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Temporal Analysis of Air Pollution Effects on Cardiovascular Diseases and Mortality in Ahvaz, Iran

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Abstract

In recent decades, air pollution has emerged as a significant environmental concern in major urban centers across Iran, exerting a profound impact on public health. This article aims to investigate the temporal correlation between air pollution and the incidence of cardiovascular disease and mortality. Utilizing secondary analysis of environmental data, hospital records, and mortality registries from Ahvaz, Iran, in 2016, our study elucidates these relationships. Our findings reveal a heightened susceptibility among men to both cardiovascular disease onset and mortality compared to women, with individuals aged 55-64 exhibiting increased vulnerability to cardiovascular diseases. The results of multivariate analysis indicate that the optimal time model, explaining 60% of the variance, is observed 23 days after pollutant emission for cardiovascular disease incidence. Conversely, for cardiovascular mortality, the most effective time model, explaining 6.5 percent of the variance, aligns with elevated air pollution levels on the same day. Notably, various pollutants, including toluene, carbon monoxide, nitrogen dioxide, sulfur dioxide, PM2.5, and PM10, exhibit distinct temporal patterns, significantly influencing cardiovascular diseases. Moreover, toluene, benzene, nitrogen dioxide, sulfur dioxide, and PM10 emerge as primary contributors to cardiovascular mortality, each demonstrating unique temporal effects. Our study further delves into the emergence of time-delayed and oscillatory patterns in the impact of air pollution on cardiovascular diseases and mortality. This study highlights the necessity of evaluating the efficacy of individual pollutants separately. Consequently, the development of effective policies and programs aimed at mitigating pollutants and their temporal repercussions on health is crucial for curtailing and managing the health complications associated with cardiovascular diseases.

Keywords: Air Pollution; Cardiovascular Diseases; Temporal Effect; Ahvaz

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INTRODUCTION

Air pollution has become a prominent issue in contemporary discourse, garnering significant attention. It is defined as the presence of various pollutants in the atmosphere, reaching levels that pose harm to humans, animals, plants, and property (Manisalidis et al., 2020; WHO, 2020). During the epidemiological transition, the harmful effects of particulate matter on health, including increased mortality and the prevalence of respiratory and cardiovascular diseases, have become increasingly apparent. According to the World Health Organization (2019), it is expected that two million premature deaths worldwide will be attributed to air pollution. Reductions in particulate matter levels, ranging from 20 to 70 $\mu g/m^3$, could lead to a decrease in air pollution-related mortality by up to 15% (WHO, 2020). Additionally, research

indicates that one out of every 50 cases of cardiac disease treated in London hospitals is linked to air pollution (Routledge et al., 2003). The World Health Report (2014) reports that air pollutants stand as the primary cause of respiratory infections, with undeniable associations with cardiac and pulmonary failures resulting in additional cases and fatalities (WHO, 2014).

Factors contributing to the degradation of air quality include the escalating urbanization trend and the consequent rise in air pollutants stemming from excessive industrial activities, chemical usage, and vehicular emissions (Guo et al., 2017; Hou et al., 2010; Kelishadi & Poursafa, 2010; Mannucci & Franchini, 2017). Additionally, the construction of unregulated dams (Routledge et al., 2003), conversion of agricultural lands into salt marshes, and deforestation exacerbate this issue (Uzoigwe et al., 2013). Furthermore, the pollution stemming from oil and gas operations, compounded by the effects of climate change (Moore, 2009; USGCRP, 2009), collectively contribute to the deteriorating air quality in Ahvaz, rendering it an unhealthy environment.

This situation leads to many consequences, ranging from chronic fatigue and lethargy to symptoms such as headaches, nausea, anxiety, stress, and, most gravely, mortality. Non-communicable diseases, including respiratory diseases, gastrointestinal disorders, nervous system dysfunctions, cancers, tumors, and cardiovascular diseases, are witnessing a surge in prevalence. In 2016 alone, cardiovascular diseases (n=921), hypertension (n=518), diabetes (n=463), cancers (n=415), respiratory diseases (n=365), lung diseases (n=304), gastrointestinal diseases (n=18), and toxication (n=17) contributed to fatalities among the citizens of Ahvaz (Mirzaie et al., 2020). This marked 30% increase in disease incidence constitutes just one facet of the alarming health risks facing the population. Notably, the frequency of hypertension and heart attack cases presenting to urban emergency services on days with poor air quality has witnessed a significant surge (Hosseini & Shahbazi, 2016; WHO, 2020). Consequently, air pollution emerges as one of the most pressing issues in the metropolis of Ahvaz (Bazgeer et al., 2015; WHO, 2020).

Hence, the article seeks to examine the temporal impact of air pollution on cardiovascular disease incidence and mortality, employing secondary analysis of environmental and hospital datasets from Ahvaz throughout the year 2016.

LITERATURE REVIEW

The article employs several theoretical frameworks, including the Epidemiological Transition Theory, Kuznets Environmental Theory, Health Disorder Theory, and Self-Care Theory, to elucidate the relationship between air pollution and cardiovascular diseases. The Epidemiological Transition Theory posits the shifting patterns of mortality from infectious to non-communicable diseases, prominently cardiovascular diseases, cancers, skin, respiratory, gastrointestinal, and infectious diseases (Abbasi-Shavazi et al., 2005; Caselli et al., 2002; McKeown, 2009; Omran, 2005). Given the significant impact of air pollution on these conditions, a close association is anticipated between the epidemiological transition and air pollution.

In 1991, economists identified an asymmetric relationship between income fluctuations and environmental quality, leading to the formulation of the Environmental Kuznets Curve. This theory posits that population growth contributes to an economic imbalance during the regional development process (Li et al., 2020; Sadeghi, 2018), consequently resulting in increased environmental pollution stemming from elevated per capita income derived from oil and gas products.

According to this theory, a portion of this environmental pollution diminishes with enhanced budget allocations and the advancement of new technologies subsequent to achieving high levels of per capita income and development (Grossman & Krueger, 1991; Heerink et al., 2001; Holtz-Eakin & Selden, 1995).

The transition in mortality rates, and consequently, the epidemiological transition and shifts in the causes of mortality, have introduced new and intricate dimensions to morbidity in modern times. Renowned theorists such as Murray and Chen (1996) have defined morbidity as encompassing the characteristics and symptoms associated with disease— encompassing illness, disability, and impairment (Hernandez & Kim, 2022). In this context, Northrup (1986) observed that morbidity is more prevalent than mortality and is particularly sensitive to environmental shifts and access to healthcare. Chronic diseases, prevalent in contemporary society, contribute significantly to health disorders. Consequently, reducing morbidity stands as a paramount developmental objective for enhancing quality of life (Northrup, 1986).

Self-care constitutes a fundamental component of health-promoting behaviors. These behaviors encompass any actions undertaken to enhance and sustain the health and well-being of individuals or groups. Emphasizing positive lifestyle choices and health-promoting behaviors aims to improve health outcomes and overall quality of life (Callaghan, 2003; McKeown, 2009). Table 1 provides an overview of prior studies and their significant findings pertaining to the subject under investigation.

Table 1. Prior Research on the Impact of Air Pollution on Cardiovascular Diseases and Mortality

Author/s	Year	Research methods	Topics	Key results
Routledge, Ayres, and Townend	2003	Meta-analysis	Why cardiologists should be interested in air pollution	The population at risk and the number of deaths attributable to cardiovascular diseases outweigh those of other health conditions. While effect sizes may exhibit significant variability across studies, meta-analyses of time series data indicate that a rise in fine particulate pollution by 10 µg/m3 correlates with a 1.8% increase in total mortality and approximately a 1.4% rise in cardiovascular mortality
Chen, Huang and Wang	2009	Panel	The Impact of Air Pollution on Human Health in China	Analyzing the impact of air pollution on human health before and after 2004, researchers discovered that the mortality elasticity attributable to air pollution increased by 23% compared to pre-2004 levels.
Mirzaii & Morshedi	2011	Survey	Referring to hospital emergencies with	A noteworthy correlation exists among dust levels, the

Author/s	Year	Research methods	Topics	Key results
			changing air pollutants	prevalence of respiratory diseases, climate variations, and seasonal patterns throughout the year.
Fattore et al.	2011	Panel	Risks exposed to human health concerning air cleanliness in Italy	Pollutants such as PM2.5, PM10, ozone, and nitrogen dioxide have undergone assessment, revealing PM2.5 to exert the most significant and immediate health impacts on Italian citizens. Additionally, ground-level ozone and nitrogen dioxide contribute to elevated mortality rates.
Sousa et al.	2012	Panel	The effect of air pollution on the incidence of respiratory diseases in Portugal	A correlation exists between asthma admission rates and air pollutant particles, particularly evident on days with heightened air pollution levels.
Kim et al.	2015	meta-analysis	The effect of airborne particles on human health	Airborne particulate matter, or PM, is implicated in the escalation of disease and the reduction of life expectancy. Additionally, particle size correlates with the susceptibility to infection.
Manisalidis et al.	2020	meta-analysis	Environmental and Health Impacts of Air Pollution: A Review	PM, comprising particles of variable but minuscule diameter, infiltrates the respiratory system through inhalation, thereby instigating respiratory and cardiovascular diseases, impairments in the reproductive and central nervous systems, and cancer. Additionally, nitrogen oxide, sulfur dioxide, volatile organic compounds (VOCs), dioxins, and polycyclic aromatic hydrocarbons (PAHs) are recognized as harmful air pollutants with deleterious effects on human health.

The studies addressing air pollution and its consequences exhibit certain gaps and weaknesses. For instance, prior research has predominantly focused on exploring the health effects stemming from air pollution emissions or those occurring solely within one day following air pollution emissions. However, in the current study, we aim to investigate the impact of individual pollutants on cardiovascular diseases over 30 days.

DATA AND METHOD

Data

The population under investigation in this study comprises all residents of Ahvaz who experienced health disorders attributed to air pollution or were referred to hospitals in Ahvaz for cardiovascular diseases and cardiovascular-related deaths throughout the year 2016.

According to Figure 1 the study utilized secondary data obtained from various data sources. Information on cardiovascular diseases and cardiovascular-related deaths was sourced from hospitals, the Behesht Zahra Organization, and the Ahvaz Environmental Organization. Health disorder data were collected through specific hospital forms, capturing details such as hospitalization date, reason for referral, age, and gender of each patient. Data from a subset of hospitals in Ahvaz, including Imam Khomeini, Golestan, Baqaei, Razi, and Shafa Hospitals, were analyzed. Daily air pollution and mortality data were acquired from three pollutant concentration stations, namely the General Administration, Naderi, and Ein Do stations. Prior to utilization, the air pollution data underwent validation and classification processes.

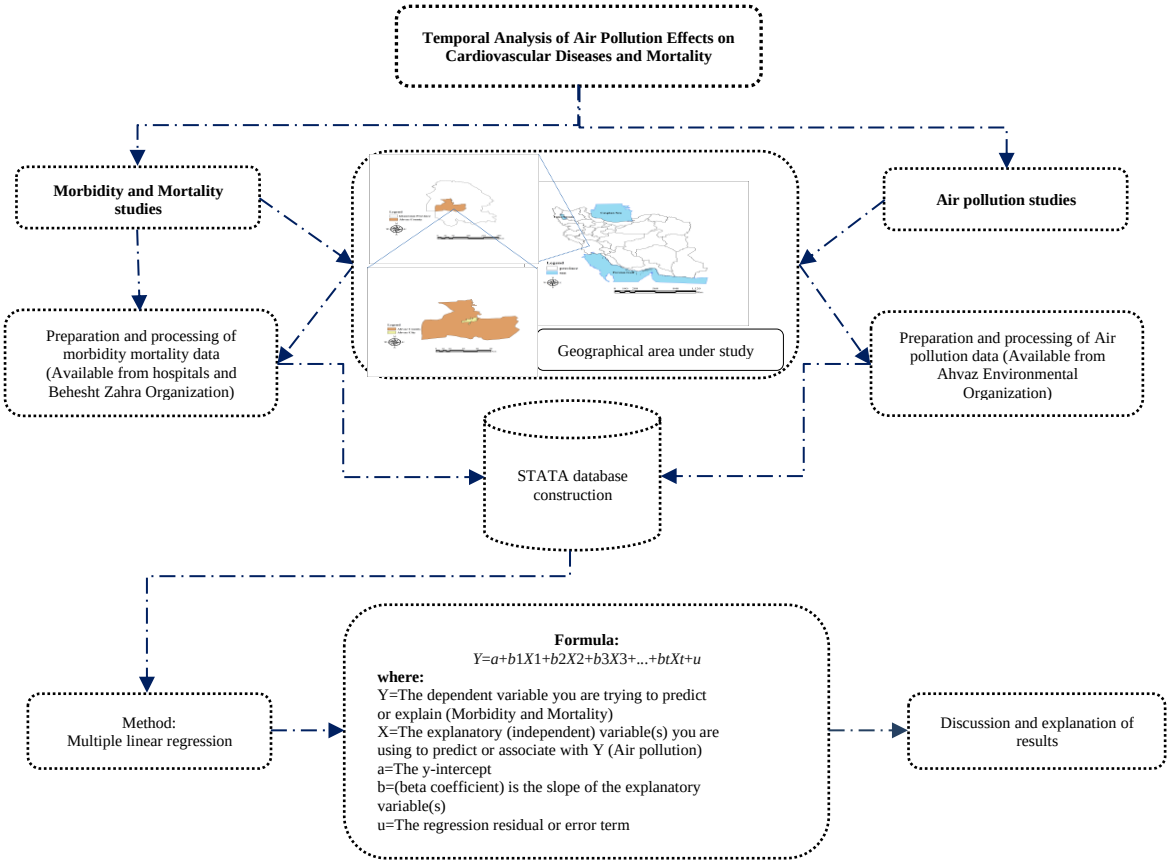


Figure 1. Flowchart of the study

Analytical Techniques

The study's independent variables encompass air pollutants, while the dependent variables include health disorders attributed to cardiovascular diseases and cardiovascular-related deaths, respectively. Data were collected, sorted, categorized, and analyzed using Microsoft Excel and STATA 16 software. Subsequently, the collected data will be subjected to multiple regression analysis matrices for final analysis (See Figure 1).

RESULT

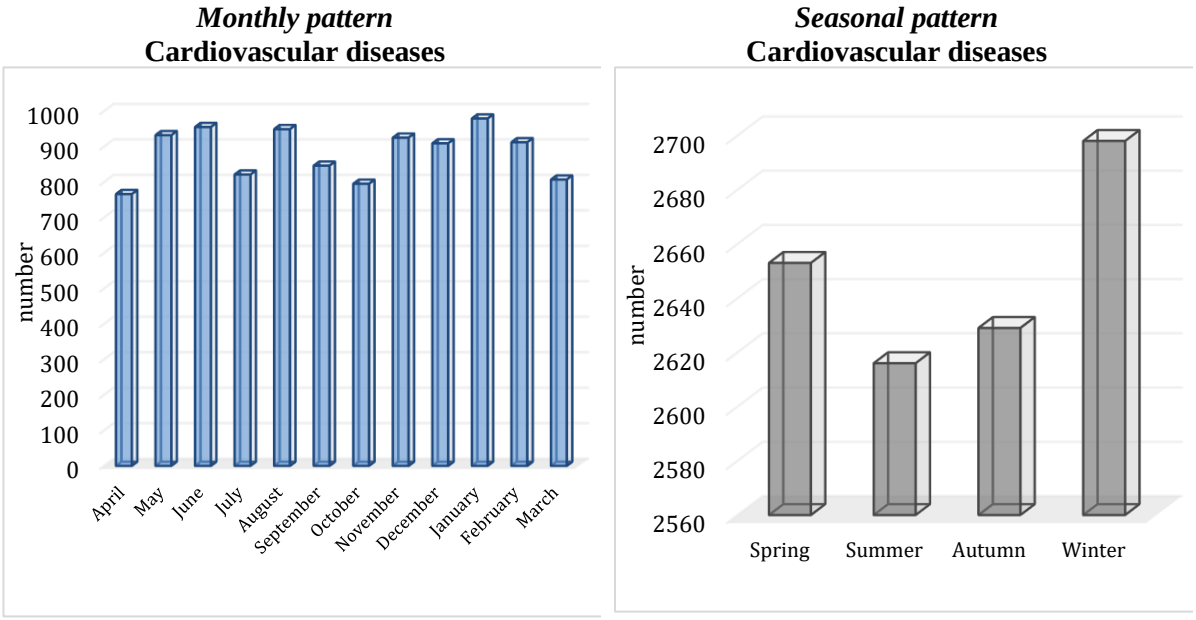
Description

Table 2 presents the daily reported data, featuring general information and key statistics such as mean, standard deviation, and lower and upper limits. The first ten rows delineate variables related to air pollution components. The last two columns provide an overview of hospital visits and cardiovascular-related deaths indicative of health disorders stemming from cardiovascular diseases. On average, 47 individuals were referred to the hospital due to cardiovascular disease, while 2.505 deaths occurred daily.

Table 2. Description of variables used in the study, 2016

Variables	Observation (in a year)	Average	Standard deviation	Minimum	Maximum
O3	365	17.45	9.25	0.3	58.33
NO	365	11.59	8.43	1.5	48.83
NO2	365	18.73	7.93	2.4	53
NOX	365	30.41	15.72	10	141
SO2	365	20.32	7.82	7	65
CO	365	1.52	0.77	22	4.14
PM2.5	365	56.20	31.95	14	274
PM10	365	167.18	0.13	43	362
BEN	365	0.29	0.03	0.08	0.85
TOLL	365	0.041	20.74	0.01	0.177
Cardiovascular Diseases	365	47.61	20.74	12	186
Cardiovascular Mortality	365	2.505	1.782	0	9

Figure 2 depicts the highest number of referrals for cardiovascular disease occurring in January, while cardiovascular deaths peak in December. Furthermore, as illustrated in the diagram, the incidence of cardiovascular disease and cardiovascular-related deaths in Ahwaz reaches its zenith during the winter months.



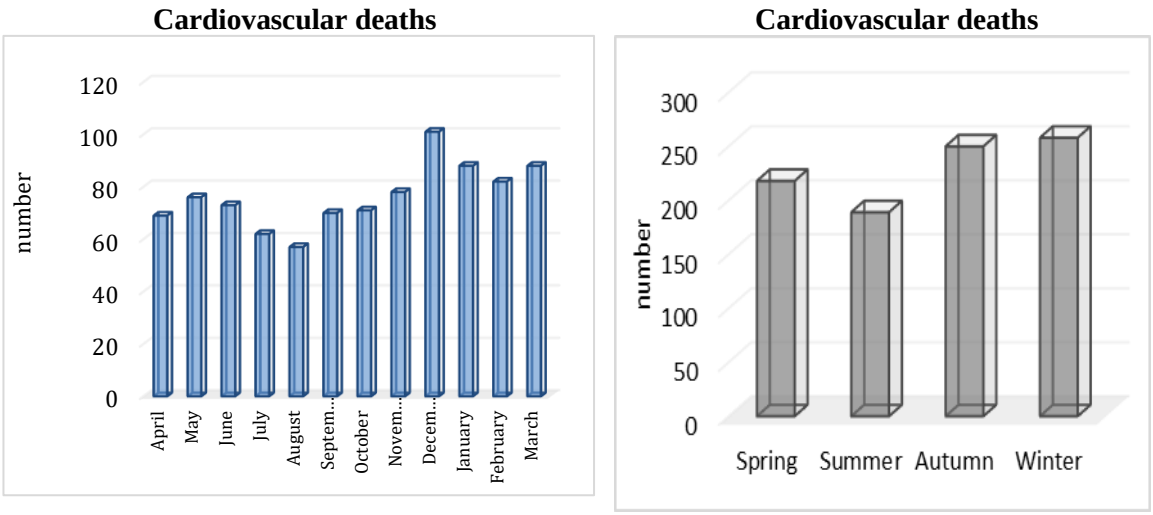


Figure 2. Seasonal and monthly distribution of cardiovascular mortality and people referring to hospitals due to cardiovascular diseases in Ahvaz, 2016

Figure 3 illustrates the mortality rates and the number of hospital referrals for cardiovascular disease segmented by age and gender in 2016. The highest frequency was observed among individuals referred to hospitals due to cardiovascular diseases (n=10596). Men exhibited a higher likelihood of hospital referrals compared to women, a trend mirrored in mortality rates. Additionally, cardiovascular diseases accounted for the highest number of referrals among individuals aged 55-64 years old (n=5085). The mortality pattern displayed an association with advancing age.

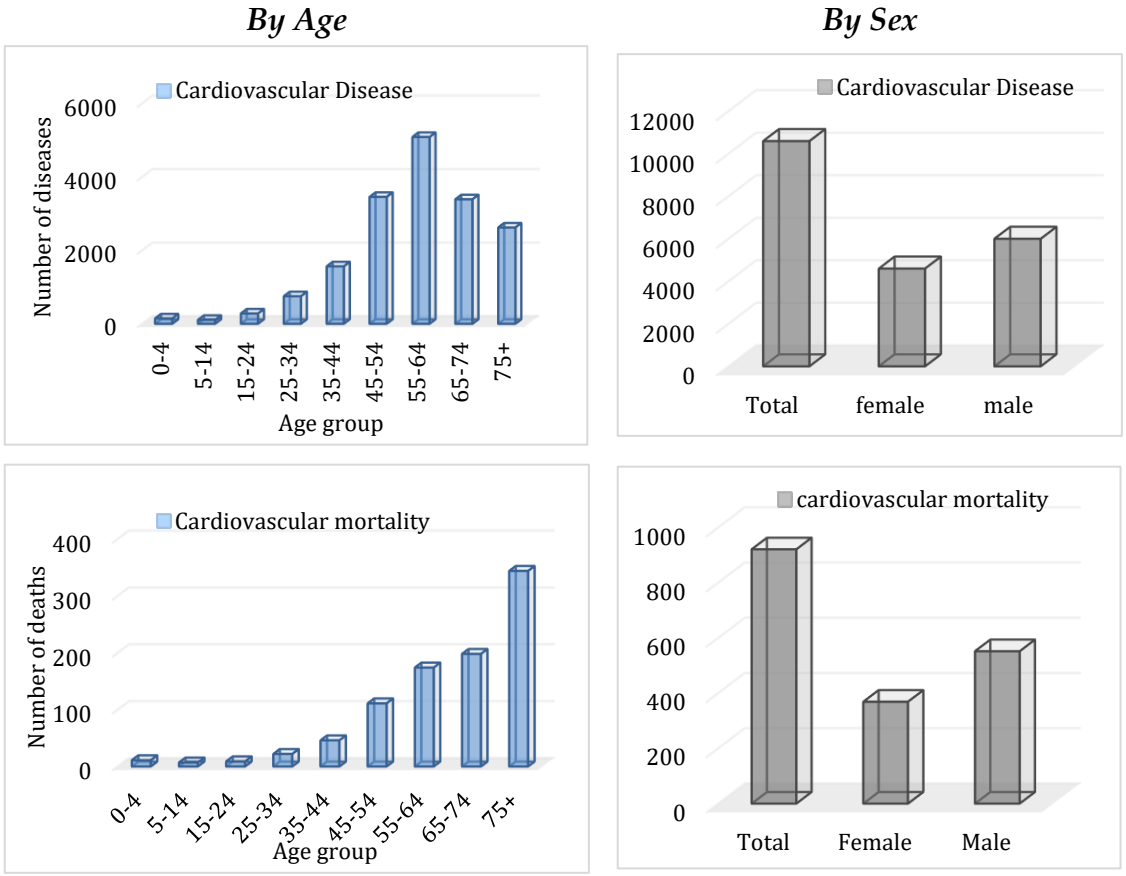


Figure 3. Age and sex distribution of referring to hospitals due to cardiovascular diseases and cardiovascular mortality in Ahvaz, 2016

Multivariable Analysis

Using multivariate regression analysis, we explored the relationship between air pollution, cardiovascular diseases, and mortality. Table 3 presents the findings of 62 regression models, juxtaposed to provide comprehensive insights. Notably, Table 4 complements these results with a descriptive integration due to substantial overlap. The analysis involves two primary pollutants, PM10 and PM2.5, treated as a standardized grouping to facilitate a deeper understanding of the effects of air pollution. Specifically, PM2.5 demonstrates a significant impact on cardiovascular disease incidence on the emission day, resulting in an increase of 0.099 hospital referrals for cardiovascular disease per unit. To present this impact using the standard grouping of the air quality index, we find that for each unit increase in PM2.5 standard, an average of 1.44 individuals are referred to hospitals due to cardiovascular disease on the day of emission.

PM10 pollutants, exhibiting a comparable effect to PM2.5, manifest distinct impact times yet wield greater potency than other variables. Analysis indicates the initial significant effect of PM10 23 day's post-pollutant emission. Consequently, a unit increase in this pollutant correlates with 0.090 hospitalizations for cardiovascular disease. In standardized terms, for each 50-point increase per unit in PM10, an average of 1.45 individuals are expected to seek hospitalization in Ahvaz 23 days following air pollution emission.

Table 3. The relationship between air pollution indicators and cardiovascular diseases and mortality

cardiovascular diseases

Independent variables	Symbol of variables	Air pollution day	1 days after air pollution	3 days after air pollution	4 days after air pollution	5 days after air pollution	6 days after air pollution	7 days after air pollution	8 days after air pollution	9 days after air pollution	10 days after air pollution	11 days after air pollution	12 days after air pollution	13 days after air pollution	14 days after air pollution	15 days after air pollution
		coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff
NO	V1	-	0.185	-	-	-	-	0.448	0.133	-	-	0.0449	0.126	0.194	0.068	0.329
NO2	V2	0.0126	0.350	-	-	-	0.0032	0.232	-	-	-	-	0.127	-	-	0.345
NOX	V3	0.014	-	0.489	0.576***	0.198	-	-	0.034	0.560	0.0205	-	-	0.058	0.111	-
SO2	V4	0.032	-	0.128	0.288	0.032	0.030	0.130	0.120	-	-	0326***	0.110	0.135	0.158	-
CO	V5	2.18	4.475**	2.11	-	0.340	0.145	1.15	1.460	2.06	-	-	2.676	-	2.02	1.778
PM2/5	V6	0.099***	0.065	0.0021	-	0.050	-	-	0.0025	-	0.0139	-	-	-	-	-
PM10	V7	-	-	-	0.006	-	0.0022	-	-	-	-	-	0.0002	-	-	0.0026
Benzene	V8	-	-	1.55	30.59	8.385	21.71	-	16.23	-	2.763	13.555	7.024	21.218	-	-
Toulon	V9	131	134.10	45.26	18.98	71.72	-	101.98	-	88.857	99.980	74.899	-	-	95.866	40.106
Modeling	R-squared	0.285	0.446	0.382	0.426	0.184	0.085	0.282	0.216	0.293	0.124	0.372	0.125	0.183	0.207	0.105

Independent variables	Symbol of variables	16 days after air pollution	17 days after air pollution	18 days after air pollution	19 days after air pollution	20 days after air pollution	21 days after air pollution	22 days after air pollution	23 days after air pollution	24 days after air pollution	25 days after air pollution	26 days after air pollution
		coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff
NO	V1	0.144	-	-	-	0.167	0.122	0.191	-	-	-	-
NO2	V2	0.208	0.462	0.0024	-	0.203	0.0674	0.270	-	0.0268	0.409	0.410
NOX	V3	-	-	0.0890	0.206	-	-	-	0.0912	0.1673	-	0.0188
SO2	V4	-	-	-	0.0545	-	0.141	0.214	-	0.0507	-	0.0171
CO	V5	0.586	2.743	0.847	2.009	1.458	2.307	5.155 *	2.118	3.358***	0.784	3.842***
PM2/5	V6	-	0.0516	0.023	0.0546	0.051	0.0304	0.0602	-	0.0293	0.049	0.0208
PM10	V7	0.0055	-	0.0028	-	-	0.0004	-	0.0290*	0.0004	-	-
Benzene	V8	30.718	-	3.386	-	-	-	-	28.142	-	33.763	-
Toulon	V9	-	47.589	46.653	-	39.958	93.944	-	-	201.0***	-	23.17
Modeling	R-squared	0.118	0.207	0.127	0.161	0.156	0.181	0.465	0.596	0.356	0.345	0.302

cardiovascular mortality

Independent variables	Symbol of variables	Air pollution day	1 days after air pollution	3 days after air pollution	4 days after air pollution	5 days after air pollution	6 days after air pollution	7 days after air pollution	8 days after air pollution	9 days after air pollution	10 days after air pollution	11 days after air pollution	12 days after air pollution	13 days after air pollution	14 days after air pollution	15 days after air pollution
		coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff
NO	V1	-	-	-	0.0501	-	0.0188	0.0054	0.0119	0.0282	0.0569	-	0.0161	0.0076	-	-
NO2	V2	-	-	0.0061	0.174	-	-	-	0.0075	-	0.0434	0.0046	0.0081	0.0060	-	-
NOX	V3	0.024	0.0093	0.0023	-	-	-	-	-	-	-	-	-	-	0.111	0.0113
SO2	V4	0.0014	-	-	-	-	-	-	-	-	-	-	-	-	-	0.0108
CO	V5	0.489	0.0778	-	-	-	-	-	0.0537	0.0604	0.1485	0.0650	0.1294	0.1383	0.142	0.0124
PM2/5	V6	-	-	-	-	-	-	0.00422	0.0027	0.0014	0.0015	-	0.00003	-	-	-
PM10	V7	-	0.00097	0.0013**	0.00005	0.00025	0.00075	0.00004	-	0.0005	-	-	0.00061	0.00042	-	0.00013
Benzene	V8	2.989	2.668	2.207	3.917***	5.359 *	2.173	0.3127	0.0968	-	1.739	2.267	-	-	-	-
Toulon	V9	-	-	-	-	-	0.0666	10.285	4.976	0.5978	-	5.112	11.267	6.537	11.715	12.931
Modeling	R-squared	0.0226	0.0229	0.0365	0.0518	0.0435	0.0319	0.0380	0.0279	0.0246	0.0388	0.0544	0.0467	0.0289	0.0166	0.0370

Independent variables	Symbol of variables	16 days after air pollution	17 days after air pollution	18 days after air pollution	19 days after air pollution	20 days after air pollution	21 days after air pollution	22 days after air pollution	23 days after air pollution	24 days after air pollution	25 days after air pollution	26 days after air pollution	27 days after air pollution	28 days after air pollution	29 days after air pollution	30 days after air pollution
		coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff	coff
NO	V1	0.0287	0.010	-	0.0129	-	0.00196	0.0566	-	0.0375	0.0832**	0.0252	0.0175	0.0072	0.0098	-
NO2	V2	-	-	-	0.0029	-	0.0118	0.0491	-	0.0269	0.0390	0.0144	0.0196	0.0103	-	-
NOX	V3	0.0129	0.0253	0.0197	-	0.0127	-	-	0.0227	-	-	-	-	-	0.00030	0.0369
SO2	V4	-	0.0147	0.0143	-	0.0275***	0.0291***	-	0.0043	-	-	-	-	0.0104	-	0.00068
CO	V5	0.0668	0.0834	0.1307	0.2527	0.1021	0.2432	0.4264*	0.0229	0.0446	0.1477	0.0839	-	0.00013	0.1003	0.1232
PM2/5	V6	-	-	-	-	0.00315	-	-	-	-	-	-	-	-	-	0.0038
PM10	V7	0.00031	-	-	0.00048	-	-	0.00046	-	0.00013	0.00068	0.0011***	0.00018	0.00075	0.00197*	-
Benzene	V8	3.4936	1.9485	1.5307	1.4643	-	-	-	-	1.5609	2.2263	4.835*	3.504***	1.2655	0.3441	14.81
Toulon	V9	-	-	-	-	-	3.157	-	1.2293	4.1996	-	-	-	-	2.6028	20.405**
Modeling	R-squared	0.0651	0.0559	0.0329	0.0197	0.0321	0.0281	0.0358	0.0174	0.0395	0.0492	0.0512	0.0333	0.0196	00512.	0.0446

In cardiovascular diseases, carbon monoxide pollutants hold particular significance due to their manifestation of a wave pattern encompassing both short-term and long-term effects. Notably, this pollutant exhibits a significant short-term effect one day after emission and a significant long-term effect on days 22, 24, 26, and 27 following emission. Conversely, in the context of cardiovascular death, pollutants such as PM10 and benzene display a similar wave pattern, featuring both short-term and long-term effects. PM10 pollutants demonstrate a significant short-term effect three days after emission, followed by significant long-term effects on days 26 and 29 post-emission. Benzene pollutants, on the other hand, exhibit a significant short-term effect on days 4 and 5 post-emission, with significant long-term effects observed on days 26 and 27 following emission.

Nitrogen dioxide exhibits a notable impact on the health of 0.576 individuals due to cardiovascular disease four days post-emission per unit increase, yet does not influence cardiovascular death. Similarly, SO_2 pollutants influence the health of 0.326 individuals approximately 11 days post-emission while affecting mortality between days 19 and 23 following emission. Conversely, toluene, characterized by the lowest amplitude of fluctuation, can influence the health of 6.6 individuals 24 days post-emission, subsequently impacting mortality 30 days after emission. Additionally, NO pollutants, which initially do not affect cardiovascular disease, can influence cardiovascular mortality 25 days following emission.

Moreover, Figure 4 illustrates that 23 days post-pollutant emission, the PM10 index exhibits the highest explanatory power of the dependent variable, accounting for approximately 60% of the variance. This is attributed to its extensive oscillation

range and significant effects on cardiovascular diseases. Additionally, the pattern observed for cardiovascular mortality demonstrates a short-term impact (R2=0.0651 on the day of air pollution), whereas for cardiovascular diseases, the impact is long-term (R2=0.59 for 23 days after emission).

Table 4. Time pattern of the effect of each pollutant on cardiovascular diseases and mortality

Independent variables	Cardiovascular Diseases	Cardiovascular Mortality
NO	-	25 days after air emission
NO2	-	-
NOX	4 days after air emission	-
SO2	11 days after air emission	20, 21 days after air emission
CO	1, 22, 24, 26, 27 days after air emission	19, 22 days after air emission
PM2/5	Air pollution day	-
PM10	23 days after air emission	3, 26, 29 days after air emission
Benzene	-	4, 5, 26, 27 days after air emission
Toulon	24 days after air emission	30 days after air emission

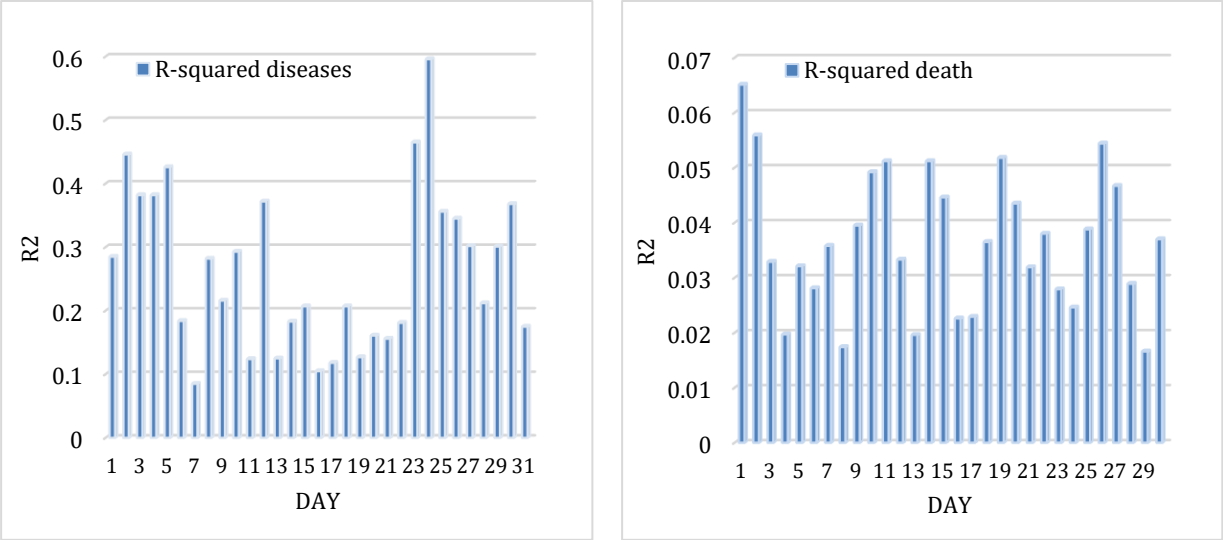


Figure 4. Fitting the model of the temporal effect of air pollutions on cardiovascular diseases and mortality

Discussion

Air pollution has emerged as an important concern significantly impacting human lives. The primary objective of this study is to investigate the temporal effectiveness of pollutants. Previous research has primarily focused on the daily impact of air pollution, neglecting discussions on its long-term effects. Hence, this study endeavored to measure the 30-day impact of pollutants through statistical estimation while also exploring their daily effects. The findings highlight a surge in hospital referrals for cardiovascular disease on days characterized by poor air quality. Consequently, there has been an alarming increase in non-communicable diseases

such as cardiovascular and respiratory diseases (Keates et al., 2017; Kraemer et al., 2016; Tolley et al., 2016).

Furthermore, the study findings revealed the statistically significant impact of PM2.5 pollutants on the day of emission. However, in terms of cardiovascular mortality, PM2.5 showed no effect, aligning with the results reported by Fatour et al. (2011) and Rajagopalan et al. (2018). Conversely, benzene exhibited both short-term (4 and 5 days post-emission) and long-term (26 and 27 days post-emission) effects on cardiovascular mortality while exerting no influence on cardiovascular diseases.

PM10 pollutants exhibit a robust and significant impact on health disorders attributable to cardiovascular diseases precisely 23 days following emission. This finding aligns with the results reported by Kim et al. (2016). While Fatour et al. (2011) did not observe a long-term effect of this variable, they confirmed its lack of significance in the short term (within one or two days post-emission). Furthermore, the results pertaining to cardiovascular mortality revealed a pattern of short-term impact (3 days post-emission) alongside long-term effects (26 and 29 days post-emission).

The CO pollutant exhibits a fluctuating pattern of impact, observed at 1 day, 22 days, 24 days, and 26 days post-emission, although the study did not confirm the emission. However, its one-day effect has been corroborated by O'Toole et al. (2009) and Lee et al. (2014). In the context of cardiovascular mortality, the results exclusively indicate a long-term effect (19 and 22 days post-emission). Exposure to CO has been implicated in the progression of atherosclerosis. Human studies have provided evidence suggesting that CO can exacerbate ischemic heart disease (Moran et al., 2010).

SO_2 pollutants exhibited a medium-term effect on cardiovascular diseases, evident 11 days post-emission. Conversely, in the context of cardiovascular mortality, the results indicated a long-term effect, observed 20 and 21 days post-emission. Interestingly, these findings diverge from those reported by Amancio and Nascimento (2012), which suggested a significant one-day effect of this variable.

NOX pollutants showed a four-day effect post-emission on cardiovascular diseases. Still, they did not have any effect on cardiovascular death, consistent with the lack of significance observed in previous studies. Toluene demonstrated a notable effect on cardiovascular diseases 24 days after emission and on cardiovascular death 30 days after emission. However, it is noteworthy that different results may arise over 30 days. Overall, several researchers (Goldberg et al., 2003; Hoek et al., 2013; Sousa et al., 2012) have confirmed a strong association between increased daily air pollution and daily health disorders. The results of the present study show the statistical association between the days following air pollution emission and health disorders, as well as mortality.

In the context of the epidemiological transition theory, observable shifts in the causes of death have occurred. Air pollution has emerged as a predominant contributor to mortality in Ahvaz, primarily attributed to cardiovascular diseases, respiratory issues, lung diseases, and, to some extent, cancers. These health outcomes are significantly influenced by particulate pollutants measuring less than 10 μm 2.5 μm, as well as carbon monoxide, sulfur dioxide, nitrogen dioxide, and toluene. Moreover, Kuznets's environmental theory has played a pivotal role in understanding health disorders and mortality. Ahvaz stands as one of the largest cities globally due to its abundant oil and gas fields.

Consequently, pollution stemming from oil and gas extraction activities, including oil flares surrounding the city, has profoundly impacted public health, notably through nitrogen dioxide pollutants directly linked to these operations. Furthermore, the theory of health disorders, a novel framework in health discourse, offers valuable insights into understanding the results concerning health outcomes. Within this theoretical framework, it becomes evident that air pollution has inflicted severe consequences on the health of Ahvaz residents, leading to increased hospital referrals and disruptions in daily activities.

CONCLUSION

The findings indicate that each pollutant triggers fluctuations in mortality rates and hospital admissions due to cardiovascular disease within distinct time frames. For instance, an increase in PM2.5 pollutant levels corresponds to an immediate rise in hospital admissions in Ahvaz on the same day. Conversely, for PM10 pollutants, hospital referrals increase approximately 23 days after emission. Furthermore, pollutants such as SO_2, CO, PM10, benzene, toluene, and NO exhibit long-term, delayed, and oscillating effects on cardiovascular mortality. Consequently, a temporal, delayed, and oscillating pattern emerges in discussions regarding the impact of air pollution on health disorders and mortality, given the unique time effects of each pollutant. Addressing and managing health disorders stemming from cardiovascular disease necessitates policy formulation and the implementation of effective programs aimed at reducing pollutants. Moreover, careful attention must be paid to the temporal effects of these pollutants on health outcomes.

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