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Air pollution and stroke mortality in Arak, Iran: Short-term and long-term exposure effects analyzed using zero-inflated negative binomial

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ABSTRACT

Introduction: Air pollution poses significant public health risks in industrial regions, with stroke mortality emerging as a critical outcome. This study examines the association between air pollutant exposure and stroke mortality in Arak, Iran - an industrial city with consistently poor air quality exceeding WHO thresholds.

Materials and methods: We conducted a time-series analysis of 1,010 stroke deaths (2019-2022) using zero-inflated negative binomial regression to model over-dispersed mortality data. Pollutant concentrations ($PM_{2.5}$, PM_{10}, NO_2, O_3, SO_2) were collected from four monitoring stations representing industrial, traffic, and residential zones. Effects were assessed for short-term (1-3 months) and long-term (6-24 months) exposures, with adjustment for meteorological and demographic confounders.

Results: NO_2 demonstrated the strongest short-term association (2-month RR: 1.50, 95% CI: 1.30-1.75, $p<0.001$). PM_{10} showed a slight increase in risk at the 2-month lag (RR: 1.06, 95% CI: 0.98–1.14), although it was not statistically significant. Long-term $PM_{2.5}$ exposure significantly increased mortality risk (24-month RR: 1.20, 95% CI: 1.05-1.58). A possible inverse association was observed for SO_2 (2-month RR: 0.59, 95% CI: 0.36–0.97), while O_3 effects varied over time.

Conclusion: Industrial emissions (particularly NO_2 and particulate matter) significantly contribute to stroke mortality in Arak. The identified exposure–response relationships highlight the importance of stricter emission controls on vehicular and industrial sources and targeted health interventions for high-risk populations. Further investigation of pollutant interactions is also essential to better understand their combined effects on stroke mortality.

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Introduction

Air pollution is a critical public health concern, especially in urban environments where industrial activities, vehicular emissions, and other anthropogenic factors contribute to the deterioration of air quality. Of all the health issues identified with air pollution, one that has received considerable attention in epidemiological research is its relationship with Cerebrovascular Disease (CVD) and stroke. Stroke is still a significant contributor to morbidity and mortality worldwide, with the need for investigation into its environmental risk factor variables within the arena of public health interventions [1]. The series of pathways regarding how air pollutants affect health outcomes regarding stroke is recognized as acute cardiovascular events, including stroke, which have been associated with short-term exposure to high levels of Particulate Matter (PM), NO_2, and other pollutants [2–4].

It has also been shown that acute peaks in air pollution are associated with immediate physiological responses, such as elevated blood pressure and vascular inflammation, which might lead to stroke [5–8]. However, chronic diseases, such as hypertension and atherosclerosis, as well-established risk factors for stroke, have more clearly been linked with long-term exposure to air pollution. Long-term exposure to accumulated air pollutants can exacerbate pre-existing medical problems and hence increase the risk of death from stroke [9]. However, translating these global findings into regional contexts is essential because local environmental, demographic, and socioeconomic factors modulate the health impacts of air pollution.

Iran holds a special place regarding atmospheric pollution, specifically due to industry and traffic, and does not have geographical characteristics that would dissipate pollutants well [10]. Several studies have shown upward trends in stroke mortality potentially linked to environmental factors [11–13]. For example, researchers investigated particle number size distributions in Tehran and found substantial differences between high-traffic and low-traffic areas, emphasizing the role of vehicular emissions in shaping urban air quality [14] . Similarly, other researchers examined the impact of Urmia Lake's declining water level on particulate matter concentrations in Tabriz and demonstrated significant health effects associated with changing PM trends [15]. Together, these regionally focused studies underscore the diversity and intensity of air pollution challenges across Iran, reinforcing the need for city-specific investigations such as the present study in Arak.

Arak as an industrial city situated in Markazi Province, central Iran, has complex air pollution problems. Recent researches indicate that $PM_{2.5}$ and PM_{10} concentrations in Arak often surpass the guidelines recommended by the World Health Organization (WHO). In one study conducted in 2020-2021, the annual average of $PM_{2.5}$ was reported to be 25.34 μg/m³, which is over 2.5 times higher than the WHO guideline value of 10 μg/m³ [16]. More recently, a study conducted from 2020 to 2022 documented another epidemiological finding: with increased $PM_{2.5}$, the rates of respiratory and cardiovascular admissions in hospitals increase significantly [17]. These recent findings pinpoint the persistence and complexity of air pollution in Arak, advising that continuous research, monitoring, and implementation of effective mitigation strategies will be necessary for improving air quality for in the city.

This study applies a zero-inflated negative binomial regression model to address excess zeros and over dispersion common in environmental health data. This advanced approach enables more precise estimation of short- and long-term exposure effects.

However, no prior study has systematically

examined both short- and long-term exposure effects using advanced count models in Arak. Therefore, this study aims to investigate the association between air pollution and stroke mortality in Arak, Iran, focusing on key pollutants ($PM_{2.5}$, PM_{10}, NO_2, O_3, and SO_2) while adjusting for meteorological and demographic factors.

Material and methods

Study design and population

This was a prospective cohort study of all patients diagnosed with stroke in Arak, Iran, between 2019 and 2022. Information was obtained from the stroke patient registration program in the city. In this study, 1010 patients who fulfilled the inclusion criteria were analyzed and followed up. The inclusion criteria included residence in Arak and confirmed stroke cases hospitalized by a neurologist, according to the WHO criteria. Patients for whom a hospitalization date was not recorded were excluded. The demographic and clinical information of new daily stroke cases, exact dates of events, and details regarding hospitalization were documented by the registry program.

Environmental data collection

The air quality data in Arak, were extracted for the period spanning 2017-2022, from four strategically selected monitoring stations across industrial zones, high-traffic areas, and residential neighborhoods representing diverse and varied pollution sources within this city. The selection has been made in such a way that it allows the derivation of the best air pollution profile for Arak and its possible impacts on public health. The inclusion of monitoring stations in the analysis has been based on the following criteria [18, 19]:

1. Geographical representation: The stations were distributed in key areas to capture widespread variations in pollutant levels caused by industrial emissions passing through highly dense vehicular traffic and residential activities.

2. Data availability: According to the WHO guidelines, only the stations with ≥75% data availability for the concentration of pollutants in every hour of the study period were considered,

3. Proximity to health data sources: The stations were chosen near the general hospitals and residential areas in order to increase the relevance of exposure data to stroke cases analyzed.

Validation and data processing

To ensure the reliability of the data:

1. Quality control measures: The pollutant concentration data on an hourly basis was strictly validated to remove negative values and outliers. The measurements that were very far from the neighboring stations or meteorological patterns were also flagged and excluded.

2. Time aggregation: While the mean daily concentrations of $PM_{2.5}$, PM_{10}, NO_2, and SO_2 were all determined, O_3 was quantified using a daily maximum of the mean over 8 consecutive hours according to the dictates of international standards of air quality.

3. Comparative analysis: Data trends from selected stations were compared against independent regional datasets to ensure consistency and reduce biases.

4. Compliance with standards: The monitoring equipment at each station met the standards of the Iranian DOE and the WHO, hence giving high-precision measurements.

Statistical analysis

The association between air pollutant exposure and stroke mortality was analyzed using a time-

series Zero-Inflated Negative Binomial (ZINB) regression model, appropriate for count data with excess zeros and over dispersion. The main analytical procedures were as follows:

● *Model framework:*

o The ZINB model was used to account for over dispersion and a high proportion of zero counts in daily stroke mortality data.

o The model includes two components:

1. A count component describing the number of stroke events.

2. A zero-inflation component estimating the probability of days with zero events using a logit link function.

● *Model fitting and estimation:*

o Parameters were estimated via maximum likelihood estimation (MLE).

o The likelihood ratio test was applied to confirm the presence of overdispersion and justify the use of the negative binomial over the Poisson model.

o Coefficients were exponentiated to obtain Relative Risks (RRs) for the count part and Odds Ratios (ORs) for the zero-inflation part.

● *Exposure variables:*

o Daily concentrations of $PM_{2.5}$, PM_{10}, NO_2, O_3, and SO_2 were used as independent variables.

o Effects were estimated for short-term exposures (1–3 months) and long-term exposures (6–24 months).

● *Confounder adjustment:*

o All models were adjusted for temperature, humidity, seasonality, and demographic variables (age, sex, smoking status, hypertension, diabetes).

● *Interpretation of components:*

o The count component estimates the effect of pollutant levels on the number of stroke deaths on days when events occurred.

o The zero component captures the probability of observing zero daily stroke deaths, representing periods of low or no pollution-related impact.

● *Model validation and robustness:*

o Diagnostic plots and residual analyses were used to assess model fit and ensure no violation of independence or homoscedasticity assumptions.

o Sensitivity analyses were performed by varying lag structures to evaluate temporal stability of associations.

All analyses were performed using the R, version 4.1.3.

Results and discussion

Demographic and clinical features in participants

The demographic and clinical characteristics of the patients participating in the study are summarized in Table 1. The mean age of the participants was 70.67+12.8 years old. Among these, 52.77% were male, 22.38% were smokers, 10% were passive smokers, 15.54% had each given a history of opium use, 80.69% had high blood pressure, and 30.4% had diabetes.

Fig. 1 presents the box and whisker plots of daily average air pollutant concentrations from 2017 to 2022 with mean, median, and IQR. Daily means of air pollution levels varied from 2.9- 194 μg/m^3 for $PM_{2.5}$, 11.3- 206.7 μg/m^3 for PM_{10}, 3.8- 56.8 ppb for O_3, 1.5- 179.5 ppb for SO_2, and 7.4-56 ppb for NO_2.

Table 1. Demographics and clinical characteristics of participants

Variable	Category	Mean (±SD) or N (%)
Age (Years)		70.67 (±12.8)
Gender	Female	477 (47.23)
	Male	533 (52.77)
Smoking	Yes	226 (22.38)
	No	784 (77.62)
Passive smoker	Yes	101 (10.0)
	No	909 (90.0)
Opium	Yes	157 (15.54)
	No	853 (84.46)
Hypertension	Hypertensive	815 (80.69)
	Normotensive	195 (19.31)
Diabetes	Yes	307 (30.40)
	No	703 (69.6)

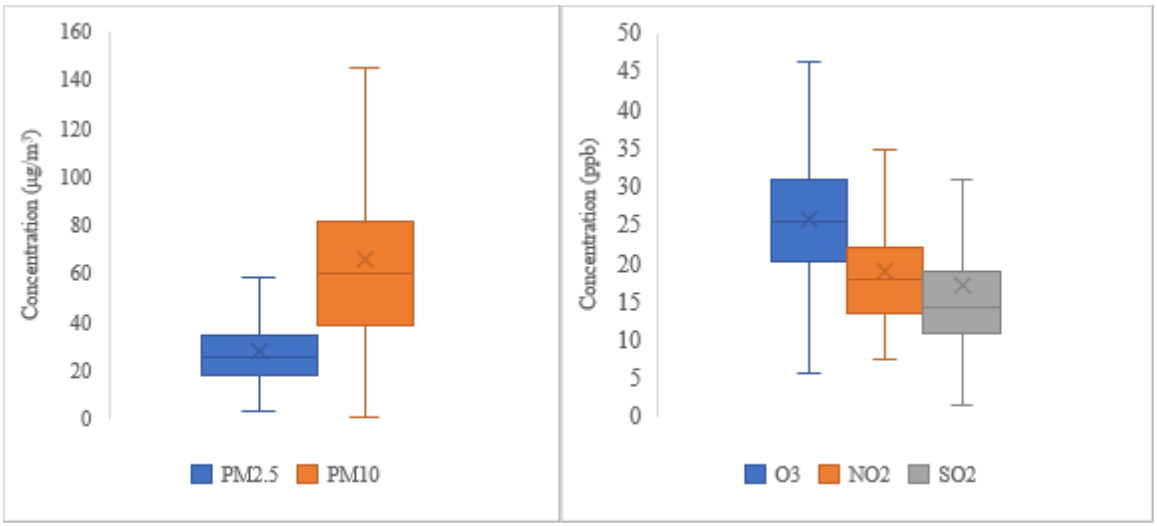

Fig. 1. Box and whisker plots with the mean, median, and interquartile range (IQR) of the daily average concentrations of air pollutants over the study period

Short-Term exposure analysis

Table 2 summarizes the associations between short-term pollutant exposures (1–3 months) and stroke mortality. A slight increase in risk was observed for PM_{10} at the 2-month lag (RR = 1.06, 95% CI: 0.98–1.14), although this relationship did not reach statistical significance. NO_2 exhibited the strongest short-term association, particularly at 2-month (RR = 1.50, 95% CI: 1.30–1.75, $p < 0.001$) and 3-month (RR = 1.35, 95% CI: 1.10–1.70, p = 0.015) intervals, indicating a robust positive relationship with stroke mortality.

In contrast, SO_2 showed an inverse association at both 2-month (RR = 0.59, 95% CI: 0.36–0.97, p = 0.04) and 3-month (RR = 0.70, 95% CI: 0.50–0.97, p = 0.029) lags, suggesting potential confounding or pollutant interaction effects. Ozone (O_3) demonstrated borderline significance at the 1-month lag (RR = 0.62, 95% CI: 0.37–1.02).

In the zero-inflated model, PM_{10} was significantly associated with stroke mortality at the 2-month lag (OR = 1.19, 95% CI: 1.02–1.36), while O_3 also showed an elevated risk at 1-month (OR = 1.70, 95% CI: 1.03–2.90). $PM_{2.5}$ and NO_2 associations were weaker or inconsistent across short-term lags.

Overall, NO_2 emerged as the key short-term pollutant of concern, followed by PM_{10}, indicating that traffic and industrial emissions may acutely influence cerebrovascular mortality in Arak.

Long-Term exposure assessment

$PM_{2.5}$ showed a significant positive association with stroke mortality at the 24-month lag (RR = 1.20, 95% CI: 1.05–1.58, p=0.01), emphasizing the cumulative impact of fine particulate matter on cerebrovascular outcomes. PM_{10} did not display significant long-term associations, with RR values remaining close to unity across all periods.

O_3 exhibited mixed effects, with an increased risk at 12 months (RR = 1.55, 95% CI: 1.26–1.90) but a decreased risk at 24 months (RR = 0.47, 95% CI: 0.34–0.66), suggesting temporal variability or potential co-pollutant interactions.

For NO_2, a negative association was found at both 12-month (RR = 0.80, 95% CI: 0.65–0.99, p=0.04) and 24-month (RR = 0.36, 95% CI: 0.20–0.60, p<0.001) lags. Similarly, SO_2 demonstrated a significant negative effect during the first 6 months (RR=0.72, 95% CI: 0.56–0.94, p=0.02), which diminished over time.

In the zero-inflated model, $PM_{2.5}$ showed a notable effect at the 24-month lag (OR=2.70, 95% CI: 1.40–3.68, p=0.07), while O_3 demonstrated a strong negative association at the same lag (OR=0.019, 95% CI: 0.001–0.18, p=0.007).

Collectively, the long-term findings highlight $PM_{2.5}$ as the dominant chronic exposure factor, with PM_{10} and O_3 contributing variably across time frames.

Table 3 shows the stroke mortality about 10-unit increments in long-term air pollutant concentrations ($PM_{2.5}$, PM_{10}, O_3, SO_2, and NO_2).

Table 2. Stroke mortality about 10-unit increments in short-term air pollutant concentrations ($PM_{2.5}$, PM_{10}, O_3, SO_2, and NO_2)

Model	Pollutants	1 month			2 months			3 months		
		RR/ OR	95% CI	P value	RR/OR	95% CI	P value	RR/ OR	95% CI	P value
Standard Poisson regression model (RR)	$PM_{2.5}$	0.86	0.72-1.014	0.07	0.86	0.73-1.03	0.105	0.91	0.76-1.08	0.34
	PM_{10}	1.034	0.98-1.09	0.18	1.06	0.98-1.14	0.14	1.04	0.96-1.12	0.28
	O_3	0.62	0.37-1.02	0.06	1.01	0.69-1.47	0.95	1.37	0.98-1.9	0.07
	NO_2	1.2*	1.03-1.4	0.02	1.5	1.3-1.75	<0.001	1.35	1.1-1.7	0.015
	SO_2	0.84	0.66-1.06	0.14	0.59	0.36-0.97	0.04	0.7	0.5-0.97	0.029
Zero-inflated model (OR)	$PM_{2.5}$	1.04	0.98-1.34	0.99	0.44	0.28-0.68	0.002	0.24	0.11-0.49	0.001
	PM_{10}	0.69	0.29-1.52	0.81	1.19	1.02-1.36	0.02	1.24	0.99-1.6	0.06
	O_3	1.7	1.03-2.9	0.64	1.02	0.56-1.84	0.96	1.1	053-2.2	0.82
	NO_2	1.65	1.72-1.59	0.76	0.95	0.74-1.2	0.66	0.78	0.5-1.2	0.29
	SO_2	0.69	0.05-8.5	0.77	0.86	0.35-2.08	0.74	0.8	0.48-1.5	0.55

RR = relative risk, OR= odds ratio, CI = confidence interval

Table 3. Stroke mortality about 10-unit increments in long-term air pollutant concentrations ($PM_{2.5}$, PM_{10}, O_3, SO_2, and NO_2)

Model	Pollutants	6 months			12 months			24 months		
		RR/ OR	95% CI	P value	RR/ OR	95% CI	P value	RR/ OR	95% CI	P value
Standard Poisson regression model (RR)	$PM_{2.5}$	0.9	0.75-1.1	0.34	1.06	0.9-1.23	0.44	1.2	1.05-1.58	0.01
	PM_{10}	0.96	0.87-1.04	0.32	1.1	0.95-1.09	0.72	0.94	0.88-1.02	0.15
	O_3	1.13	0.9-1.4	0.29	1.55	1.26-1.9	0.003	0.47	0.34-0.66	0.0001
	NO_2	0.75	0.52-1.05	0.09	0.8	0.65-0.99	0.04	0.36	0.2-0.6	0.0002
	SO_2	0.72	0.56-0.94	0.02	0.82	0.67-1.01	0.06	0.94	0.83-1.07	0.36
zero-inflated model (OR)	$PM_{2.5}$	0.9	0.58-1.4	0.63	0.48	0.19-1.2	0.13	2.7	1.4-3.68	0.07
	PM_{10}	0.95	0.87-1.04	0.11	1.24	0.9-1.7	0.19	0.67	0.34-1.29	0.23
	O_3	1.13	0.9- 1.42	0.29	2.43	0.8-7.35	0.12	0.019	0.001-0.18	0.007
	NO_2	0.74	0.52-1.05	0.09	0.18	0.016-2.05	0.016	2.7	0.9-4.56	0.09
	SO_2	0.72	0.56-0.94	0.25	0.44	0.15-1.98	0.15	1.5	0.7-3.2	0.28

RR = relative risk, OR= odds ratio, CI = confidence interval

The findings of this study provide important knowledge on the relationship between air pollution and stroke mortality in both short- and long-term exposure. It indicates that a rise in the PM_{10} and NO_2 levels insignificantly raises the incidence of stroke. These results are consistent with previous research demonstrating the adverse cardiovascular and cerebrovascular effects of air pollutants. For example, one systematic review highlighted that ambient air pollution exposure is linked with stroke-related hospital admissions and mortalities, therefore underlining again that air quality also represents a serious public health issue [7]. The reported associations of PM_{10} exposure with stroke mortality are consistent with findings reported of many researchers, who documented strong associations of PM_{10} exposure and ischemic strokes, as well as hemorrhagic strokes [20]. Similarly, the present study's NO_2 data supports a study by researchers in 2018, who identified NO_2 as a major traffic pollutant impacting cerebrovascular outcomes [1]. The consistency of effects associated with NO_2 across studies further confirms its role as a good indicator of car pollution and stroke risk.

For O_3, variable correlations presented in this study align with findings by many researchers in 2023, who demonstrated temporal variations in the effect of O_3 during and after the COVID-19 lockdown in industrial Iranian cities [21]. The variable association between exposure to O_3 and stroke mortality may depend on atmospheric chemistry, co-pollutant interaction, and meteorology.

The positive long-term association of $PM_{2.5}$ exposure and stroke death correspond to global cohort studies, which reported that long-term exposure to fine particulate matter significantly increases cardiovascular death. Furthermore, the magnitude of $PM_{2.5}$-associated risk in this current analysis is of comparable degree compared to other Asian megacities, indicating additive exposure and long-term health burden [22, 23].

Unexpected negative correlations with SO_2 were also observed, in agreement with observations from some earlier regional analyses [24]. Such reverse associations may reflect uncontrolled confounding, interaction between more than one pollutant, or "harvesting effects," whereby vulnerable subjects experience early deaths during peak pollution episodes, lowering artificially subsequent risk estimates.

The results of the present study offer significant local data for Arak, an industrialized metropolis with continuously poor air quality. The short-term strong correlation between NO_2 and the long-term effect of $PM_{2.5}$ indicate the added effect of vehicle exhaust and industrial emissions. The results accord with regional monitoring data that have ranked Arak as one of the most polluted cities in Iran [10, 17]. From the public health viewpoint, the findings document the overriding significance of: Stricter emission restrictions on traffic and industry emissions; Development of early warning systems of air pollution peaks; and Specialized interventions for vulnerable subjects (for example, elderly or hypertensive individuals). Urban policy needs to place emission reduction measures and encourage urban greenings as air pollution reduction strategies. Evidence further highlights the fact that even marginal pollutant cuts would achieve significant reductions in stroke death.

Although the study gives significant insights, there are some limitations to be considered. Initially, the exposure estimation relied on ambient air measurements, which might not be representative of people-level variability, especially concerning indoor and occupational situations. Second, while crucial confounding factors like humidity and temperature were controlled, other factors for example, physical activity level, or medication use may have influenced the results. Third, negative or inverse associations for SO_2 should

be treated with caution, because they may be statistical artefacts, residual confounding, or data-specific effects. Further studies should focus on: Findings of multi-pollutant and interaction models to comprehend intricate air effects. And carry out comparisons between various Iranian cities having dissimilar characteristics of air pollutants. Longitudinal and mechanistic research are essential to distinguish the biological mechanisms by which NO_2, $PM_{2.5}$, and PM_{10} affect cerebrovascular endpoints. Efforts to estimate the relative efficacy of local emission reduction rules would likewise enhance the connection between policy evidence and policy action.

Conclusion

This study demonstrated strong correlation between short- and long-duration exposure to major air pollutants, i.e., NO_2, PM_{10}, and $PM_{2.5}$, and increased stroke death rates in Arak, Iran. These results provide strong localized evidence of the link between emissions by industry and vehicles and the risks of cerebrovascular morbidity within one of the most industrially polluted Iranian cities. It was particularly observed that inverse or negative associations were revealed for SO_2, which might reveal the existence of confounding factors, complex interaction among pollutants, or localized air circumstances. These findings highlight the importance of future studies to understand the combined and interactive effects of different pollutants on health endpoints.

This evidence highlights the strong need for specialized environmental policy focusing on the minimization of emissions arising both from industry and traffic. Enacting stricter air quality regulations, supporting clean energy technologies, and increasing urban green spaces are capable of significantly lowering pollutant levels and advancing the health of the populace.

It would be indispensable for future investigations to emphasize longitudinal and mechanistic studies and to unwind the biological processes through which air pollutants cause stroke. Assessing the effect of air quality interventions and combining the exposure data and clinical and individual-level data would also be invaluable for evidence-informed policy-making. Overall, this study adds meaningful provincial perspectives to the growing body of literature highlighting the idea of improving air quality as a direct investment in the health of the populace, particularly in rapidly industrializing regions like Arak and many urban centers throughout Iran.

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Competing interests

All authors declared no conflict of interest.

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Ethical considerations

"Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely ob-served by the authors." All stages of research were conducted following the Declaration of Helsinki and the Ethical Statements of the Ethics Committee of Arak University of Medical Sciences. This study was approved by the Ethics Committee of Arak University of Medical Sciences (Ethical code: IR.ARAKMU.REC.1401.126).

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