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The association between short-term PM2.5 exposure and the incidence of NSTEMI: A case-crossover study

Shayan Pourghazi[1], Roxana Sadeghi[1], Niloufar Taherpour[2], Mohammad Haji Aghajani[3], Mohammad Parsa Mahjoob[1], Amir Heidari[1*]

1- Department of Cardiology, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
2- Prevention of Cardiovascular Disease Research Center, Department of Epidemiology, School of Public Health and Safety, Shahid Beheshti University of Medical Sciences, Tehran, Iran
3- Prevention of Cardiovascular Disease Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran

Correspondence:

Amir Heidari;
Department of Cardiology, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran;
Email: amir_h2026@yahoo.com

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Abstract

BACKGROUND: The incidence of acute myocardial infarction (AMI) is on the rise, partly due to exposure to particulate matter (PM). However, the nature of the events and individuals at higher risk is unclear. This study examines the relationship between air pollution exposure, specifically particles with a diameter <2.5 microns (PM2.5), and the occurrence of non-ST-segment elevation myocardial infarction (NSTEMI).

METHODS: In this case-crossover study, NSTEMI patients in Imam Hossein Hospital during 2021–2024 were considered. PM2.5 particle levels in Tehran during the 24 hours before NSTEMI admission and during three control periods (7, 14, and 21 days earlier) were recorded. Data were analyzed using Stata 17 and conditional logistic regression.

RESULTS: Of 4,686 patients, 216 (4.61%) experienced NSTEMI. The median PM2.5 level was 91.5 µg/m³ (interquartile range = 78–113). PM2.5 levels did not differ between risk and control times (P = 0.740). Median PM2.5 levels were highest in autumn, followed by winter, and lowest in spring (P < 0.001). PM2.5 levels were not strongly associated with the occurrence of NSTEMI (P = 0.268). Considering PM2.5 levels, the occurrence of NSTEMI during winter was 3.42-fold greater than in autumn (OR = 3.42, 95% CI = 1.07–10.59). A significant association between PM2.5 levels and NSTEMI was observed only in winter, where each 1 µg/m³ increase in PM2.5 was associated with slightly reduced odds of NSTEMI (OR = 0.98, 95% CI: 0.97–0.99).

CONCLUSION: Exposure to PM2.5 was not related to the incidence of NSTEMI. Nevertheless, seasonal factors, particularly in autumn and winter, could be responsible for NSTEMI events.

Keywords: Air Pollution; Fine Particulate Matter; Non-ST-Segment Elevation Myocardial Infarction; Cardiovascular Diseases

Introduction

Air pollution is a critical issue in urban areas, with negative impacts on the climate, public health, and the environment[1]. It is an acute issue, particularly in developing countries such as Iran, where population growth, industrialization, traffic, dust storms, and droughts have significantly deteriorated air quality[2–4]. In large cities such as Tehran, air pollution is a significant problem. Similarly, local governments have implemented initiatives to enhance air quality and diminish health risks posed by pollution[3].

Long-term exposure to high levels of air pollution seriously affects human health, contributing to a variety of diseases, including cardiovascular and respiratory diseases, dry eyes, diabetes, immune impairment, neurological diseases, and reduced fertility, as well as shortening life expectancy[5–9]. The most significant urban air pollutants are carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and particulate matter (PM), all of which are linked to cardiovascular, respiratory, and neurological diseases[10]. Of these contaminants, PM is the most health-hazardous[11–13]. Exposure to PM, particularly particles with diameters smaller than 2.5 microns (PM2.5), is highly relevant to human health and is one of the strongest predictors of mortality rates in populations[14,15]. Research has demonstrated that PM2.5 can penetrate deep into the lungs and adversely affect cardiovascular health[16,17]. The consequences of exposure to these toxicants include increased inflammation, mitochondrial dysfunction, and coagulation issues[18], all of which carry a risk of inducing cardiovascular disease (CVD), such as myocardial infarctions and congestive heart failure[18,19].

There is growing concern over the increasing levels of particulate matter in urban centers, especially in most developing nations. Due to the limited availability of information on the association between PM2.5 and CVD in Iran, this study aims to explore the association between short-term PM2.5 air pollution and NSTEMI incidence in patients admitted to the hospital.

Methods

Study Design and Patient Selection

This case-crossover study was conducted from 27 July 2021 to 5 November 2024 on all NSTEMI patients who were referred to Imam Hossein Educational Hospital's emergency department and admitted to the CCU. This study was approved by the Ethics Committee of the School of Medicine, Shahid Beheshti University of Medical Sciences (approval code: IR.SBMU.MSP.REC.1403.107). The Coronary Angiography and Angioplasty Registry (CAAR)[20] was used to obtain demographic and clinical information from the patients. CAAR was established to track patients undergoing coronary angiography and angioplasty who are referred to Imam Hossein Hospital, a major referral center in eastern Tehran, Iran.

Inclusion and exclusion criteria

Adult patients who are 18 years and older residing in Tehran with NSTEMI who fulfill the requirements of the study, if they have symptoms based on acute coronary syndrome (ACS) and raised troponin (Troponin I > 0.05 ng/mL) but were negative for alterations according to ECG for STEMI. Patients were excluded from the study if they were diagnosed with STEMI or unstable angina upon hospitalization, had active infectious, autoimmune, or inflammatory illnesses (such as lupus or rheumatoid arthritis), or had traveled outside of Tehran within one month of hospitalization.

Data collection

All NSTEMI patients from 27 July 2021 to 5 November 2024 were listed. Demographic and clinical data, including age, gender, history of chronic conditions, and their treatment, were collected from the CAAR system after completing the patient list. Air pollution data prior to patients' admission were collected to assess short-term exposure to PM2.5. This included observations 24 hours before admission, as well as 7, 14, and 21 days prior. The data were obtained from the Tehran Air Quality Control Company and the Tehran Environmental Protection Department,

both of which operate continuously and measure particulate matter hourly (https://airnow.tehran.ir/home/DataArchive.aspx). The intensity of pollution is monitored continuously with 21 operational air quality monitoring stations distributed throughout Tehran. The mean daily concentration of particulate matter (PM) was calculated using the beta radiation absorption method with a Geiger-Müller counter for particles smaller than 2.5 microns. Data were provided by the nearest meteorological station to the patient's residence.

Risk period and control period

For each patient, a risk period was defined as the 24 hours before hospitalization due to NSTEMI. Three control periods matching the risk period were selected as 7, 14, and 21 days before hospitalization. Control periods were chosen at weekly fixed intervals before the baseline in order to avoid seasonal and weekly trend-based biases.

Statistical analysis

Normal distribution of quantitative variables was checked with histograms. For the measurement of quantitative variables, mean ± SD or median and interquartile range (IQR = Q1-Q3) were employed, while percentages (%) and frequencies were utilized for the description of qualitative variables. The Kruskal-Wallis non-parametric test was employed for comparing the mean of PM2.5 between the study groups since normal distribution was not present. Finally, conditional logistic regression was used to explore the association between PM2.5 levels and the risk of NSTEMI in univariate and multivariable models (while adjusting for seasonal effects), considering one risk period and three control periods per subject. Statistical inferences were reported in two-tailed form with a significance level of less than 0.05. The outcomes of statistical models were expressed as odds ratios (OR) with a 95% confidence interval (CI). Statistical analysis was conducted using Stata version 17 (StataCorp LLC, College Station, TX 77845, USA).

Results

Demographic and clinical information of patients

Out of a total of 4,686 patients living in Tehran, 225 were diagnosed with NSTEMI, accounting for a prevalence of 4.80% (95% CI = 4.27%-5.45%). Of these 225 NSTEMI patients, 216 met the inclusion criteria. The mean age of the NSTEMI patients was 61.71 ± 11.75 years, and 62.04% were younger than 65 years. Among them, 71.30% were male. The prevalence of overweight and obesity in this patient group was 63.91%. Additionally, 52.31% had a family history of ischemic heart disease (IHD) in at least one first-degree relative. The most common underlying conditions among the patients were hypertension (44.91%), diabetes mellitus (41.67%), and IHD (37.96%). Furthermore, 12.50% had a history of myocardial infarction, and 41.67% were smokers. Additional patient data are shown in Table 1.

Descriptive analysis of PM2.5 levels at different times and seasons

In this study, a total of 52 NSTEMI cases occurred in spring, 57 in summer, 50 in autumn, and 57 in winter. According to the results presented in Table 2, the overall median PM2.5 concentration during the study was 91.5 μg/m³, with an interquartile range of 78–113 μg/m³ and a minimum–maximum range of 40–229 μg/m³. This indicates that the median PM2.5 concentration was higher than the ideal concentration of 25 μg/m³. The median PM2.5 during the risk season as a whole was 90.5 μg/m³, which was lower than that of the three control seasons (P = 0.740). Autumn had the highest median PM2.5 value (median = 104, IQR = 85–132), followed by winter, while spring had the lowest median PM2.5 value (median = 79, IQR = 67–93) in Tehran (P < 0.001). At any of the study periods and seasons, the permissible PM2.5 level in Tehran was never below 25 μg/m³.

Association between PM2.5 level and season with NSTEMI incidence

Univariate analysis based on conditional logistic regression, as shown in Table 3, indicated that

Table 1. Demographic and clinical information of NSTEMI patients

Factors	Total (n =216)
General information	
Age (Years)	61.71 ± 11.75
≤65	134 (62.04)
>65	82 (37.96)
Gender	
Female	62 (28.70)
Male	154 (71.30)
Body Mass Index (BMI, kg/m²)	27.56 ± 4.36
<25 kg/ m²	65 (30.09)
≥25 kg/ m²	151 (69.91)
Vital status	
Alive discharged	213 (98.61)
Deceased	3 (1.39)
Habits and medical history (Yes)	
Smoking status	
Non-smoker	109 (50.46)
Current smoker	90 (41.67)
Ex-smoker	17 (7.87)
Myocardial Infarction (MI)	27 (12.50)
Cerebrovascular Accident (CVA) / Transient Ischemic Attack (TIA)	20 (9.26)
Ischemic Heart Diseases (IHDs)	82 (37.96)
Diabetes	90 (41.67)
Hypertension	97 (44.91)
Dyslipidemia	79 (36.57)
Heart failure	68 (31.48)
Cancer	6 (2.78)
Chronic respiratory diseases	10 (4.63)
Chronic Kidney disease	14 (6.48)
Peripheral Vascular Diseases (PVDs)	5 (2.31)
Positive family history of IHD	113 (52.31)
Drug history (Yes)	
ASA	88 (40.74)
Clopidogrel	28 (12.96)
Nitrate	41 (18.93)
Anti-hypertensive gents	105 (48.61)
Lipid-lowering agents	82 (37.96)
Insulin or anti-diabetic agents	67 (31.02)
Other[1]	6 (2.78)

Data presented as mean ± standard deviation or frequency and percentage
[1]Such as DOAC, Warfarin, Nicorandil, Ranolazine

Table 2. Descriptive analysis of PM2.5 levels at different times and seasons

Sub groups	PM2.5 (μm/m³)		
	Median (Q1–Q3)	Min – Max	P_value
Total	91.5 (78 – 113)	40 – 229	-
Duration			
24 hours before event (Risk period)	90.5 (79 – 103.5)	42 – 229	0.740
7 days before event (Control period)	91 (79 – 117)	40 - 196	
14 days before event (Control period)	91.5 (78 – 115)	40 - 229	
21 days before event (Control period)	92 (76 – 113)	44 - 171	
Season			
Spring	79 (67 – 93)	40 – 229	**<0.001***
Summer	89 (80 – 106)	66 – 163	
Autumn	104 (85 – 132)	45 – 172	
Winter	97 (76 – 124)	50 – 176	

Data presented as median and interquartile range (Q1 – Q3)
*Statistically significant, P_value<0.05

Table 3. Association between PM2.5 level and season with NSTEMI incidence

Factors	Crude model OR (95%CI)	P_value	Adjusted model OR, 95%CI	P_value
Fine Particulate Matter Air Pollution				
PM2.5 (μm/m³)	0.99 (0.98 – 1.002)	0.184	0.99 (0.98 – 1.002)	0.268
Seasons				
Spring	2.42 (0.51 – 11.39)	0.262	2.06 (0.42 – 9.97)	0.367
Summer	2.77 (0.56 – 13.70)	0.211	2.63 (0.53 – 12.99)	0.233
Autumn	Reference	-	Reference	-
Winter	**3.74 (1.18 – 11.84)**	**0.025***	**3.42 (1.07 – 10.59)**	**0.038***

[1]OR, 95%CI: Odds Ratio, 95% Confidence Interval
Crude model: assessed the association between PM2.5 levels **or** seasons and NSTEMI occurrence
Adjusted model: assessed the association between PM2.5 levels and seasons with NSTEMI occurrence
*Statistically significant, P_value<0.05

while the season was significantly associated with the occurrence of NSTEMI (P = 0.025), PM2.5 concentration was not significant (P = 0.184). Multivariable conditional logistic regression analysis, adjusting for both PM2.5 concentrations and season types, indicated that the winter season had a 3.42-fold greater incidence of NSTEMI than autumn (Odds Ratio [OR] = 3.42, 95% CI = 1.07–10.59, P = 0.038).

Association between PM2.5 levels and NSTEMI incidence in different subgroups

Table 4 shows the association between PM2.5 and NSTEMI in different subgroups based on the patients' clinical conditions. Based on univariate conditional logistic regression analysis, PM2.5 level was only significantly associated with NSTEMI in patients with no history of dyslipidemia (OR = 0.99, 95% CI = 0.98–0.99, P = 0.046). However, after controlling for seasonal influence in the multivariable analysis, PM2.5 level was not significantly associated with NSTEMI in any of the subgroups analyzed (P > 0.05). Furthermore, in patients with a history of diabetes (OR = 1.0006, 95% CI = 0.99–1.01), dyslipidemia (OR = 1.002, 95% CI = 0.99–1.01), and heart failure (OR = 1.0007, 95% CI = 0.98–1.01), a one-unit increase in PM2.5 levels had an insignificant association as a risk factor for NSTEMI (P > 0.05).

Finally, stratified analyses by season showed that a statistically significant association between PM2.5 and NSTEMI was found only in winter, where each 1 μg/m³ increase in PM2.5 was associated with a slightly reduced odds of NSTEMI (OR = 0.98, 95% CI = 0.97–0.99). No statistically significant associations were observed in other seasons.

Discussion

The findings indicated that air quality in the study area was poor, yet there were no significant differences between the risk and control periods for PM2.5 levels. However, detectable differences between the seasons for PM2.5 levels were noted, with autumn and winter having higher levels and spring having lower levels (P < 0.001). Various factors could influence PM2.5 levels throughout the year. Carbonaceous materials play a critical role in PM2.5[21], and increased domestic energy consumption and fossil fuel burning during the winter months, along with weather inversions, appear to generate higher concentrations of PM2.5 at this time. During winter, air quality deteriorates because temperature inversions keep air pollutants near the surface[4]. Conditional logistic regression results indicated that seasonality was statistically significant (P = 0.025), but PM2.5 concentrations were not significantly related to NSTEMI events (P = 0.184). Multivariable conditional logistic regression analysis confirmed this, showing that NSTEMI occurrence was 3.42 times higher during winter compared to autumn. Moreover, after adjustment for seasonal effects, PM2.5 levels did not show a significant association with NSTEMI occurrence in any of the subgroups. This suggests that air pollutants such as PM2.5 may

Table 4. Association between PM2.5 levels and NSTEMI incidence in different subgroups

Subgroups	Crude model OR, 95%CI[1]	P_value	Adjusted model OR, 95%CI[1]	P_value
Gender				
Female	0.99 (0.98 – 1.009)	0.720	0.99 (0.98 – 1.009)	0.634
Male	0.99 (0.98 – 1.002)	0.172	0.99 (0.98 – 1.003)	0.221
Age				
≤65 years	0.99 (0.99 – 1.007)	0.776	0.99 (0.99 – 1.008)	0.870
>65 years	0.99 (0.98 – 1.001)	0.095	0.99 (0.98 – 1.001)	0.100
Smoking status				
Current smoker	0.98 (0.97 – 1.0007)	0.052	0.99 (0.97 – 1.0007)	0.764
Non-smoker	1.001 (0.99 – 1.01)	0.766	1.001 (0.99 – 1.01)	0.068
Ex-smoker	0.98 (0.96 – 1.01)	0.362	0.98 (0.95 – 1.02)	0.477
Diabetes				
Non-diabetic	0.99 (0.98 – 1.0005)	0.065	0.99 (0.98 – 1.0007)	0.070
Diabetic	1.0006 (0.99 – 1.01)	0.903	1.0006 (0.99 – 1.01)	0.908
Hypertension				
Non- hypertensive	0.99 (0.98 – 1.001)	0.093	0.99 (0.98 – 1.001)	0.103
Hypertensive	0.99 (0.98 – 1.008)	0.866	0.99 (0.99 – 1.009)	0.978
History of dyslipidemia				
No	**0.99 (0.98 – 0.99)**	**0.046***	0.99 (0.98 – 1.0003)	0.060
Yes	1.001 (0.99 – 1.01)	0.708	1.002 (0.99 – 1.01)	0.688
History of IHD				
No	0.99 (0.98 – 1.001)	0.119	0.99 (0.98 – 1.001)	0.125
Yes	0.99 (0.98 – 1.009)	0.823	0.99 (0.98 – 1.01)	0.983
History of heart failure				
No	0.99 (0.98 – 1.001)	0.118	0.99 (0.98 – 1.001)	0.142
Yes	1.0003(0.98 – 1.01)	0.954	1.0007(0.98 – 1.01)	0.905
Family history of IHD				
Negative	0.99 (0.98 – 1.007)	0.618	0.99 (0.98 – 1.007)	0.640
Positive	0.99 (0.98 – 1.003)	0.164	0.99 (0.98 – 1.003)	0.211
History of ASA use				
No	0.99 (0.98 – 1.001)	0.102	0.99 (0.98 – 1.001)	0.111
Yes	0.99 (0.98 – 1.008)	0.787	0.99 (0.98 – 1.009)	0.912
Season				
Spring	1.006 (0.99 – 1.01)	0.317	-	-
Summer	0.99 (0.97 – 1.01)	0.498	-	-
Autumn	1.008 (0.99 – 1.02)	0.271	-	-
Winter	**0.98 (0.97 – 0.99)**	**0.020***	-	-

[1]OR, 95%CI: Odds Ratio, 95% Confidence Interval
Crude model: assessed the association between PM2.5 levels and NSTEMI occurrence in each subgroup
Adjusted model: assessed the association between PM2.5 levels and season with NSTEMI occurrence in each subgroup
*Statistically significant, P_value<0.05

not contribute noticeably to NSTEMI occurrence and that certain clinical and environmental factors may play a more vital role.

Various factors can be attributed to temperature and NSTEMI incidence, particularly in the winter season. Low temperatures tend to increase blood viscosity, thereby raising the risk of thrombosis. Furthermore, exposure to decreased atmospheric temperatures has been associated with increased levels of serum catecholamines, fibrinogen, and platelets, as well as increases in blood pressure and heart rate[22,23]. Cold temperatures also cause vasoconstriction, leading to increased cardiac workload and decreased oxygen delivery, which results in ischemia[24]. Mortality rates due to IHD and cerebrovascular disease may also be attributed to thrombosis as a result of blood concentration during cold temperatures[22,25]. Research by Seah et al. (2022)[26] identified reduced temperatures as being associated with an increased risk of NSTEMI. Kobayashi et al. (2023)[27] explored how daily temperature changes influence the incidence of STEMI and postulated that avoidance of cold temperatures could prevent STEMI[27]. Furthermore, Shuka et al. (2024)[28] found that ACS patients were hospitalized primarily in spring and autumn,

showing a strong association between rising ACS incidence and reduced temperatures or sudden changes in atmospheric pressure[28]. Similarly, Hodzic et al. (2018)[29] emphasized fluctuations in ACS events according to seasonal weather conditions, noting that cold exposure is a significant cardiovascular risk factor[29].

Although our research did not involve STEMI patients, Zhang et al. (2016)[30] reported that a 10 μg/m³ increment in PM2.5 (lagged one day) was associated with a 5% higher risk of STEMI among patients aged ≥65 years (OR = 1.05). However, consistent with our findings, no association was detected between PM2.5 levels and NSTEMI[30]. Similarly, Gardner et al. (2014)[31] found no association between PM2.5 and NSTEMI, while an 18% increased risk of STEMI was associated with a 7.1 μg/m³ rise in PM2.5[31]. Pope et al. (2015)[32] also found that short-term exposure to air pollution particles in patients with coronary artery disease was associated with STEMI occurrence rather than NSTEMI. In that study, a rise of 10 μg/m³ in PM2.5 per day was linked with an 8–15% increased risk of STEMI[32].

Mechanistically, STEMIs differ from NSTEMIs because STEMIs result in acute complete blockade of the artery by plaque rupture, while NSTEMIs secondary to plaque rupture do not produce complete occlusion. Plaque rupture is inescapable but does not by itself cause total arterial obstruction; instead, a cascade of coordinated cellular mechanisms must take place before thrombosis formation[31]. Short-term exposure to high-level PM2.5 accelerates blood coagulation, increases susceptibility to thrombosis, disrupts endothelial function, and renders the plaque more vulnerable. In such situations, occlusive arterial thrombosis with resulting plaque rupture is more likely to occur; therefore, enhanced exposure to particulate matter, i.e., PM2.5, leads to complete occlusion of the coronary artery, resulting in full-thickness injury to the myocardium and severely elevated enzyme or troponin levels in STEMI compared to NSTEMI[30].

In contrast to our research, which determined that various clinical subgroups did not have a significant effect on the incidence of NSTEMI—perhaps due to sample size—other studies have shown that younger age, history of smoking, CAD presence, and hyperlipidemia[32], female sex, rural residence, and lower income[33] can also be contributing factors, in addition to the effect of PM2.5, emphasizing the difficulty in precisely defining cardiovascular events in these patients.

Finally, based on our results, stratified analyses revealed a statistically significant association between PM2.5 and NSTEMI during the winter season. Interestingly, the observed direction of effect of PM2.5 on NSTEMI in winter was inverse, which may reflect behavioral, environmental, or physiological factors unique to colder months such as reduced physical activity, increased indoor time, or possible measurement bias[34].

It is important to emphasize that Tehran, as the capital city and the center of the province, experiences high levels of air pollution throughout almost all seasons of the year. Since all PM2.5 concentrations considered in this study exceeded permissible limits, this indicates a consistently elevated exposure among residents. This continuous exposure may intensify the observed associations between air pollution and NSTEMI incidence, but it also poses a challenge for comparing these results with regions where air quality is within acceptable standards. Future research should aim to include populations with a wider range of pollution levels, including those within permissible limits, to better understand the dose-response relationship.

A number of limitations must be noted in this study. While the timeframe for identifying NSTEMI patients was prolonged, the small sample size and single-center study design could have compromised statistical power and restricted the accuracy of event risk estimation. In addition, we grounded our results on ambient concentrations of PM2.5 obtained from monitoring stations and the air quality control system for patient exposure, but did not consider the distance of each patient from the monitoring location. Further, results may have been influenced

by information bias since patients provided simple information themselves, such as their location for 21 days before the event. Our research only considered air pollution and PM2.5 and did not take into account other environmental factors, such as additional pollutants, humidity, wind, and temperature, which could also have influenced patient conditions. Although we considered the short-term impact of PM2.5 exposure, the long-term impact can be substantial and was not addressed in this research. Therefore, we suggest a more extensive analysis through a multicenter study conducted over a longer period of time, incorporating additional environmental conditions and clinical subgroups, to validate our findings.

Conclusion

The findings of this study indicate that exposure to PM2.5 air pollution is not associated with the occurrence of NSTEMI acute coronary events, although the occurrence of NSTEMI was higher in the cold seasons. This suggests that air pollution and PM2.5 alone are not significant determinants of NSTEMI. Other environmental factors (e.g., seasonal variations such as cold weather) and clinical conditions might also have a substantial impact, which should be investigated further.

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Conflict of interests

The authors declare no conflict of interest.

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Author’s Contributions

Study Conception or Design: SP, AH
Data Acquisition: SP, RS
Data Analysis or Interpretation: NT
Manuscript Drafting: SP, MPM, AH
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All authors have approved the final manuscript and are responsible for all aspects of the work.

References

1. Li L, Qian J, Ou CQ, Zhou YX, Guo C, Guo Y. Spatial and temporal analysis of Air Pollution Index and its timescale-dependent relationship with meteorological factors in Guangzhou, China, 2001-2011. Environ Pollut. 2014 Jul;190:75-81. https://doi.org/10.1016/j.envpol.2014.03.020
2. Saniei R, Zangiabadi A, Sharifikia M, Ghavidel Y. Air quality classification and its temporal trend in Tehran, Iran, 2002-2012. Geospat Health. 2016 May 31;11(2):442. https://doi.org/10.4081/gh.2016.442
3. Torbatian S, Hoshyaripour A, Shahbazi H, Hosseini V. Air pollution trends in Tehran and their anthropogenic drivers. Atmos Pollut Res. 2020;11(3):429-42. https://doi.org/10.1016/j.apr.2019.11.015
4. Mohammadi H, Cohen D, Babazadeh M, Rokni L. The effects of atmospheric processes on tehran smog forming. Iran J Public Health. 2012;41(5):1-12.
5. Wong CS, Lin YC, Hong LY, Chen TT, Ma HP, Hsu YH, Tsai SH, Lin YF, Wu MY. Increased Long-Term Risk of Dementia in Patients With Carbon Monoxide Poisoning: A Population-Based Study. Medicine (Baltimore). 2016 Jan;95(3):e2549. https://doi.org/10.1097/MD.0000000000002549
6. Kim KH, Kabir E, Kabir S. A review on the human health impact of airborne particulate matter. Environ Int. 2015 Jan;74:136-43. https://doi.org/10.1016/j.envint.2014.10.005
7. Weichenthal S, Villeneuve PJ, Burnett RT, van Donkelaar A, Martin RV, Jones RR, et al. Long-term exposure to fine particulate matter: association with nonaccidental and cardiovascular mortality in the agricultural health study cohort. Environ Health Perspect. 2014 Jun;122(6):609-15. https://doi.org/10.1289/ehp.1307277
8. Correia AW, Pope CA 3rd, Dockery DW, Wang Y, Ezzati M, Dominici F. Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. counties for the period from 2000 to

2007. Epidemiology. 2013 Jan;24(1):23-31. https://doi.org/10.1097/ede.0b013e3182770237
9. Dix-Cooper L, Eskenazi B, Romero C, Balmes J, Smith KR. Neurodevelopmental performance among school age children in rural Guatemala is associated with prenatal and postnatal exposure to carbon monoxide, a marker for exposure to woodsmoke. Neurotoxicology. 2012 Mar;33(2):246-54. https://doi.org/10.1016/j.neuro.2011.09.004
10. Alesheikh AA, Oskouei A, Atabi F, Helali H. Providing interoperability for air quality in-situ sensors observations using GML technology. Int J Environ Sci Technol. 2005;2:133-40. https://doi.org/10.1007/BF03325867
11. Amarloei A, Fazlzadeh M, Jafari AJ, Zarei A, Mazloomi S. Particulate matters and bioaerosols during Middle East dust storms events in Ilam, Iran. Microchem J. 2020;152:104280. https://doi.org/10.1016/j.microc.2019.104280
12. Naddafi K, Nabizadeh R, Rostamy R, Ebrahimi Kalan M, Hassanvand MS, Fazlzadeh M. Indoor air quality in waterpipe cafés: exposure level to particulate matter. Environ Sci Pollut Res Int. 2019 Sep;26(26):26605-26616. https://doi.org/10.1007/s11356-019-05546-8
13. Rostami R, Kalan ME, Ghaffari HR, Saranjam B, Ward KD, Ghobadi H, et al. Characteristics and health risk assessment of heavy metals in indoor air of waterpipe cafés. Build Environ. 2021;190:107557. https://doi.org/10.1016/j.buildenv.2020.107557
14. Yu Y, Yao S, Dong H, Wang L, Wang C, Ji X, Ji M, Yao X, Zhang Z. Association between short-term exposure to particulate matter air pollution and cause-specific mortality in Changzhou, China. Environ Res. 2019 Mar;170:7-15. https://doi.org/10.1016/j.envres.2018.11.041
15. Amini H, Trang Nhung NT, Schindler C, Yunesian M, Hosseini V, Shamsipour M, et al. Short-term associations between daily mortality and ambient particulate matter, nitrogen dioxide, and the air quality index in a Middle Eastern megacity. Environ Pollut. 2019 Nov;254(Pt B):113121. https://doi.org/10.1016/j.envpol.2019.113121
16. Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, et al. Passage of inhaled particles into the blood circulation in humans. Circulation. 2002 Jan 29;105(4):411-4. https://doi.org/10.1161/hc0402.104118
17. Nemmar A, Vanbilloen H, Hoylaerts MF, Hoet PH, Verbruggen A, Nemery B. Passage of intratracheally instilled ultrafine particles from the lung into the systemic circulation in hamster. Am J Respir Crit Care Med. 2001 Nov 1;164(9):1665-8. https://doi.org/10.1164/ajrccm.164.9.2101036
18. Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. Lancet. 1995 Jan 21;345(8943):176-8. https://doi.org/10.1016/s0140-6736(95)90173-6
19. Heydari G, Taghizdeh F, Fazlzadeh M, Jafari AJ, Asadgol Z, Mehrizi EA, et al. Levels and health risk assessments of particulate matters (PM2.5 and PM10) in indoor/outdoor air of waterpipe cafés in Tehran, Iran. Environ Sci Pollut Res Int. 2019 Mar;26(7):7205-7215. https://doi.org/10.1007/s11356-019-04202-5
20. Aghajani MH, Sadeghi R, Mahjoob MP, Heidari A, Omidi F, Sistanizad M, et al. The Coronary Angiography and Angioplasty Registry (CAAR) in Iran: Registry Protocol and Results of the Pilot Study. Med J Islam Repub Iran. 2024 Jul 15;38:80. https://doi.org/10.47176/mjiri.38.80
21. Zhang J, Wang H, Yan L, Ding W, Liu R, Wang H, et al. Analysis of chemical composition characteristics and source of PM2.5 under different pollution degrees in autumn and winter of Liaocheng, China. Atmosphere (Basel). 2021;12(9):1180. https://www.mdpi.com/2073-4433/12/9/1180
22. Keatinge WR, Coleshaw SR, Cotter F, Mattock M, Murphy M, Chelliah R. Increases in platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter. Br Med J (Clin Res Ed). 1984 Nov 24;289(6456):1405-8. https://doi.org/10.1136/bmj.289.6456.1405
23. Rus AA, Mornoş C. The Impact of Meteorological Factors and Air Pollutants on Acute Coronary Syndrome. Curr Cardiol Rep. 2022 Oct;24(10):1337-49. https://doi.org/10.1007/s11886-022-01759-5
24. Ikäheimo TM. Cardiovascular diseases, cold exposure and exercise. Temperature (Austin). 2018 Feb 1;5(2):123-46. https://doi.org/10.1080/23328940.2017.1414014
25. Neild PJ, Syndercombe-Court D, Keatinge WR, Donaldson GC, Mattock M, Caunce M. Cold-induced increases in erythrocyte count, plasma cholesterol and plasma fibrinogen of elderly people without a comparable rise in protein C or factor X. Clin Sci (Lond). 1994 Jan;86(1):43-8. https://doi.org/10.1042/cs0860043
26. Seah A, Ho AFW, Soh S, Zheng H, Pek PP, Morgan GG, et al. Ambient temperature and hospital admissions for non-ST segment elevation myocardial infarction in the tropics. Sci Total Environ. 2022;850:158010.

https://doi.org/10.1016/j.scitotenv.2022.158010
27. Kobayashi S, Sakakura K, Jinnouchi H, Taniguchi Y, Tsukui T, Watanabe Y, et al. Influence of daily temperature on the occurrence of ST-elevation myocardial infarction. J Cardiol. 2023 Jun;81(6):544-52. https://doi.org/10.1016/j.jjcc.2022.12.005
28. Shuka N, Cabeli A, Simoni L, Gina M, Kondi L, Dado E. Impact of Temperature and Atmospheric Pressure on Hospitalizations of Patients Presenting With Acute Coronary Syndrome. Cureus. 2024 Feb 24;16(2):e54833. https://doi.org/10.7759/cureus.54833
29. Hodzic E, Perla S, Iglica A, Vucijak M. Seasonal Incidence of Acute Coronary Syndrome and Its Features. Mater Sociomed. 2018 Mar;30(1):10-4. https://doi.org/10.5455/msm.2018.30.10-14
30. Zhang Q, Qi W, Yao W, Wang M, Chen Y, Zhou Y. Ambient Particulate Matter (PM2.5/PM10) Exposure and Emergency Department Visits for Acute Myocardial Infarction in Chaoyang District, Beijing, China During 2014: A Case-Crossover Study. J Epidemiol. 2016 Oct 5;26(10):538-45. https://doi.org/10.2188/jea.je20150209
31. Gardner B, Ling F, Hopke PK, Frampton MW, Utell MJ, Zareba W, et al. Ambient fine particulate air pollution triggers ST-elevation myocardial infarction, but not non-ST elevation myocardial infarction: a case-crossover study. Part Fibre Toxicol. 2014 Jan 2;11:1. https://doi.org/10.1186/1743-8977-11-1
32. Pope CA, Muhlestein JB, Anderson JL, Cannon JB, Hales NM, Meredith KG, et al. Short-Term Exposure to Fine Particulate Matter Air Pollution Is Preferentially Associated With the Risk of ST-Segment Elevation Acute Coronary Events. J Am Heart Assoc. 2015 Dec 8;4(12):e002506. https://doi.org/10.1161/jaha.115.002506
33. Kuźma Ł, Dąbrowski EJ, Kurasz A, Święczkowski M, Jemielita P, Kowalewski M, et al. Effect of air pollution exposure on risk of acute coronary syndromes in Poland: a nationwide population-based study (EP-PARTICLES study). Lancet Reg Health Eur. 2024 Apr 22;41:100910. https://doi.org/10.1016/j.lanepe.2024.100910
34. Ferguson T, Curtis R, Fraysse F, Olds T, Dumuid D, Brown W, et al. Weather associations with physical activity, sedentary behaviour and sleep patterns of Australian adults: a longitudinal study with implications for climate change. Int J Behav Nutr Phys Act. 2023 Mar 14;20(1):30. https://doi.org/10.1186/s12966-023-01414-4