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Air pollution and hospitalization due to angina pectoris in Tehran, Iran: A time-series study ☆

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Abstract

Background: Health effects of air pollution have been studied in many different parts of the world. Although a fairly large number of studies have explored the cardiovascular impacts of air pollution, because of its unique location we studied the association between air pollutants and hospital admissions due to angina pectoris in Tehran for the first time. *Methods*: This is a retrospective time-series study. The variables of the study include the levels of five air pollutants—nitrogen dioxide (NO_2), carbon monoxide (CO), ozone (O_3), sulfur dioxide (SO_2), and particulate matter $<10\,\mu m$ in aerodynamic diameter (PM_{10})—as independent variables; daily hospitalizations due to angina pectoris in 25 academic hospitals in Tehran as the dependent variable; and mean daily temperature and humidity, seasonality, time trend, and day of the week as potential confounders. All variables were measured during a 5-year period from 21 March 1996 to 20 March 2001. The data of mean daily levels of NO_2, CO, O_3, SO_2, and PM_{10} were collected from one of the stations of Tehran's Air Quality Control Corp. Data were analyzed using Poisson regression models. Relative risks of angina pectoris admissions were calculated for an increase in 1 mg/m^3 for CO and 10 μg/m^3 for the other pollutants. *Results*: Daily admissions due to angina pectoris were significantly related to the CO level, after controlling for confounder effects. Each unit increase in the CO level caused a 1.00934 increase in the number of admissions (95% CI, 1.00359–1.01512). This association was verified with a lag of 1 day. There was no significant association between the other air pollutants and the number of daily admission due to angina pectoris. *Conclusions*: We found that with increasing levels of the pollutant CO, the number of admissions due to cardiac angina rose. Ischemic heart disease is the leading cause of death in Iran. Air pollution control will reduce the number of this preventable disease and resulting deaths.

Keywords: Air pollution; Angina pectoris; Time series; Environmental epidemiology

1. Introduction

Associations between air pollution and health status have been identified most clearly in episodic situations during past decades. For example, in the early 1950s, a period of high pollution in London was associated with estimated excess deaths of over 4000 (Ministry of Health of Great Britain, 1954; Brunekreef and Holgate, 2002). This episode, as well as others in Europe and North

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America, has demonstrated that high concentrations of air pollution lead to an increase in mortality (Nemery et al., 2001). In later studies, investigators found that levels below even current air pollution guidelines are associated with adverse effects on health (Brunekreef and Holgate, 2002; Simpson et al., 1997; Anderson et al., 1996; Delfino et al., 1994; Yunesian, 2001; Zmirou et al., 1996; Dab et al., 1996; Ponce de Leon et al., 1996; Touloumi et al., 1996; Schwartz and Morris, 1995). Studies of the impact on mortality of air pollution have established an association with cardiovascular mortality; despite this, the short-term relationship between hospital admissions due to cardiovascular diseases and air pollution has just been assessed in recent years. Some studies of this connection have been completed recently in North and South America (Burnett et al., 1997a, b, 1999; Morris et al., 1995; Schwartz, 1997, 1999; Morris and Naumova, 1998; Lin et al., 2003), in Europe (D'Ippoliti et al., 2003; Ponka and Virtanen, 1996; Poloniecki et al., 1997; Wordley et al., 1997; Prescott et al., 1998; Ballester et al., 2001), in Australia (Petroeschevsky et al., 2001), and in Hong Kong (Wong et al., 1999). Some argue that it is not yet certain whether air pollution has cardiovascular effects (Mittleman and Verrier, 2003).

The issue of the cardiovascular impact of air pollution requires extensive attention for several reasons: (1) the association between even low levels of air pollution; (2) the high frequency of cardiovascular consequences associated with air pollution; (3) the ubiquity of exposure; and (4) the presence of susceptible groups in the population, such as the elderly, in whom the cardiovascular impact of air pollution increases due to the existing comorbidities (Petroeschevsky et al., 2001).

Up to now the cardiovascular effect of air pollution has not been assessed in Tehran, the capital of Iran. Motor vehicles are responsible for the majority of the air pollution in Tehran. They are the major sources of CO, NO_2, and PM_{10}. Tehran is not located next to a river or even close to a sea. Mountains surround the city in the north and east. Meteorological parameters such as mean temperature and sunshine are higher than those of most other cities studied. For these reasons the results of other studies cannot be extrapolated to Tehran. We studied the short-term association between air pollution levels and angina pectoris hospital admissions in the city of Tehran for a 5-year period from 21 March 1996 to 20 March 2001.

2. Methods

This study was part of an ecological time-series project in which daily levels of air pollutants and emergency admissions for angina pectoris in the academic hospitals of Tehran were related while controlling for major confounding variables.

2.1. Data

2.1.1. Health measures

The age, gender, and date of admission of patients were obtained from the registry databases or records of 25 academic hospitals from all over Tehran according to a primary discharge diagnosis of angina pectoris or corresponding codes (i.e., 413 and I20) of the *Ninth Revision of the International Classification of Diseases* (ICD-9) and the *Tenth Revision of the International Classification of Diseases* (ICD-10).

2.1.2. Exposure data

The pollutants data, including the 24-h average levels for nitrogen dioxide (NO_2), carbon monoxide (CO), sulfur dioxide (SO_2), and particulate matter $<10\,\mu m$ in aerodynamic diameter (PM_{10}) and 8-h maximum levels for ozone (O_3), were obtained from the Tehran Air Quality Control Corp.

2.1.3. Data of control variables

The daily data for temperature and relative humidity were obtained from Tehran Air Quality Control Corp.

2.2. Analysis

The daily number of hospital admissions was used as the dependent variable in a Poisson regression model. Using Poisson regression, we analyzed data in the following steps (Schwartz et al., 1996): First, a core model was built for the dependent variable, controlling for the main confounding variables, including long-term trend, seasonality, temperature, humidity, and weekly fluctuations in admissions (using dummy variables for holidays, preholidays, postholidays, and the other days of the week). Second, the association with air pollutants was estimated examining the delayed relation up to the third lag of pollutants. Last, to analyze the independence of the associations, multipollutant models were also examined, and the pollutants that were not significant were excluded from the full model with a backward method. The effect of pollution was calculated using a relative risk (RR) corresponding to an increase in $1\,mg/m^3$ for CO and $10\,\mu g/m^3$ for the other pollutants.

3. Results

Table 1 shows the summary statistics of the health and environmental data in Tehran. The mean daily number of angina pectoris hospital admissions was 23.48.

Table 1
Summary of the number of hospital admissions due to angina pectoris, the concentrations of air pollutants, and meteorological variables, Tehran (1996–2001)

	Mean (SD)	Minimum	P25	P50	P75	Maximum	*n*
Angina admissions	23.48 (9.26)	0	17	24	30	60	1826
SO_2, 24 h ($\mu g/m^3$)	73.74 (33.30)	0.30	48.23	74.05	98.64	499.26	1716
NO_2, 24 h ($\mu g/m^3$)	60.01 (39.69)	0.30	29.39	47.42	84.55	324.78	1669
CO, 24 h (mg/m^3)	10.83 (4.51)	1.65	7.75	10.00	13.33	57.89	1713
O_3, 8 h ($\mu g/m^3$)	9.71 (9.50)	0.69	3.34	6.53	14.44	198.00	1717
PM_{10}, 24 h ($\mu g/m^3$)	97.81 (80.87)	12.26	70.46	90.99	113.75	2254.68	1716
Temperature (°C)	19.47 (9.44)	−2.13	10.84	20.42	28.35	35.75	1689
Relative humidity (%)	39.12 (17.38)	0	26.51	33.68	47.50	100	1692

n, number of days with valid observations.

Table 2
Partial correlations between each air pollutant level and the number of angina admissions at various lag times of from 0 to 3 days, conditional on confounding variables, including long-term trend, seasonality, temperature and humidity, day of week, and other air pollutant levels, Tehran (1996–2001)

	Lag 0	Lag 1	Lag 2	Lag 3
SO_2, 24 h	0.0266	0.0504	0.0326	0.0349
NO_2, 24 h	0.1010	0.1239	0.0854	0.0905
CO, 24 h	0.0737	0.1367	0.0255	0.0156
O_3, 8 h	−0.1405	−0.1585	−0.1164	−0.1153
PM_{10}, 24 h	0.0269	0.0270	−0.0097	−0.0050

Table 3
Association between air pollution and the number of angina admissions, Tehran (1996–2001)

Air pollutants	L	Angina admissions[a]	*P* value
SO_2, 24 h	1	0.99995 (0.99397; 1.00507)	0.861
NO_2, 24 h	1	1.00618 (1.00261; 1.00976)	0.001
CO, 24 h	1	1.00957 (1.00600; 1.01315)	0.000
O_3, 8 h	1	0.96396 (0.94256; 0.98582)	0.001
PM_{10}, 24 h	1	1.00451 (0.99998; 1.00906)	0.051

L, lag with the best fit.
[a]Results are expressed as the relative risk (and 95% CI) of angina admission for an increase in the pollutant levels of 10 $\mu g/m^3$ (1 mg/m^3 for CO).

Table 2 illustrates partial correlations between each of the air pollutant levels and the number of angina admissions (at various lag times of from 0 to 3 days), conditional on confounding variables, including long-term trend, seasonality, temperature and humidity, day of week; and other air pollutants levels. As shown in this table, the partial correlations between all air pollutants and the admissions have the highest value at a 1-day lag.

Table 3 shows the estimates for the association between each air pollutant and the number of hospital admissions due to angina in a "one-pollutant model". A significant association was found for 24-h NO_2, lag 1; 24-h CO, lag 1; and 8-h O_3, lag 1. A nearly significant association was observed for 24-h PM_{10}, lag 1.

NO_2, CO, O_3, and PM_{10} were included in a "multi-pollutant model". Twenty-four hour CO, lag 1 is the only air pollutant that had a significant association with angina hospital admissions, so that a rise in its level of 1 mg/m^3 was associated with an increment of 0.9% in the expected number of hospital admissions due to angina pectoris (RR of 1 mg/m^3, 1.00934; 95% CI, 1.00359–1.01512).

4. Discussion

For the first time, we studied the short-term relationship between air pollution and emergency hospital admissions for angina pectoris in Tehran. We found that although several pollutants were associated with admissions in the one-pollutant models, CO was the only pollutant correlated with this outcome in the multi-pollutant model. As expected, there was a great degree of correlation among the levels of air pollutants in this study. Due to colinearity, NO_2 and ozone became statistically insignificant and therefore were excluded from the multi-pollutant model.

Statistically, a 1-day lag was chosen using partial correlations between each air pollutant level and the number of angina admissions. We offer several rationale: there is a delay from the onset of an increase in the air pollutant level to the appearance of angina symptoms. Moreover, socioeconomic and cultural factors can affect the time between the onset of symptoms and the active solicitation of medical care. One of the characteristics of Iranian people is that they are patient and do not seek health care immediately. Economic issues are also important. In addition, some patients first visit physicians in private clinics or their offices and then are referred to hospitals for admission; the entire process delays hospital admission.

Results obtained from studies of the association between air pollution and hospital admissions for

cardiovascular diseases in various parts of the world differ. Our findings are consistent with the findings reported by Lin et al. (2003). They also reported that only CO had a statistically significant association with emergency room visits due to ischemic cardiovascular disease (i.e., angina pectoris and myocardial infarction).

In another study, Schwartz and Morris (1995) found an association between pollutants PM_{10} and CO and hospital admissions for cardiovascular diseases in Detroit, MI, USA. On the other hand, they found that SO_2 was the only pollutant associated with admissions due to ischemic heart disease in that study. In the study of air pollution and cardiovascular disease in Tucson, AZ, USA, Schwartz (1997) reported associations between cardiovascular admissions and PM_{10} and CO. In a study of air pollution and heart disease in eight US counties, Schwartz (1999) found associations between PM_{10} and CO and cardiovascular admissions.

Wong et al. (1999) found no association between any air pollutants and hospital admissions due to ischemic heart disease in Hong Kong. It should be noted that CO concentrations were not measured in this study. In the study of air pollution in Valencia, Spain (Ballester et al., 2001), SO_2 was associated with emergency hospital admissions for cardiovascular diseases, which is not consistent with our findings. However, Ballester et al. found an association between CO and cardiovascular admissions in that study. In the study of air pollution and myocardial infarction in Rome, Italy, D'Ippoliti et al. (2003) reported a positive and significant association between PM_{10}, CO, and NO_2 and admissions due to acute myocardial infarction.

Petroeschevsky et al. (2001) examined associations between outdoor air pollution and hospital admissions in Brisbane, Australia. They found no significant association between air pollutants and cardiovascular admissions. In Canada the association between air pollution and admissions for cardiovascular disease has been studied by Burnett et al. Their findings established a strong association between O_3 and cardiovascular admissions and only a weak association with CO in Toronto (Burnett et al., 1997a). These findings are not consistent with our results. However, in another study of 10 Canadian cities, they found a strong association between CO and admissions for cardiovascular disease (Burnett et al., 1997b). The weak association in the Toronto study is thought to be due to low levels of CO during the period of study.

As we briefly mentioned above, most of the previous studies on air pollution and cardiovascular admissions report different results regarding individual pollutants. CO is the only pollutant associated with heart disease admissions in all of these papers, including our paper.

There has been a great advance concerning the possible biologic mechanism of cardiovascular impairments due to air pollution in recent years. Bridge and tunnel officers who were occupationally exposed to high levels of CO had a higher risk of developing arteriosclerotic heart disease (Stern et al., 1988). Allred et al. (1989, 1991) found that even exposure to low doses of CO can produce significant effects on cardiac function during exercise in subjects with coronary artery disease. In addition, there is evidence that high concentrations of SO_2, total suspended particulates, or CO are associated with an increase in heart rate (Peters et al., 1999).

This study has several limitations. Like other ecological studies, a major limitation of this study is a lack of precise estimation of exposure. Thus, caution should be taken in interpreting any cause-and-effect relations. Another limitation of this retrospective study was that it used data from hospital databases and records designed for other means. In some cases there was a lack of fulfillment of the date variable. In addition, the diagnosis registration systems were different among hospitals studied, so a misclassification bias in the diagnosis of angina was likely. This seems to be nondifferential and not to be connected with daily levels of air pollutants.

In a study of assessing diagnostic classification in an emergency department in Canada, Stieb et al. (1998) showed no evidence of diagnostic bias in relation to daily air pollution level. Our quality-control system of hospital data collection showed that the quality of collection was above the acceptable level. We monitored and improved the process of hospital data collection using a "lot quality-assurance sampling" method; i.e., the data were recollected for those hospitals that were found to have an insufficient quality of data collection—according to coverage and correctness of data collection—in order to make sure that they reached the expected level of quality of collection (unpublished data).

There are several stations for the measurement of air pollution in Tehran, but we used data from the only source that was consistently active during the study period. As shown in previous studies (Wordley et al., 1997), there may be significant differences between air pollution levels measured at different stations, but there is usually consistency between their fluctuations, and we can obtain data from one station as a proxy of the air pollution changes required for the time-series studies.

In conclusion, this study adds to our knowledge of the impact of air pollution on ischemic heart diseases, the leading cause of death in Iran. Many studies carried out in other parts of the world have demonstrated the adverse effects of air pollution on human health. These works have many implications for the redesigning of public health policy regarding air pollution in Iran and elsewhere.

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