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Effect of short-term fluctuations in outdoor air pollution on the number of hospital admissions due to acute myocardial infarction among inhabitants of Krakow, Poland

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Short title: Air pollution and hospitalisation for MI

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Conflict of interest: none declared.
Abstract

Introduction: Air pollution is reaching alarming proportions worldwide; however, previous studies concerning the association between air pollution and myocardial infarction (MI) provided conflicting results.

Objectives: We evaluated a relationship between short-term fluctuations in outdoor particulate matter (PM) and nitrogen dioxide (NO₂) levels and the number of hospitalisations due to MI among the inhabitants of Krakow, Poland.

Patients and methods: Data on hospitalisations, daily pollutant concentrations, infections, and meteorological parameters were collected from December 2012 to September 2015. Data were assessed using a time-series regression analysis with a distributed lag model.

Results: An increase of 10 µg/m³ in PM₂.₅ levels was associated with a higher risk of hospital admission due to MI: odds ratio (OR) 1.32; 95% CI: 1.01-1.40; P=0.0002. For PM₁₀ the effect was observed only with a simultaneous decrease of 1°C in the mean daily temperature: OR 1.08; 95% CI: 1.01–1.17; P=0.03. The significant effects were observed at lags 5 and 6. The effect of NO₂ was significant at lags 0 and 1, but only in patients aged ≥70 years (OR 1.13; 95% CI: 1.01–1.23; P=0.007) and those with pulmonary disorders (OR 1.12; 95% CI:1.01–1.31; P=0.01).

Conclusions: In all age groups, the short-term elevation in PM₂.₅ levels was associated with an increased number of daily hospital admissions for MI, whereas for PM₁₀ the effect was significant only with a simultaneous decrease in temperature. The effect of NO₂ was observed only in older individuals and patients with pulmonary disorders. A negative clinical effect was more delayed in time in the case of exposure to PM than to NO₂.

Key words: air pollution, myocardial infarction, particulate matter
Introduction

Both indoor and outdoor air pollution is a serious threat to public health, as it is associated with approximately 8 million premature deaths each year [1]. There are significant regional variations in terms of the degree of air pollution as well as its chemical composition, sources of emission, and geographical location, which also affects the concentrations of pollutants and determines their effect on human health.

In Poland, the limits set by the World Health Organization (WHO) for the levels of particulate matter (PM) are far exceeded, which constitutes a major problem. The main source of PM is low-stack emission (i.e. emission from residential and small-industry chimneys below the level of 40 m above ground), and to a lesser extent, transport and other sources [2]. The highest PM concentrations are observed during the heating season, especially when people use low-quality coal and harmful waste for heating purposes or use old coal heating furnaces. According to the WHO report, Krakow, otherwise known for its rich cultural and architectural heritage, is also one of the most polluted European cities in terms of PM concentrations in air [3]. Air pollution has been present in Krakow for many years. The fight against pollution is hindered by the fact that the city is located in a valley, there is a small number of windy days in a year, and there is a high density of buildings. Therefore, PM concentrations, especially daily values, far exceed the permissible levels, which results in more than 100 days of exposure to highly polluted air per year.

Previous studies indicated that air pollution not only aggravates the symptoms of cardiovascular diseases but is now also considered one of cardiovascular risk factors [4].

The effect of short-term exposure to air pollution on the risk of hospitalisation for acute myocardial infarction (MI) has already been studied, with variable, and sometimes conflicting, results. These discrepancies result from regional differences in pollutant concentrations, studied populations, individual susceptibilities, methods of measuring
exposure to air pollution, or statistical analysis methods [5]. Additional factors affecting the incidence of MI are weather conditions [6,7]. Thus, the impact of these environmental factors on human health should always be analysed on a local basis with consideration for those regional differences.

We aimed to assess the impact of short-term fluctuations in outdoor PM (PM$_{10}$ and PM$_{2.5}$) and the gaseous pollutant nitrogen dioxide (NO$_2$) on the number of hospitalisations due to MI among the inhabitants of Krakow (Poland), who are chronically exposed to air pollution.

**Patients and methods**

From the database of John Paul II Hospital in Krakow (one of the two largest hospitals in Lesser Poland Voivodeship), 5592 patients hospitalised between December 2012 and September 2015 with a final diagnosis at discharge of ST-segment elevation MI (STEMI) or non-ST-segment elevation MI (NSTEMI) were selected. Subjects with MI who died in hospital were also included in the study. Patients with unstable angina were excluded. After an analysis of confounding factors (different methods of measuring pollutant concentrations, lack of air pollution monitoring stations), the study population was limited to include only those inhabitants who had lived in Krakow for at least 30 years and resided there at the time of MI onset. The final study sample included 3545 participants.

The clinical diagnosis of STEMI and NSTEMI was made by cardiologists on the basis of the presence of symptoms, elevation of troponin or creatine kinase-MB levels, as well as presence of ST-segment elevation on electrocardiography in patients with STEMI.

In all participants coronary angiography was performed to confirm coronary artery disease (CAD). Normal coronary arteries were defined as a stenosis of less than 20%. Hypertension was defined as a blood pressure of 140/90 mmHg or higher on at least two separate measurements or the use of antihypertensive agents. Hyperlipidaemia was defined as hypercholesterolaemia, mixed hyperlipidaemia, or hypertriglyceridaemia according to current
guidelines [8]. Diabetes was diagnosed in line with applicable guidelines according to the Polish Diabetes Association [9].

Data collection

Data on air quality were obtained from the Regional Inspectorate for Environmental Protection in Krakow for the period from December 2012 to September 2015. Hourly data on PM_{10}, PM_{2.5}, and NO_{2} concentrations were collected from monitoring stations located in the following streets: Krasińskiego, Bujaka, and Bulwarowa. Pollutant concentrations were measured with automatic and manual methods. Daily city-level exposure to pollutants was estimated using hourly data obtained from the above stations. A 78% completeness criterion was applied for aggregate data calculation. If the daily average concentration of any parameter was not available in any station, the daily city-level concentration for that day was classified as “missing.” About 5% to 22% of the observations were missing during the study, and those missing data were excluded from the analysis.

Daily meteorological data were obtained from the Krakow branch of the Institute of Meteorology and Water Management for the same period.

The retrospective study protocol was approved by the Ethics Committees in Krakow. 153/KBL/OIL/2016 -BG; KBET 1072.61.20.283. 2018- EK

Statistical analysis

A time-series regression analysis [10-12] was used to examine the association between short-term fluctuations in air pollutants (PM_{2.5}, PM_{10}, NO_{2}) and hospital admissions for MI, using a generalised linear model and distributed lag model with the Poisson distribution. We used a previously verified generalised linear model to evaluate the relationship between air pollutants and hospital admissions, with the number of hospital admissions as the dependent variable [10-12] and the daily mean level of each individual air pollutant as the main exposure variable [12]. To examine the delayed effect of air pollutants, weather conditions, and
infections, we used a distributed lag model with a family of the Poisson distribution for a lag of 0 up to 6 days. A flexible spline function of time with 8 knots per year was used to control for the long-term trend and seasonal effects [10-11], and natural cubic spline functions with 4 degrees of freedom were used to adjust for the effects of temperature changes, relative humidity, and atmospheric pressure. Infections were also added to the model as a potential confounder. The analysis was performed separately for patients younger than 70 years and those aged 70 years or older.

To minimise the co-linearity effect, each air pollutant was modelled individually. Continuous variables were expressed as mean values with standard deviation and quartile distribution, whereas categorical variables, as numbers and percentages. When appropriate, medians with interquartile ranges were used. Non-normally distributed data were compared with the Mann–Whitney U test.

The pollutant concentrations obtained at the four monitoring stations were combined to present average values. Moreover, because patients served as their own controls, there was a near-perfect matching for participant-specific characteristics that did not vary over time (e.g., age, smoking status, other risk factors for CAD) [13]. All statistical tests were two-sided with an α value of 0.05. All analyses were performed with StatSoft Statistica 12 software (StatSoft, 2017). Forrest plots were developed with MedCalc software.

**Results**

The demographic and clinical data of Krakow inhabitants hospitalised due to MI between December 2012 and September 2015 are presented in Table 1. Daily concentrations of the main air pollutants (PM$_{10}$, PM$_{2.5}$, NO$_2$), weather conditions (humidity, atmospheric pressure, and temperature), and number of hospitalisations due to MI are presented in Table 2.
During the study, the average annual concentrations of pollutants were 54.2 µg/m³ for PM₁₀, 37.6 µg/m³ for PM₂.₅, and 42.3 µg/m³ for NO₂. The average daily concentrations of PM were higher in the autumn–winter months (heating season due to lower temperatures) compared with the spring–summer months (PM₁₀, 75.2 µg/m³ and 34 µg/m³, respectively, p<0.0001 and PM₂.₅, 55 µg/m³ and 21.3 µg/m³, respectively, p<0.00001) (Figure 1). No differences in NO₂ concentrations were observed between the seasons (38.4 µg/m³ and 37.5 µg/m³, respectively; P=0.70).

The median number of daily MI hospitalisations in the periods of elevated PM₂.₅ levels was higher compared with the months when no additional heating was needed (P=0.007) Changes in the number of daily hospitalisations and levels of pollutants according to seasons are presented in Figure 1. As the study was completed in September 2015, data for winter 2015 were not shown.

After adjustment for the effect of temperature changes, relative humidity, atmospheric pressure, and infections, an increase in the PM₂.₅ level of 10 µg/m³ was associated with an increase in the number of daily MI hospitalisations in both age groups: odds ratio (OR) 1.32; 95% CI: 1.01–1.40; P=0.0002 (Figure 2). A significant effect was observed until after a few days since exposure (lag, 5 to 6 days).

On the other hand, the effect of PM₁₀ was significant only with a simultaneous decrease in the mean daily temperature of 1°C: OR 1.08; 95% CI: 1.01–1.17; P=0.03 (Figure 3). Effect was also delayed until 5 to 6 days after exposure.

When the mean daily temperature increased by 1°C, the effect of PM₁₀ lost significance: OR 1.00; 95% CI: 1.00–1.01; P=0.07 in all age groups.

Regardless of the confounders such as weather conditions or infections, the significant effect of NO₂ was observed at lags 0 and 1, but only in patients aged 70 years or older (OR 1.13; 95% CI:1.01–1.23; P=0.007) (Figure 4A) and in patients with pulmonary disorders including
chronic obstructive lung disease and asthma (OR 1.12; 95% CI:1.01–1.31; P=0.01) (Figure 4B). In the remaining population, the association between NO₂ concentrations and daily hospital admissions for MI was nonsignificant.

**Discussion**

To our knowledge this is the first Polish study to indicate that even after adjustment for other potential confounders such as changes in temperature or barometric pressure, relative humidity, and infections, a short-term elevation of the PM₂.₅ level is associated with an increased number of hospital admissions due to MI. The effect of PM₂.₅ on the hospitalisation rate was observed in all age groups and regardless of coexistent disorders. Although in our study the effect of PM₂.₅ was independent of climate changes and infections, it should be emphasised that in Poland the highest PM₂.₅ concentrations are observed in the heating season (autumn–spring months) and usually overlap with periods of lower temperature or temperature inversion. An inversion is responsible for trapping pollutants and their accumulation at ground level. Low temperature was previously shown to be an independent risk factor for MI [6,7]; however, the mean outdoor temperatures are now higher than in the past. Moreover, global warming effects are observed also in the autumn–spring months, which can lessen the effect of low temperature on MI risk.

Several other studies have investigated the relationship between the PM₂.₅ concentration and MI onset, but the results are inconsistent [5,6,13-16]. In a case-crossover study, Barnett et al. [14] reported a significant relationship between PM₂.₅ and the risk of hospital admissions for cardiovascular causes (including MI) in older patients [14]. Also Zanobetti et al [15] reported that PM₂.₅ significantly increased the risk of MI. In contrast, Sullivan et al [16] did not observe a significant relationship between PM₂.₅ levels and MI. A more complex relationship between air pollutants and weather changes was observed by Huang et al. [6]. They reported a
combined effect of PM (both PM$_{2.5}$ and PM$_{10}$), carbon monoxide, and climatic changes on the risk of MI hospitalisations in Taiwan. High temperatures (>26°C) and low atmospheric pressure (<1009 hPa) on the previous day were shown to be associated with an increase in the incidence of MI. The combination of high PM concentrations or carbon monoxide with low temperatures (<21°C) and of high humidity levels with low temperatures was also associated with an increased MI incidence [6].

Another important issue in studies on the effect of air pollution on MI incidence is the time between exposure to air pollutants and development of MI. Our study revealed that in chronically exposed inhabitants of Krakow (average annual PM$_{2.5}$ levels of about 38 µg/m$^2$; the limit recommended by the WHO, 10 µg/m$^3$), the effect of short-term fluctuations in PM$_{2.5}$ levels on the risk of MI admissions was delayed, with significant effects observed at lags 4 and lag 6. We believe that in the case of exposition to PM$_{2.5}$, the time necessary for an atherosclerotic plaque destabilization and rupture as a result of oxidative stress, inflammation and endothelial dysfunction [17-21] is longer than in case of exposition to gaseous pollutants, and takes a few days since acute exposition.

In contrast to our results, other studies reported the negative effects of PM$_{2.5}$ on the cardiovascular system between 0 and 2 days since exposure to the elevated levels of pollutants [5,10].

The effect of PM$_{10}$ on MI risk was also investigated in a number of studies, with some showing a positive association but most showing a minor or no effect of exposure to PM$_{10}$ on the onset of MI [5,17-20]. In our study the effect of exposure to PM$_{10}$ on the risk of hospital admission was significant but only with a simultaneous decrease in temperature, which was shown to be an independent environmental risk factor for MI in previous studies.[4] The effect lost significance with an increase in temperature. Although PM$_{10}$ includes also smaller particles (PM2.5 and ultrafine particles), the correlations between exposure to PM$_{10}$ (as
compared with PM$_{2.5}$) and negative cardiovascular outcomes are weaker because, depending on the sources of PM$_{10}$ (in Poland seasonal differences in PM$_{10}$ concentration are observed), there may be considerable differences in the proportion of PM$_{2.5}$ found in PM$_{10}$. Unlike PM$_{10}$, PM$_{2.5}$ and ultrafine particles (UFP) with their small aerodynamic diameter may penetrate the lung alveoli directly into the bloodstream and thus result in more serious adverse cardiovascular effects because of a higher potential to generate oxidative stress [4,21-29].

We also investigated the relationship between NO$_2$ concentrations and the number of hospitalisations due to MI. In Poland NO$_2$ concentrations that exceed WHO standards are observed only in some cities and close to main arteries, while excessive PM$_{10}$ and PM$_{2.5}$ levels are observed in most Polish cities, villages and their mean annual concentrations are much higher than in other European cities. In our study a short-term elevation in NO$_2$ concentrations resulted in hospital admissions only in patients older than 70 years and in those with pulmonary disorders. This result is in line with other authors findings. [4,22-26]. In contrast, in the HEAPSS study, a significant effect of NO$_2$ on the risk of hospitalisation due to a first MI event was observed also in younger persons [24]. Also in our study, similarly to others, the effect of NO$_2$ was observed on the same or the next day of exposure. As gaseous pollutant NO$_2$ affects mainly the lungs, but may exert a secondary effect on the systemic circulation more rapidly than PM$_{2.5}$, mainly by inducing increased vasomotor tone, heart overload, oxidative stress, sudden blood thrombogenicity, and hypoxia.[4, 22-30] Older patients and those with pulmonary disorders are more prone to negative effects of NO$_2$.

In our study most patients with MI (90%) suffered from significant CAD confirmed by angiography. This observation is in line with the results of previous studies showing that patients with established CAD are especially prone to different environmental factors, such as air pollutants and temperature lowering, which trigger MI by destabilisation of atherosclerotic plaque or an increase in heart overload [31-35].
Our study has several limitations. First, pollution measured by outdoor monitors does not reflect personal exposure. For example, we did not consider individual exposure to indoor pollution sources related to cooking, heating, or smoking. In case of solid fuels used for heating or cooking there may be present a considerable exposure to PM and carbon monoxide coming directly from a heating appliance to the room. In such case indoor PM concentrations may be much larger than those in the ambient air. Second, the exposure values were averaged for the whole city, while the actual levels of pollutants and their chemical composition may vary between different city areas. However, in practice, exposure values have to be averaged, given the limited number of pollution monitors available. It should be also emphasised that the effect of ambient air pollution on human health results from interactions between its many different chemical components. Despite the norms adopted by the WHO and European Union for specific air pollutants, it is not possible to determine the threshold levels below which pollutants have no harmful effects on health. This is because the range of individual susceptibility in a population is so wide that some individuals may be susceptible event at the lowest level.

In conclusion, this study demonstrates that regardless of weather conditions and infections, the short-term fluctuation in outdoor PM$_{2.5}$ levels was positively associated with the number of daily hospital admissions for MI among chronically exposed inhabitants of Krakow. However, for PM$_{10}$ the effect was significant only with a simultaneous decrease in average air temperature. Regardless of weather conditions the significant effect of NO$_2$ was observed only in patients aged 70 years or older and those with pulmonary disorders. The effects of PM$_{2.5}$ and PM$_{10}$ were delayed until a few days after exposure, whereas those of NO$_2$ were observed on the same or the next day after exposure. It is possible that fluctuations in air pollutant levels in combination with weather changes possibly trigger MI by destabilisation of atherosclerotic plaque in individuals with established CAD.
Acknowledgements

The authors are greatly indebted to Ing R Listwan Regional Inspectorate for Environmental Protection, Kraków, for providing a part of data (concentrations of the pollutants).

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Contribution statement: EK - conceived idea of the study, contribution to the design of the research, analyzed the data, edition of the paper, funding for the project; GG - analyzed the data, edition of the paper, funding for the project; ŁN - statistical analysis and analyzed the data, edition of the paper; MK BG, PS - data collection, analyzed the data, edition of the paper; PP, JN, KŻ - contribution to the design of the study.

References


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13. Pope CA, Muhlestein JB, Anderson JL, et al. Short-term exposure to fine particulate matter air pollution is preferentially associated with the risk of ST-segment elevation


Table 1. Characteristics of the study population.

<table>
<thead>
<tr>
<th>Demographic and clinical data</th>
<th>n = 3545</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years] (SD)</td>
<td>72.9 (11.6)</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>1602 (45.1)</td>
</tr>
<tr>
<td>NSTEMI, n (%)</td>
<td>2127 (62)</td>
</tr>
<tr>
<td>STEMI, n (%)</td>
<td>1347 (38)</td>
</tr>
<tr>
<td>NSTEMI with normal coronary arteries, n (%)</td>
<td>149 (4.2)</td>
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<tr>
<td>STEMI with normal coronary arteries, n (%)</td>
<td>21 (0.6)</td>
</tr>
<tr>
<td>Arterial hypertension, n (%)</td>
<td>2079 (58.6)</td>
</tr>
<tr>
<td>Diabetes, n (%)</td>
<td>907 (25.6)</td>
</tr>
<tr>
<td>Obesity, n (%)</td>
<td>418 (11.7)</td>
</tr>
<tr>
<td>Hyperlipidaemia, n (%)</td>
<td>1994 (56.2)</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>356 (10.0)</td>
</tr>
<tr>
<td>Infections before hospital admission, n (%)</td>
<td>56 (1.5)</td>
</tr>
<tr>
<td>Primary PCI, n (%)*</td>
<td>3220 (90.8)</td>
</tr>
<tr>
<td>Previous MI, n (%)*</td>
<td>425 (12)</td>
</tr>
<tr>
<td>Heart failure, n (%)</td>
<td>496 (14)</td>
</tr>
<tr>
<td>In–hospital deaths n (%)</td>
<td>194.9 (5.5)</td>
</tr>
</tbody>
</table>

*PCI – Percutaneous coronary intervention
Table 2. Descriptive statistics for daily air pollutant concentrations, weather conditions and hospitalisations in the years from 2012 to 2015.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Percentile</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean (SD)</th>
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<tbody>
<tr>
<td></td>
<td>25th</td>
<td>50th</td>
<td>75th</td>
<td></td>
</tr>
<tr>
<td><strong>Air pollutants</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NO\textsubscript{2} (µg/m\textsuperscript{3})</td>
<td>53.0</td>
<td>64.0</td>
<td>75.0</td>
<td>22.0</td>
</tr>
<tr>
<td>PM\textsubscript{10} (µg/m\textsuperscript{3})</td>
<td>35.0</td>
<td>53.0</td>
<td>88.0</td>
<td>16.0</td>
</tr>
<tr>
<td>PM\textsubscript{2.5} (µg/m\textsuperscript{3})</td>
<td>24.0</td>
<td>37.0</td>
<td>64.0</td>
<td>11.0</td>
</tr>
<tr>
<td><strong>Weather conditions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature average (°C)</td>
<td>1.5</td>
<td>7.6</td>
<td>14.8</td>
<td>-13.5</td>
</tr>
<tr>
<td>Average relative humidity (%)</td>
<td>74.0</td>
<td>83.2</td>
<td>89.4</td>
<td>48.1</td>
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<tr>
<td>Average barometric pressures (KPa)</td>
<td>1011.8</td>
<td>1016.7</td>
<td>1020.8</td>
<td>980.7</td>
</tr>
<tr>
<td><strong>Number of hospitalisations</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>STEMI</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

* daily hospitalisations for MI
Figure 1. Changes in the number of daily hospitalisations due to myocardial infarction and air pollutant levels (PM$_{10}$, PM$_{2.5}$ and NO$_2$) between December 2012 and 2014.
Figure 2. Effect of a 10-μg/m$^3$ increase in PM$_{2.5}$ levels on hospital admissions due to myocardial infarction. Data are presented as odds ratios (and 95% CIs).
Figure 3. Effect of a 10-µg/m³ increase in PM₁₀ levels on hospital admissions due to myocardial infarction. Data are presented as odds ratios (and 95% CIs).
Figure 4A. Effect of a 10-µg/m³ increase in NO₂ levels on hospital admissions due to myocardial infarction in patients aged 70 years or older. Data are presented as odds ratios (95% CIs).
Figure 4B. Effect of a 10-µg/m³ increase in NO₂ levels on hospital admissions due to myocardial infarction in patients with pulmonary disorders. Data are presented as odds ratios (95% CIs).