\sum U_t^2} = 1.920 \approx 2$ .

Since d ≈ 2, suggesting no significant first-order autocorrelation in the residuals.

**TEST FOR Multicollinearity**

**Hypotheses:**

H<sub>0</sub>: Multicollinearity does not exist

H<sub>1</sub>: Multicollinearity exist

**Table 11:** Multicollinearity Test Results (VIF and Tolerance)

	Model: Death rate	
	Tolerance	VIF
PM <sub>2.5</sub>	0.292	3.425
PM <sub>10</sub>	0.859	1.164
NO <sub>2</sub>	0.327	3.056
SO <sub>2</sub>	0.362	2.766
O <sub>3</sub>	0.495	2.02

**Variance Inflating Factor (VIF):** If the VIF values less than 5, there is no multicollinearity, whereas values above 10 indicate the presence of serious multicollinearity. Since the VIF values for this model fall within the acceptable range, it can be concluded that the independent variables are not affected by multicollinearity.

**LIMITATIONS**

A principal limitation of this study is the reliance on annual aggregate data, which precludes determination of temporal ordering between exposure and mortality and fails to capture short-term

variations or seasonal confounding. In addition, the ecological design, restricted sample size, and potential unmeasured confounders limit the capacity for causal inference. Accordingly, we refrain from advancing policy recommendations and interpret the findings solely as descriptive associations.

## DISCUSSION

Using annual aggregate data, we estimated associations between yearly mean air pollutant concentrations and mortality rates. The fitted model was:  $Y = 16.109 - 0.050(\text{PM}_{2.5}) + 0.028(\text{PM}_{10}) - 0.083(\text{NO}_2) - 0.031(\text{SO}_2) + 0.028(\text{O}_3)$ . The inverse association for  $\text{PM}_{2.5}$ ,  $\text{NO}_2$ , and  $\text{SO}_2$  are inconsistent with the broader toxicological and epidemiological evidence linking these pollutants to increased mortality risk. Several limitations of the annual aggregate design likely contribute to these findings such as, temporal aggregation to calendar years precludes assessment of acute and lagged effects of air pollution on mortality rates.

Also, with annual observations, the model has limited statistical power and is vulnerable to spurious correlation arising from shared temporal trends: both pollutant concentrations and mortality rates declined over the study period, and regression cannot distinguish causal effects from common trends driven by unobserved factors.

Lastly, the ecological design cannot account for individual-level exposure-response relationships and is subject to residual confounding by time-varying socioeconomic, healthcare and behavioral factors that were not included in the model.

The negative signs for  $\text{PM}_{2.5}$  and  $\text{PM}_{10}$  despite  $\text{PM}_{2.5}$  being a component of  $\text{PM}_{10}$  likely indicate model misspecification or omitted variable bias rather than distinct health effects. The positive association with  $\text{O}_3$  may similarly reflect seasonal confounding with temperature or other unmeasured factors.

Given these limitations, the associations should be considered exploratory and hypothesis-generating. The findings highlight the inadequacy of annual aggregate data for air pollution, mortality research and emphasize the need for analyses with finer temporal resolution, appropriate lag structures, and adjustment for key confounders. Confirmation using disaggregated data and causal designs is required before drawing policy implications.

The Jarque–Bera test confirms that the residuals are normally distributed. The Breusch–Pagan test shows no evidence of heteroscedasticity, validating the assumption of constant variance and the reliability of the estimated standard errors. The Durbin–Watson statistic falls within acceptable bounds, indicating no autocorrelation among residuals. Furthermore, the Variance Inflation Factor (VIF) values remain within acceptable limits, suggesting that multicollinearity is not a concern among the explanatory variables.

## CONCLUSION

This analysis of annual aggregate data found inverse associations between mortality rates and mean concentrations of  $\text{PM}_{2.5}$ ,  $\text{NO}_2$ , and  $\text{SO}_2$ , while  $\text{PM}_{10}$  and  $\text{O}_3$  were positively associated. However, the reliance on annual data, limited sample size, and lack of adjustment for socioeconomic and healthcare confounders preclude causal interpretation. The results diverge from established epidemiological evidence and are best regarded as exploratory, hypothesis-generating associations that likely reflect residual confounding and shared temporal trends rather than true exposure effects. We hypothesize that the observed inverse associations are artifacts of

unmeasured socioeconomic factors and aggregation bias, which should be tested in future research using monthly or daily individual-level data.

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