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Associations between ambient air pollution and mortality during a 7-year ecological time-series study

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Abstract

Introduction

Air pollution remains a major public health concern, particularly in large metropolitan areas. This study aimed to evaluate the short-term association between ambient air pollution levels and non-accidental mortality in Istanbul between 2013 and 2019.

Material and Methods

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In this ecological time-series study, daily mortality data were analyzed using quasi-Poisson regression models incorporating distributed lag non-linear models (DLNM) to estimate exposure–response relationships. Citywide daily mean concentrations of major air pollutants were derived from all eligible monitoring stations. Effect estimates were expressed as relative risks (RRs) with 95% confidence intervals (CIs) per 10 μg/m³ increase in particulate matter concentrations.

Results: During the 7-year period, average particulate matter levels frequently exceeded World Health Organization (WHO) guideline values. A 10 μg/m³ increase in PM_{10} was associated with a 1.7% increase in daily mortality (RR: 1.017; 95% CI: 1.009–1.025), while a similar increase in $PM_{2.5}$ was associated with a 3.3% increase (RR: 1.033; 95% CI: 1.021–1.046). Mortality increases were more pronounced among individuals aged ≥65 years. Periods of consecutive high pollution were also associated with excess mortality.

Conclusion: Short-term increases in particulate matter concentrations were associated with elevated mortality in Istanbul, particularly among older adults. These results support the relevance of WHO guideline values in the local context and highlight the importance of sustained air quality control policies.

Keywords: air pollution, health effects, particulate matter

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Introduction

Air pollution is one of the leading environmental risk factors for global morbidity and mortality, contributing substantially to the global burden of disease. It is estimated that approximately 7 million premature deaths annually are attributable to ambient and household air pollution exposure.[1] Despite international policy efforts, only a small proportion of the global population lives in areas that meet the World Health Organization (WHO) air quality guidelines.[1] The health burden of air pollution disproportionately affects low- and middle-income countries, where rapid urbanization and industrialization often outpace environmental control measures.

Air pollution originates from multiple sources, including residential heating, traffic emissions, industrial activity, and power generation. More than 200 emission sources have been identified globally.[2] Effective control strategies require region-specific monitoring of pollutant levels and identification of

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dominant emission sources. Although extensive epidemiological evidence from Europe and North America has demonstrated associations between short-term particulate matter exposure and mortality, evidence from developing countries remains comparatively limited. Regional studies are therefore essential to inform locally appropriate environmental and public health policies.

In Turkey, rapid population growth, industrial expansion, and increased motor vehicle use have intensified concerns regarding urban air quality. Istanbul, the most populous city in the country, accounts for approximately 20–25% of national air pollutant emissions. Major emission sources in Istanbul include traffic, domestic heating, industrial activities, and energy production.[3] In addition, the city is affected by regional pollutant transport due to its geographic location and intense air and maritime traffic.

Previous research evaluating air pollution trends in Istanbul between 2003 and 2013 reported a gradual decline in pollutant concentrations.[4] However, despite this downward trend, particulate matter levels frequently remain above WHO guideline values. There is limited recent evidence evaluating the short-term relationship between ambient air pollution and mortality in Istanbul using contemporary monitoring data and advanced time-series methods.

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Therefore, the aim of this study was to evaluate the association between daily air pollutant concentrations and non-accidental mortality in Istanbul between 2013 and 2019 using an ecological time-series approach. By providing updated local evidence, this study seeks to contribute context-specific results to support air quality management and public health policy in Turkey.

Material and Methods

Design:

This ecological time-series study evaluated the short-term association between daily ambient air pollution levels and non-accidental mortality in Istanbul between January 1, 2013, and December 31, 2019. Daily mortality data were obtained from the Turkish Statistical Institute (TURKSTAT) and included all registered non-accidental deaths occurring in Istanbul during the study period. Accidental deaths were excluded. Population data were derived from official annual statistics to calculate crude mortality rates.[5] Air

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pollution data was obtained from 37 stations where measurements were taken during the study period by the National Air Quality Monitoring Network and the Istanbul Metropolitan Municipality Air Quality Monitoring Center. Meteorological data, including daily mean temperature, relative humidity, and wind speed, were obtained from the National Meteorological Service.[6,7] Hourly measurements from all active fixed monitoring stations were aggregated into daily averages for each pollutant. In accordance with European Environment Agency (EEA) data completeness criteria, only stations with valid measurements for at least 75% of days within a given year were included. Citywide daily pollutant concentrations were calculated as the arithmetic mean of daily values across all eligible stations. This approach is widely used in ecological time-series studies evaluating short-term air pollution effects.[2]

Statistical Analyses:

Two complementary analytical approaches were used in this study. First, a time-series analysis using distributed lag non-linear models (DLNM) was conducted to estimate the association between daily pollutant concentrations and mortality across the full study period (Tables 2–3; Figures 1–4). Second, an episode-based analysis was performed to evaluate mortality during periods of consecutive high pollution levels (Tables 4–5).

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Daily non-accidental mortality counts were analyzed using quasi-Poisson regression models to account for overdispersion in daily count data within a time-series framework. To simultaneously estimate non-linear exposure–response relationships and delayed effects, distributed lag non-linear models (DLNM) were implemented using a cross-basis function for each pollutant, allowing flexible modeling of both the concentration–response association and the lag structure. Lag effects were examined over a 10-day period (lag 0–10 days), and the lag–response function was modeled using natural cubic splines.

The degrees of freedom for spline terms, including lag structures and meteorological variables, were selected during model development based on Akaike Information Criterion (AIC), deviance statistics, and residual diagnostics. Meteorological variables (daily mean temperature, relative humidity, and wind speed) were included as continuous predictors and modeled using natural cubic spline functions with three degrees of freedom to account for potential non-linear confounding effects. Long-term and

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seasonal trends were controlled using a natural cubic spline of calendar time with seven degrees of freedom per year. Four sequential models were constructed, and the final model included the pollutant cross-basis term, adjustment for calendar time, categorical terms for day of the week and day of the year, and meteorological variables incorporated as smooth functions. Effect estimates are presented as relative risks (RRs) with 95% confidence intervals (CIs), corresponding to a 10 $\mu g/m^3$ increase in pollutant concentrations. For graphical presentation, cumulative exposure–response curves were derived from the cross-basis function and centered at the concentration associated with minimum predicted mortality risk.

In addition to the DLNM time-series analysis, an episode-based analysis was conducted to evaluate mortality during short periods of elevated air pollution. High-pollution episodes were defined as periods during which at least two pollutants simultaneously exceeded their 90th percentile concentrations for three consecutive days. A lag period of three days was considered to capture potential delayed mortality effects following exposure. Reference periods were defined as calendar-matched days from other study years during which pollutant concentrations did not exceed the 90th percentile threshold. High-pollution episodes were defined as periods during which at least two pollutants simultaneously exceeded their pollutant-specific 90th percentile concentrations for a minimum of three consecutive days. Episodes could therefore extend beyond three days if elevated concentrations persisted. This approach was used to minimize confounding related to seasonal variation in mortality and environmental conditions. For each episode, the crude mortality rate (CMR) was calculated by dividing the number of deaths by the product of the population size of the corresponding year and the number of days in the episode period. The CMR during high-pollution episodes was compared with the CMR of the reference period using a Z-test. The relative risk (RR) was calculated as the ratio of the CMR during the episode period to the CMR of the reference period and ninety-five percent confidence intervals (95% CI) were presented. Excess deaths attributable to high-pollution episodes were estimated by multiplying the difference between crude mortality rates by the population size and the number of days during which pollutant levels remained elevated. Spearman correlation analysis was used because the relationships between pollutant levels did not meet the assumptions of normal distribution.

Results

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Between 2013 and 2019, the daily average number of deaths in Istanbul ranged from 168 to 207, corresponding to daily mortality rates between 1.19 and 1.34 per 100,000 population. Mortality exhibited clear seasonal variation, with higher rates during winter months and lower rates during summer across all study years. During the study period, the overall mean (±SD) daily concentration was 49.2 ± 23.6 μg/m³ for PM_{10} and 24.4 ± 11.5 μg/m³ for $PM_{2.5}$. Particulate matter levels were generally lower during summer months. Annual and seasonal pollutant distributions are presented in Table 1 and supplementary materials.

In the fully adjusted model, increases in particulate matter concentrations were associated with a significant increase in daily mortality rates. A 10 μg/m³ increase in PM_{10} was associated with a 1.7% increase in daily mortality (RR: 1.017; 95% CI: 1.009–1.025). Similarly, a 10 μg/m³ increase in $PM_{2.5}$ was associated with a 3.3% increase in daily mortality (RR: 1.033; 95% CI: 1.021–1.046).

When alternative centering values were applied (WHO guideline values, national regulatory limits, and the 90th percentile of observed concentrations), effect estimates remained positive and statistically significant, although slightly attenuated at higher reference values (Tables 2 and 3). Exposure–response curves demonstrated a non-linear pattern, with increasing mortality risk at higher pollutant concentrations (Figures 3 and 4).

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Between 2013 and 2019, several episodes were identified during which at least two pollutants exceeded their 90th percentile concentrations for three or more consecutive days. In 2014, a 9-day episode was associated with a mortality rate ratio of 1.07 (95% CI: 1.02–1.12), corresponding to an estimated 129 excess deaths compared with calendar-matched reference periods. Similar increases were observed during episodes in 2016 (RR: 1.07; 95% CI: 1.00–1.13) and 2017 (RR: 1.11; 95% CI: 1.04–1.18). Among individuals aged ≥65 years, excess mortality during high-pollution episodes was more pronounced. (Table 4, Table 5 and supplementary materials).

Spearman correlation analysis demonstrated strong positive correlations between PM_{10} and $PM_{2.5}$ ($r = 0.795$, $p < 0.01$) and between PM_{10} and NO_2 ($r = 0.736$, $p < 0.01$). O_3 showed negative correlations with several pollutants. Correlations between pollutants and mortality counts are presented in Tables 6 and 7.

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Discussion

This ecological time-series study evaluated the short-term association between ambient air pollution and non-accidental mortality in Istanbul over a seven-year period. The findings indicate that increases in particulate matter concentrations, particularly $PM_{2.5}$ and PM_{10}, were significantly associated with higher daily mortality, with stronger effects observed among individuals aged 65 years and older. In the fully adjusted model, a 10 μg/m³ increase in PM10 was associated with a 1.7% increase in daily mortality, while the corresponding increase for PM2.5 was 3.3%. These effect estimates are broadly consistent with results reported in large multicity studies from Europe, North America, Latin America, and Asia.[9,10,11] Previous meta-analyses and systematic reviews have reported mortality increases ranging from approximately 0.5% to 2% per 10 μg/m³ increase in particulate matter concentrations.[13,15] Differences in baseline pollution levels, particle composition, and population vulnerability may partly explain variations in effect magnitude across regions. Minimum predicted mortality risk concentrations derived from the DLNM curves were 40 μg/m³ for PM10 and 17 μg/m³ for PM2.5. These values were close to the WHO 24-hour guideline values of 45 μg/m³ for PM10 and 15 μg/m³ for PM2.5, respectively. However, they should be interpreted cautiously as model-derived estimates rather than definitive safe thresholds.

In our study, associations were stronger for $PM_{2.5}$ compared with PM_{10}, which is biologically plausible. Fine particulate matter penetrates deeper into the respiratory tract and may trigger systemic inflammation, oxidative stress, and cardiovascular dysfunction.[16,17] Similar findings have been reported in prior epidemiological studies, particularly among older adults and individuals with pre-existing cardiopulmonary conditions.[12,14]

Exposure–response curves demonstrated a non-linear pattern. However, the apparent flattening or reversal of risk at very low concentrations should be interpreted cautiously. Confidence intervals were wide in these ranges, suggesting limited precision and possible residual confounding rather than a true protective effect.

The episode-based analysis further demonstrated increased mortality during periods of consecutive high pollution. These episodes predominantly occurred during winter and early spring, which is consistent with seasonal increases in residential heating and unfavorable meteorological conditions

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limiting pollutant dispersion. Previous studies conducted in Istanbul have similarly reported seasonal variation in pollutant concentrations.[4] Not all high-pollution episodes were associated with increased mortality, suggesting that episode duration, pollutant mixture, meteorological conditions, baseline vulnerability, and random variation may influence the observed mortality response.

Air pollution episodes in Turkey are sometimes compared with the “Polish smog” phenomenon, which is largely driven by residential coal combustion and characterized by high wintertime concentrations of fine particulate matter.[18,19] In contrast, Istanbul’s pollution mixture reflects a combination of traffic emissions, industrial activity, residential heating, and regional transport.[20,21] Differences in emission sources and physicochemical composition may influence toxicity profiles and should be considered when interpreting and generalizing results.

Despite a gradual decline in average pollutant concentrations over the study period, particulate matter levels frequently exceeded WHO guideline values. The minimum mortality risk concentrations identified in our analysis were close to WHO recommended thresholds. While these findings are consistent with WHO guideline values, the ecological observational design does not permit causal inference. Rather than establishing proof, this study provides locally relevant evidence supporting the public health importance of maintaining particulate matter concentrations below internationally recommended limits.[1] Several pollutants showed moderate to strong correlations, particularly between PM10 and PM2.5. Because of this collinearity, pollutants were analyzed in single-pollutant models. Although this approach is widely used in air pollution time-series studies, it may limit the ability to fully disentangle the independent effects of correlated pollutants. Strong correlations between PM10, PM2.5, and NO_2 indicate that the independent effects of individual pollutants cannot be fully disentangled. Therefore, the single-pollutant estimates should be interpreted as pollutant-specific associations within a correlated urban pollution mixture rather than isolated causal effects.

This study has several strengths, including the use of multiple monitoring stations, a long follow-up period, and advanced time-series modeling incorporating non-linear and lagged effects. However, certain limitations should be acknowledged. The analysis relied on aggregated mortality data and did not include cause-specific mortality. Potential residual confounding,

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including unmeasured infectious disease outbreaks, cannot be excluded. Additionally, citywide average pollutant concentrations may not fully capture individual-level exposure variability. Seasonal population mobility may influence mortality patterns in large metropolitan areas such as Istanbul. For example, temporary decreases in the resident population during summer holiday periods or short-term population increases related to tourism may affect daily mortality rates. Because annual population estimates were used to calculate mortality rates in this study, short-term seasonal fluctuations in population size could not be fully accounted for. However, this limitation is unlikely to substantially influence the relative risk estimates derived from the time-series models.

Overall, this study provides context-specific confirmation of the established association between short-term particulate matter exposure and increased mortality. These findings highlight the continued need for sustained air quality control policies and targeted protection of vulnerable populations in large metropolitan settings.

Conclusion

Short-term increases in particulate matter concentrations were associated with elevated non-accidental mortality in Istanbul, with stronger effects observed among individuals aged 65 years and older. $PM_{2.5}$ demonstrated a greater relative impact on mortality compared with PM_{10}. Minimum mortality risk concentrations identified in this study were close to World Health Organization guideline values, supporting their relevance in the local context. However, given the ecological design, these findings should be interpreted as supportive evidence rather than proof of causality. The observed increase in mortality during consecutive high-pollution episodes further highlights the need for sustained air quality monitoring and timely public health interventions, particularly for vulnerable populations in large metropolitan areas.

Ethics approval

The study was approved by the Ethics Committee of Istanbul University-Cerrahpasa, Cerrahpasa Medical Faculty (Approval number and date: 827645-06.11.2023). As this study was based on aggregated, publicly available air quality and mortality data, informed consent was not required. The study was conducted in accordance with the Declaration of Helsinki, and

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it was reported according to Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement.

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Data Availability Statements

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors report that no funding has been received for the study.

Authors' contributions

SNA and GC designed the study. SNA collated the data. SNA, MY and GC performed the statistical analysis. SNA and GC interpreted the results. SNA wrote the manuscript. SNA and MY created the tables and figures. SNA, MY and GC revised the manuscript. GC supervised the whole study. All authors read and approved the final manuscript.

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Table 1. Daily Average Air Pollution Parameter Levels in Istanbul Between 2013-2019 years

Parameters	2013	2014	2015	2016	2017	2018	2019	Average of 2013-2019	
								Mean (± Standard Deviation)	Median (25-75.percentile)
PM_{10}	57.8(±27.1)	54.1(±22.3)	52.7(±26.9)	48.1(±24.9)	51.6(±21.4)	44.6(±19.1)	40.5(±17.5)	49.2(±23.6)	44.1(34.3-59.6)
CO	629.2 (±263.0)	541.1 (±231.6)	562.0 (±225.6)	612.1 (±204.7)	509.2 (±173.2)	526.2 (±163.0)	551.7 (±161.3)	573.2(±209.2)	513.3(435.5-648.8)
NO	38.4(±25.4)	45.8(±40.8)	43.1(±36.2)	40.8(±31.2)	41.3(±28.3)	36.5(±28.8)	35.9(±32.3)	40.3(±32.4)	29.4(20.5-48.7)
NO_2	53.9(±15.6)	50.2(±14.4)	48.6(±14.4)	46.0(±14.5)	48.4(±14.9)	41.7(±14.7)	44.2(±14.4)	47.6(±15.2)	44.7(36.2-57.4)
NOX	106.1(±36.6)	110.6(±52.1)	100.3(±59.8)	94.8(±53.6)	95.2(±45.0)	84.4(±50.7)	97.9(±62.7)	98.4(±52.7)	87.6(59.5-124.4)
O_3	38.5(±19.2)	36.1(±13.3)	34.4(±11.8)	33.8(±12.0)	34.4(±13.3)	37.8(±14.2)	36.7(±13.6)	36.0(±14.2)	36.0(25.4-46.3)
$PM_{2.5}$	25.1(±8.1)	24.7(±9.2)	24.3(±10.4)	31.2(±15.7)	25.4(±11.9)	20.4(±10.0)	19.6(±9.9)	24.4(±11.5)	21.4(17.0-28.6)
SO_2	4.4(±2.7)	4.4(±2.4)	5.0(±2.9)	5.9(±3.3)	5.5(±2.4)	3.6(±1.8)	4.4(±1.9)	4.7(±2.6)	3.9(2.9-5.7)

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Table 2. Relationship Between Number of Deaths Attributed to a 10 µg/m3 Increase in PM_{10} Levels

Limit Values Determined for PM_{10} Levels	Attributable Deaths RR (95% CI)
World Health Organization (45 µg/m³)	1.017 (1.009-1.025)
Turkey legislation (50 µg/m³)	1.017 (1.010-1.025)
90th percentile (80.7 µg/m³)	1.013 (1.008-1.018)
Minimum RR point (40 µg/m³)	1.012 (1.005-1.018)

*RR: Relative risk

Table 3. Relationship Between Number of Deaths Attributed to a 10 µg/m3 Increase in $PM_{2.5}$ Levels

Limit Values Determined for $PM_{2.5}$ Levels	Attributable Deaths RR (95% CI)
World Health Organization (15 µg/m³ (24 hours)	1.033(1.021-1.046)
90th percentile (39 µg/m³)	1.018 (1.008-1.028)
Minimum RR point (17 µg/m³)	1.012(1.005-1.018)

*RR: Relative risk

Table 4. Evaluation of the Relationship between Days with Consecutive Increases in Air Pollution Parameters and Number of Deaths

DATE	NUMBER OF DAYS WITH INCREASE	INCREASED POLLUTANTS	NUMBER OF DEATHS	CMD (X 100000)	NUMBER OF DEATHS IN REFERENCE PERIOD	CMD IN REFERENCE PERIOD (X 100,000)	RR (%95 CI)	p VALUE
10.01.2014-18.01.2014	9	PM_{10}, CO	2126	1,64	12397	1,54	1,07 (1,02-1,12)	**0,006**
25.11.2016-30.11.2016	6	PM_{10}, CO, $PM_{2.5}$, NO, NOX	1254	1,41	7062	1,32	1,07 (1,00-1,13)	**0,03**
10.10.2017-15.10.2017	6	PM_{10} , NO_2	1176	1,30	6281	1,18	1,11 (1,04-1,18)	**0,001**

*Crude Mortality Rate (CMD)

The full set of identified high-pollution episodes, including non-significant and inverse associations, is provided in the supplementary material. Tables 4 summarize episodes associated with statistically significant excess mortality and should therefore be interpreted together with the supplementary results.

Table 5. Evaluation of the Relationship between the Days with Consecutive Increases in Air Pollution Parameters and the Number of Deaths in People 65 Years and Older

DATE	NUMBER OF DAYS WITH INCREASE	INCREASED POLLUTANTS	NUMBER OF DEATHS	CMD (X 100000)	NUMBER OF DEATHS IN REFERENCE PERIOD	CMD IN REFERENCE PERIOD (X 100,000)	RR (%95 CI)	p VALUE
10.01.2014-18.01.2014	9	PM_{10}, CO	1404	1.09	8135	1.01	1.07 (1.01-1.14)	**0,014**

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10.10.2017-15.10.2017	6	PM_{10}, NO_2	715	0.79	3812	0.72	1.11 (1.02-1.20)	**0,012**
24.04.2018-29.04.2018	6	PM_{10}, NO_2	834	0.91	4310	0.81	1.12 (1.04-1.21)	**0,002**

*Crude Mortality Rate (CMD)

The full set of identified high-pollution episodes, including non-significant and inverse associations, is provided in the supplementary material. Tables 5 summarize episodes associated with statistically significant excess mortality and should therefore be interpreted together with the supplementary results.

Table 6. Evaluation of Correlation Between Air Pollution Parameters in Istanbul

	PM_{10}	**CO**	**NO**	**NO_2**	**NOX**	**O_3**	**$PM_{2.5}$**
PM_{10}							
CO	.606**						
NO	.585**	.693**					
NO_2	.736**	.692**	.786**				
NOX	.593**	.609**	.814**	.781**			
O_3	-.344**	-.679**	-.763**	-.547**	-.524**		
$PM_{2.5}$	.795**	.675**	.631**	.699**	.605**	-.462**	
SO_2	.535**	.710**	.656**	.686**	.560**	-.590**	.591**

* Spearman Correlation (<0.25 very weak relationship; 0.26-0.49 weak relationship; 0.50-0.69 moderate relationship; 0.70-0.89 high relationship; 0.90-1.0 very high relationship) ** p<0.01 * p<0.05

Table 7. Relationship Between Air Pollution Parameters and Number of Deaths

	PM_{10}	**CO**	**NO**	**NO2**	**NOX**	**O3**	**$PM_{2.5}$**	**SO_2**
Total Mortality	-.014	.276**	.226**	.097**	.083**	-.386**	.102**	.387**
Adult Mortality	-.029	.277**	.216**	.076**	.069**	-.382**	.081**	.364**
Infant Mortality	.087**	.005	.058**	.102**	.064**	-.013	.102**	.106**
Over 65 Mortality	-.033	.274**	.224**	.083**	.074**	-.386**	.068**	.356**

* Spearman Correlation (<0.25 very weak relationship; 0.26-0.49 weak relationship; 0.50-0.69 moderate relationship; 0.70-0.89 high relationship; 0.90-1.0 very high relationship) ** p<0.01 * p<0.05

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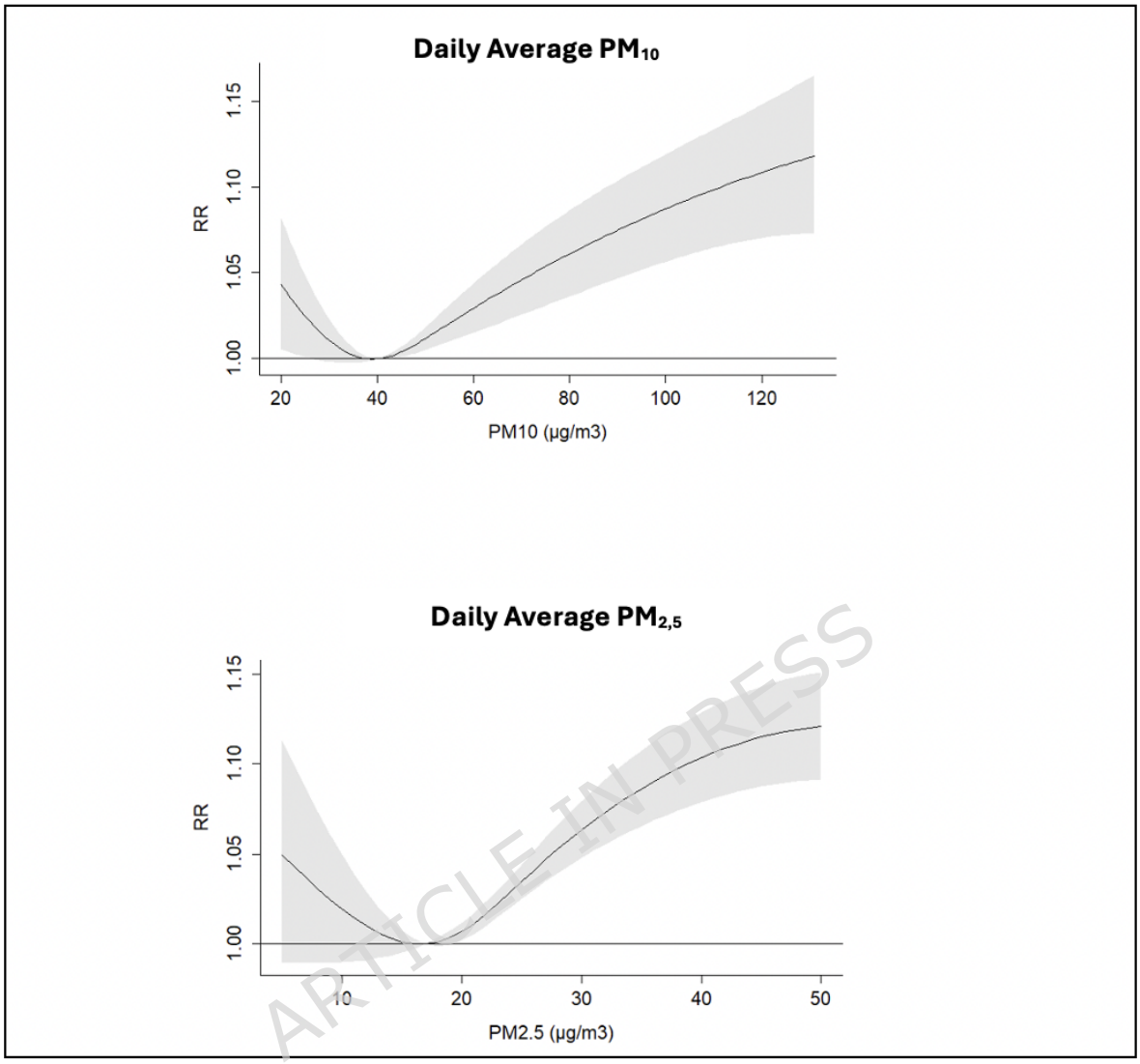


Figure 1. Relationship between Particulate Matter concentrations and relative risk of mortality

* The curves represent relative risks of mortality estimated from quasi-Poisson regression models. Shaded areas indicate 95% confidence intervals.

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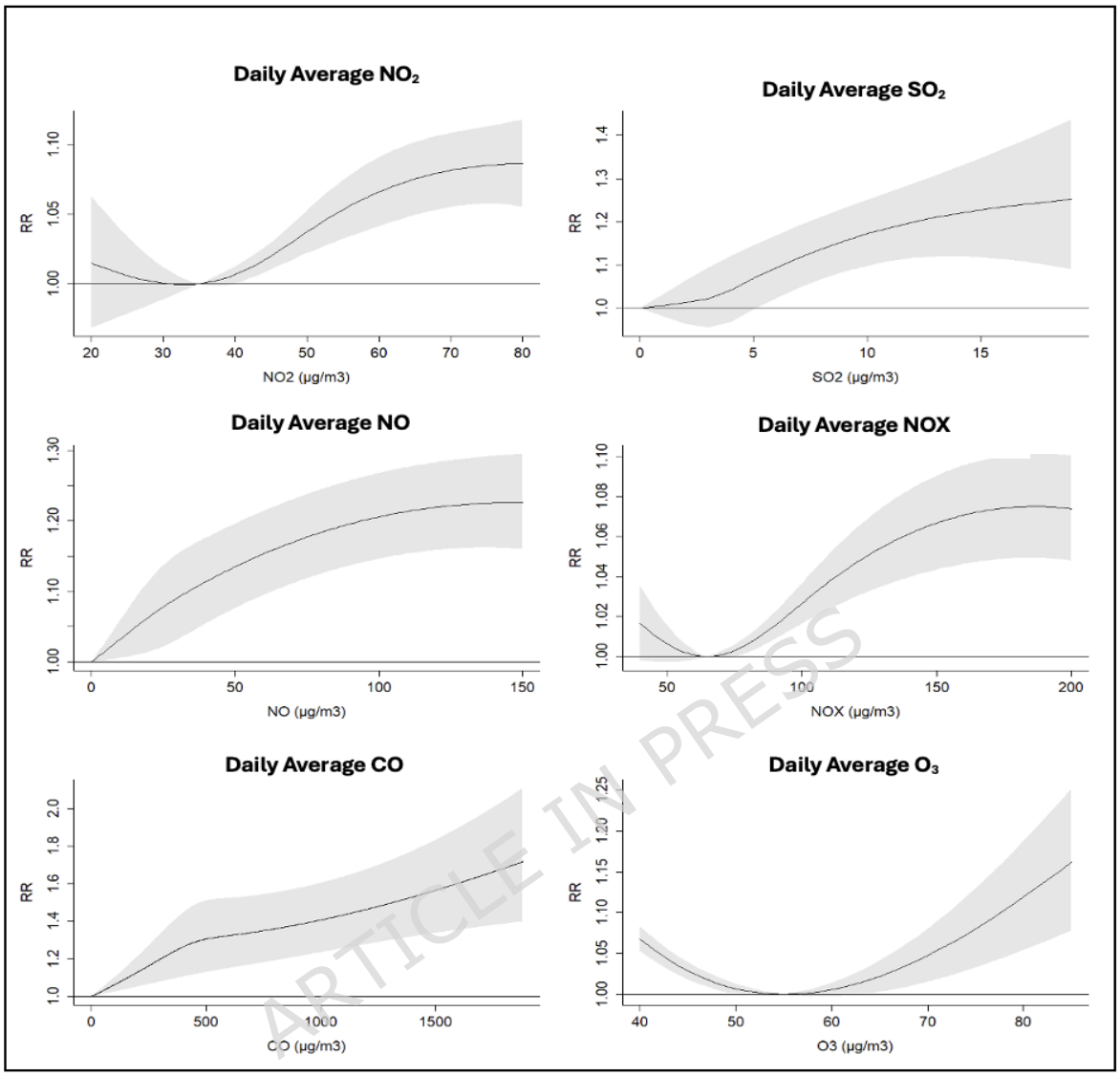


Figure 2. Relationship between pollutant concentrations and relative risk of mortality

*The curves represent relative risks of mortality estimated from quasi-Poisson regression models. Shaded areas indicate 95% confidence intervals.

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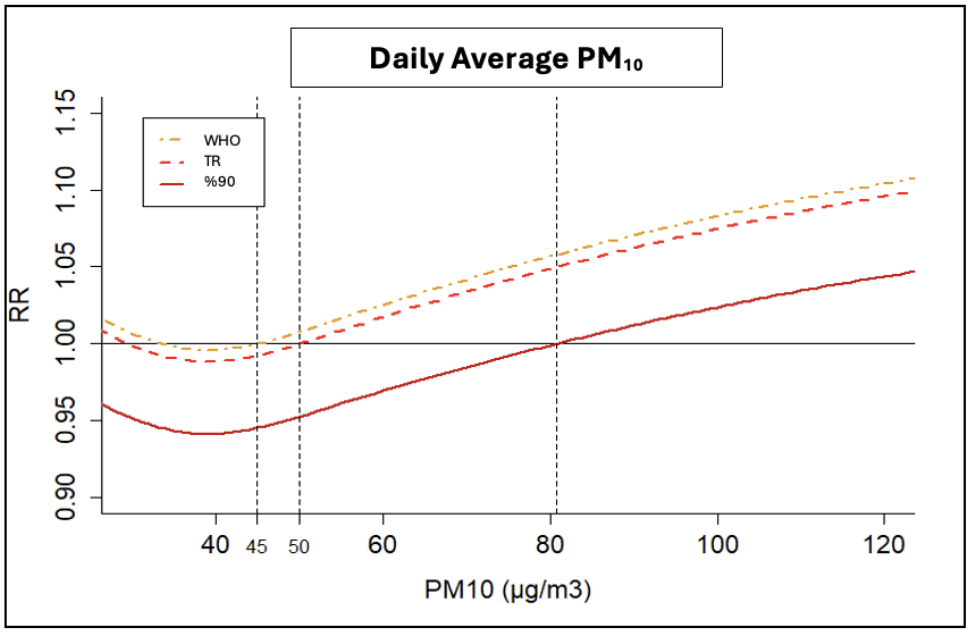


Figure 3. Exposure-response relationship between PM_{10} concentration and relative risk of mortality

* Solid curve represents the estimated cumulative relative risk derived from the fully adjusted quasi-Poisson DLNM model, with shaded areas indicating 95% confidence intervals. Vertical dashed lines represent the World Health Organization (WHO) 24-hour guideline value (45 µg/m³), the national regulatory limit (50 µg/m³), the 90th percentile of observed concentrations (80.7 µg/m³), and the concentration associated with minimum predicted mortality risk.

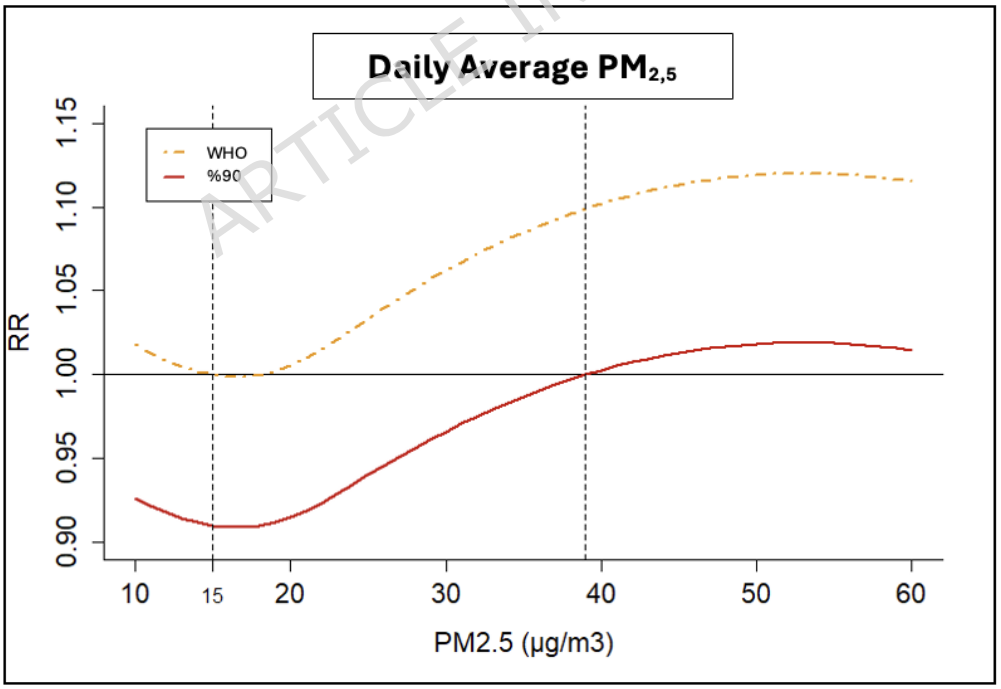


Figure 4. Exposure-response relationship between $PM_{2.5}$ concentration and relative risk of mortality

*Solid curve represents the estimated cumulative relative risk derived from the fully adjusted quasi-Poisson DLNM model, with shaded areas indicating 95% confidence intervals. Vertical dashed lines represent the World Health Organization (WHO) 24-hour guideline value (15 µg/m³), the 90th percentile

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of observed concentrations (39 µg/m³), and the concentration associated with minimum predicted mortality risk.

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Table 3. Relationship Between Number of Deaths Attributed to a 10 µg/m3 Increase in $PM_{2.5}$ Levels
Table 4. Evaluation of the Relationship between Days with Consecutive Increases in Air Pollution Parameters and Number of Deaths
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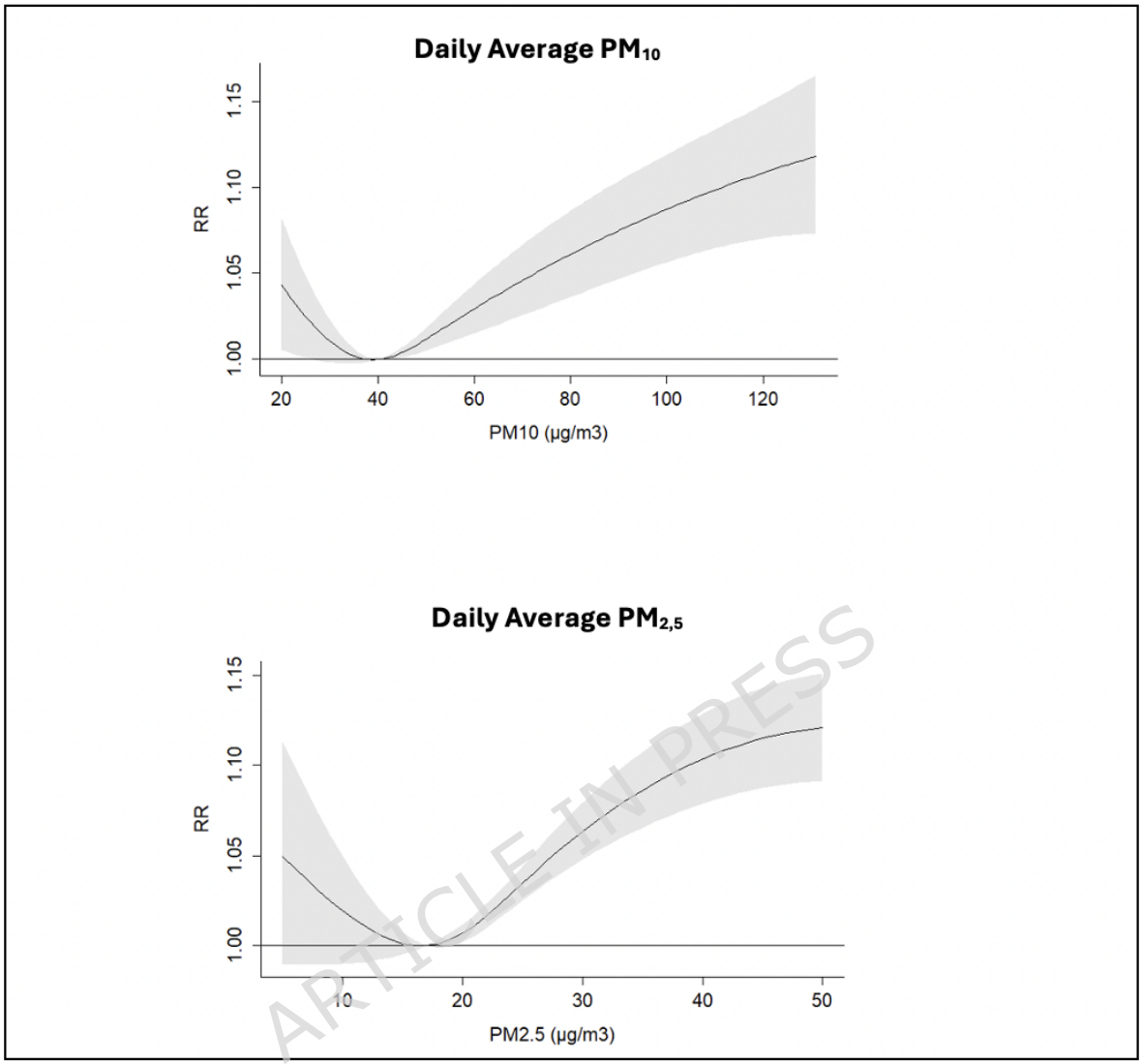


Figure 1. Relationship between levels of Particulate Matter pollutants and number of deaths

*The lines in the graphs show the impact of each pollutant level on the number of deaths, while the gray areas around these lines represent the 95% confidence interval of the relative risk

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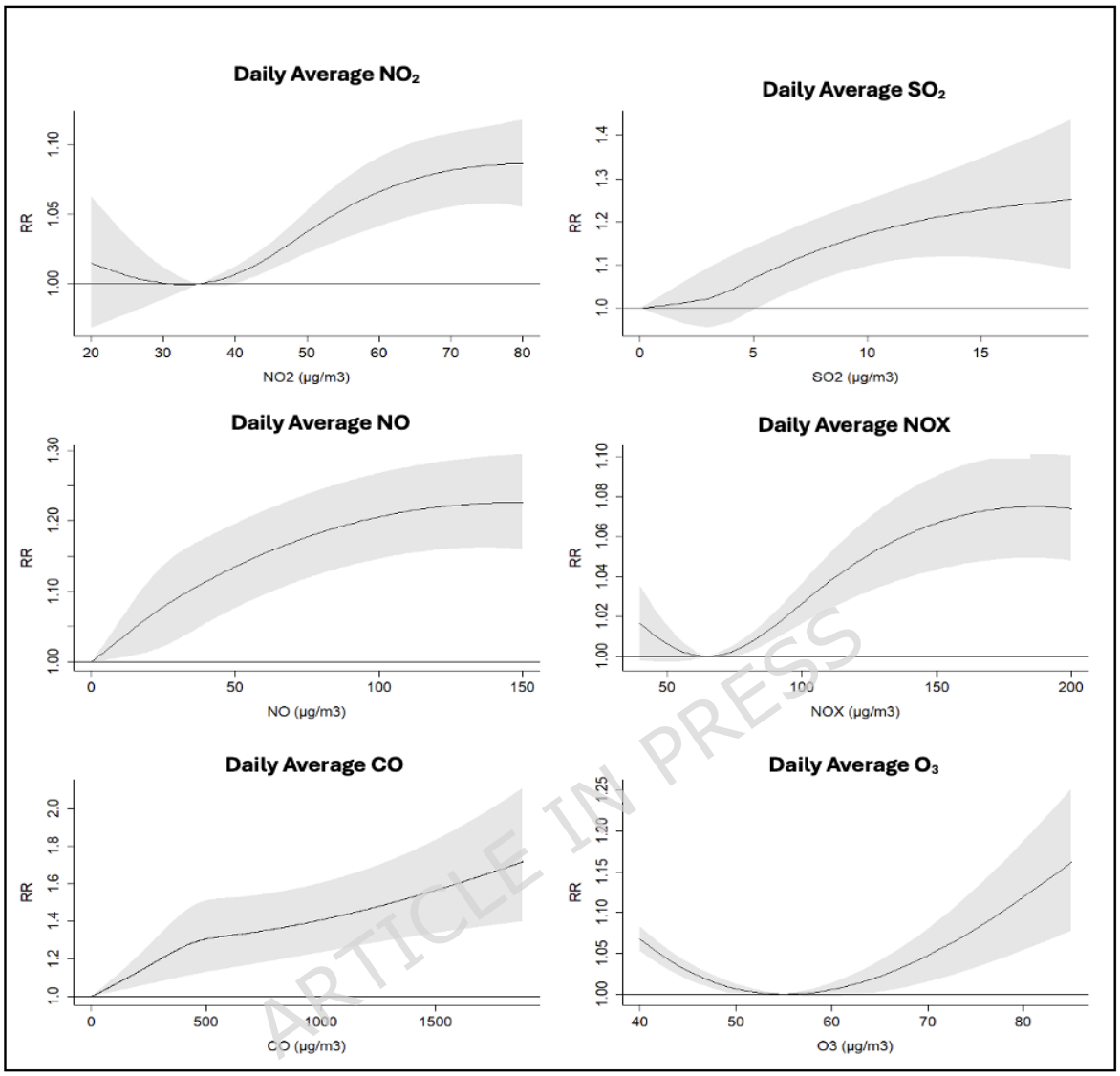


Figure 2. Relationship between Levels of Pollutants and Number of Deaths
*The lines in the graphs show the impact of each pollutant level on the number of deaths, while the gray areas around these lines represent the 95% confidence interval of the relative risk

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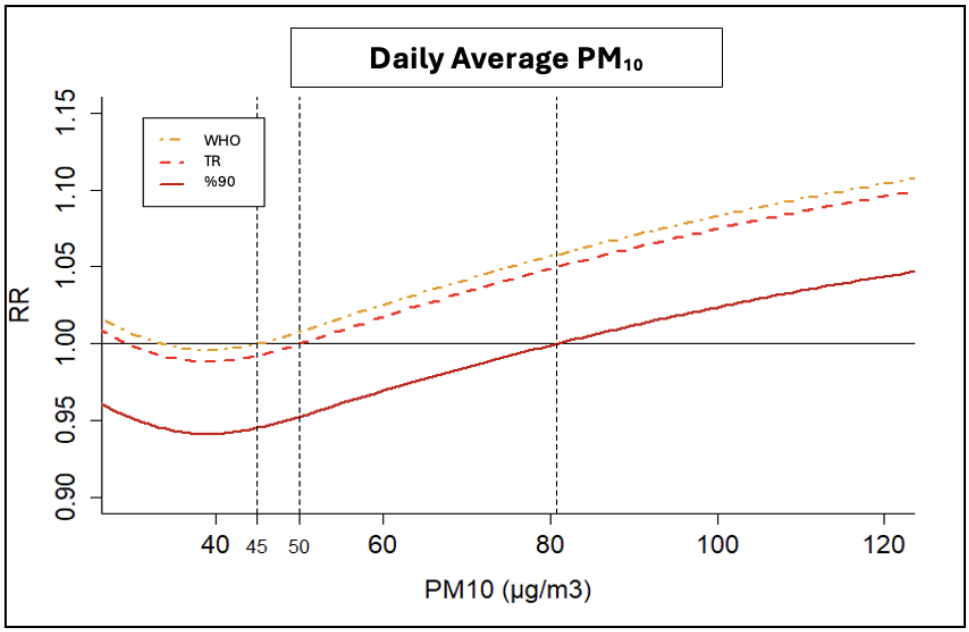


Figure 3. The Relationship between Increase in PM_{10} Levels and Number of Deaths

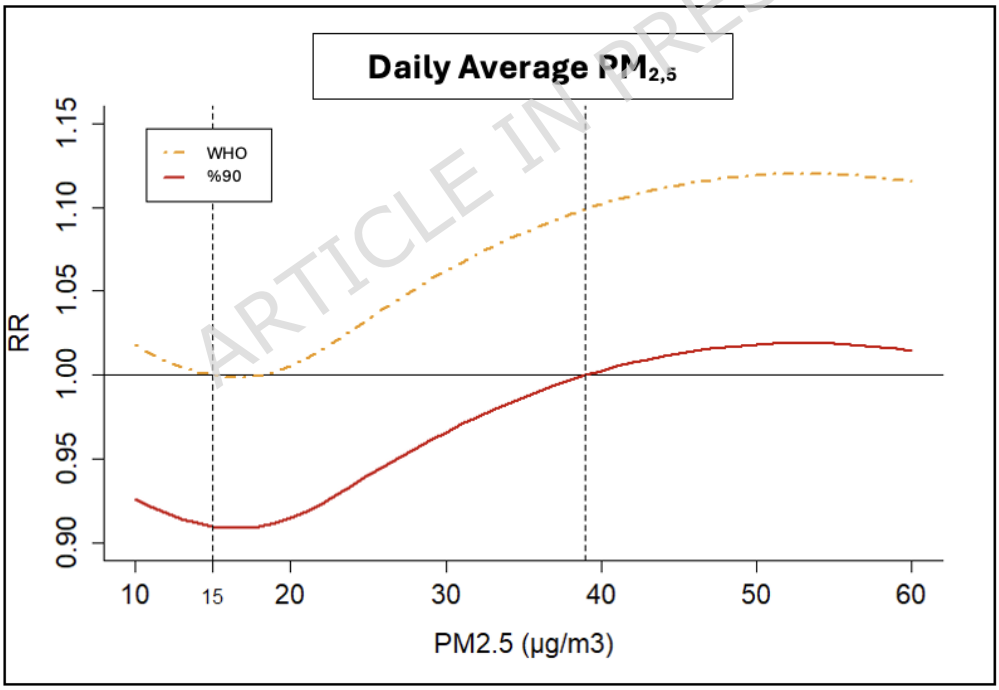


Figure 4. The Relationship between Increase in $PM_{2,5}$ Levels and Number of Deaths

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Table 1. Daily Average Air Pollution Parameter Levels in Istanbul Between 2013-2019 years

Parameters	2013	2014	2015	2016	2017	2018	2019	Average of 2013-2019	
								Mean (± Standard Deviation)	Median (25-75.percentile)
PM_{10}	57.8(±27.1)	54.1(±22.3)	52.7(±26.9)	48.1(±24.9)	51.6(±21.4)	44.6(±19.1)	40.5(±17.5)	49.2(±23.6)	44.1(34.3-59.6)
CO	629.2 (±263.0)	541.1 (±231.6)	562.0 (±225.6)	612.1 (±204.7)	509.2 (±173.2)	526.2 (±163.0)	551.7 (±161.3)	573.2(*±209.2)*	513.3(435.5-648.8)
NO	38.4(±25.4)	45.8(±40.8)	43.1(±36.2)	40.8(±31.2)	41.3(±28.3)	36.5(±28.8)	35.9(±32.3)	40.3(*±*32.4)	29.4(20.5-48.7)
NO_2	53.9(±15.6)	50.2(±14.4)	48.6(±14.4)	46.0(±14.5)	48.4(±14.9)	41.7(±14.7)	44.2(±14.4)	47.6(*±15.2*)	44.7(36.2-57.4)
NOX	106.1(±36.6)	110.6(±52.1)	100.3(±59.8)	94.8(±53.6)	95.2(±45.0)	84.4(±50.7)	97.9(±62.7)	98.4(*±52.7*)	87.6(59.5-124.4)
O_3	38.5(±19.2)	36.1(±13.3)	34.4(±11.8)	33.8(±12.0)	34.4(±13.3)	37.3(±14.2)	36.7(±13.6)	36.0(±14.2*)*	36.0(25.4-46.3)
$PM_{2,5}$	25.1(±8.1)	24.7(±9.2)	24.3(±10.4)	31.2(±15.7)	25.4(±11.9)	20.4(±10.0)	19.6(±9.9)	24.4*(*±11.5)	21.4(17.0-28.6)
SO_2	4.4(±2.7)	4.4(±2.4)	5.0(±2.9)	5.9(±3.3)	5.5(±2.4)	3.6(±1.8)	4.4(±1.9)	4.7(*±2.*6)	3.9(2.9-5.7)

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Table 2. Relationship Between Number of Deaths Attributed to a 10 µg/m3 Increase in PM_{10} Levels

Limit Values Determined for PM_{10} Levels	Attributable Deaths RR (95% CI)
World Health Organization (45 µg/m³)	1.017 (1.009-1.025)
Turkey legislation (50 µg/m³)	1.017 (1.010-1.025)
90th percentile (80.7 µg/m³)	1.013 (1.008-1.018)
Minimum RR point (40 µg/m³)	1.012 (1.005-1.018)

*RR: Relative risk

Table 3. Relationship Between Number of Deaths Attributed to a 10 µg/m3 Increase in $PM_{2,5}$ Levels

Limit Values Determined for $PM_{2,5}$ Levels	Attributable Deaths RR (95% CI)
World Health Organization (15 µg/m³ (24 hours)	1.033(1.021-1.046)
90th percentile (39 µg/m³)	1.018 (1.008-1.028)

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Minimum RR point (17 μg/m³)	1.012(1.005-1.018)

*RR: Relative risk

Table 4. Evaluation of the Relationship between Days with Consecutive Increases in Air Pollution Parameters and Number of Deaths

DATE	NUMBER OF DAYS WITH INCREASE	INCREASED POLLUTANTS	NUMBER OF DEATHS	CMD (X 100000)	NUMBER OF DEATHS IN REFERENCE PERIOD	CMD IN REFERENCE PERIOD (X 100000)	RR (%95 CI)	p VALUE
10.01.2014-18.01.2014	9	$PM_{10,}$ CO	2126	1,64	12397	1,54	1,07 (1,02-1,12)	**0,006**
25.11.2016-30.11.2016	6	$PM_{10,}$ CO, $PM_{2,5}$, NO, NOX	1254	1,41	7062	1,32	1,07 (1,00-1,13)	**0,03**
10.10.2017-15.10.2017	6	PM_{10} , NO_2	1176	1,30	6281	1,18	1,11 (1,04-1,18)	**0,001**

*Crude Mortality Rate (CMD)

Table 5. Evaluation of the Relationship between the Days with Consecutive Increases in Air Pollution Parameters and the Number of Deaths in People 65 Years and Older

DATE	NUMBER OF DAYS WITH INCREASE	INCREASED POLLUTANTS	NUMBER OF DEATHS	CRD (X 100000)	NUMBER OF DEATHS IN REFERENCE PERIOD	CRD IN REFERENCE PERIOD (X 100000)	RR (%95 CI)	p VALUE
10.01.2014-18.01.2014	9	$PM_{10,}$,CO	1404	1,09	8135	1,01	1,07 (1,01-1,14)	**0,014**
10.10.2017-15.10.2017	6	PM_{10} , NO_2	715	0,79	3812	0,72	1,11 (1,02-1,20)	**0,012**
24.04.2018-29.04.2018	6	PM_{10} , NO_2	834	0,91	4310	0,81	1,12 (1,04-1,21)	**0,002**

*Crude Mortality Rate (CMD)

Table 6. Evaluation of Correlation Between Air Pollution Parameters in Istanbul

	PM_{10}	**CO**	**NO**	**NO_2**	**NOX**	**O_3**	**$PM_{2,5}$**
PM_{10}							
CO	.606**						
NO	.585**	.693**					
NO_2	.736**	.692**	.786**				
NOX	.593**	.609**	.814**	.781**			
O_3	-.344**	-.679**	-.763**	-.547**	-.524**		
$PM_{2,5}$	.795**	.675**	.631**	.699**	.605**	-.462**	
SO_2	.535**	.710**	.656**	.686**	.560**	-.590**	.591**

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