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Air pollution in relation to very short-term risk of ST-segment elevation myocardial infarction: Case-crossover analysis of SWEDEHEART

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ABSTRACT

Objective: Studies have related air pollution to myocardial infarction (MI) events over days or weeks, with few data on very short-term risks. We studied risk of ST-segment elevation MI (STEMI) within hours of exposure to air pollution while adjusting for weather.
Methods: We performed a case-crossover study of STEMI cases in Stockholm, Sweden (Jan 2000–June 2014) based on SWEDEHEART. Exposures during hazard periods up to 24 h prior to admission were compared to bidirectionally sampled control periods. Risks attributable to sulphur dioxide (SO_2), nitrogen dioxide (NO_2), ozone and particulate pollutants ($PM_{2.5}$, PM_{10}) were studied in conditional logistic regression models for interquartile range increments.
Results: Risk of STEMI (n = 14,601) was associated with NO_2 (strongest at 15-h lag) and with $PM_{2.5}$ (strongest at 20-h lag), in single-pollutant models adjusting for air temperature and humidity (NO_2: odds ratio (OR; 95% confidence interval) 1.065 (1.031–1.101); $PM_{2.5}$: 1.026 (1.001–1.054)). After adjusting models for atmospheric pressure (significantly associated with STEMI risk at 14–24-h lags), NO_2 remained highly statistically significant (1.057 (1.022–1.094)) but not $PM_{2.5}$ (1.024 (0.997–1.052)). No associations were seen for SO_2, ozone or PM_{10}.
Conclusion: Risk of STEMI rises within hours of exposure to air pollutants, with strongest impact of NO_2. These findings are complementary to earlier reports which have not acknowledged widely the importance of very short-term fluctuations in air pollution.

1. Introduction

Air pollution is believed to be a key risk factor for cardiovascular disease [1] and the risk of myocardial infarction (MI) is likely to start rising within hours of exposure [2]. Previous reports have differed considerably in terms of temporal resolution, with most studies using long exposure times ranging from 24 h up to several weeks [3–5]. This is due to limitations inherent to many data sources: administrative or registry data are commonly available at daily resolution, and an apparent paucity of data on short-term risk has been recognised [6]. In one of the few analyses of data at very fine temporal resolution, a role was suggested for both nitrogen dioxide (NO_2) and particulate air pollution (PM_{10}), although, as exposure-to-event lags were analysed as 6-h terms, the actual hour-by-hour risk pattern remains uncertain [7]. Similarly, an earlier investigation of very short-term lags prior to MI events suggested a role of fine particles ($PM_{2.5}$), but confidence intervals were

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wide due to small sample size (n = 772), and analyses were limited to lags up to 6 h [2]. Lastly, air pollution acts in concert with weather to create an environment predisposing to MI [8], which must be taken into account when estimating risk attributable to air pollution [9].

On this background, we investigated the relationship between air pollution and MI events, after adjusting for weather, in the prospective clinical registry SWEDEHEART (Swedish Web System for Enhancement and Development of Evidence-Based Care in Heart Disease Evaluated According to Recommended Therapies) [10], a nationwide database of MI cases in Sweden, admission times for which are documented to the nearest minute i.e. at very high temporal resolution. To leverage maximally on this strength, we chose *pre-hoc* to focus on ST-segment elevation MI (STEMI) cases as the distinct symptom onset that characterizes this group should minimize the inherent uncertainty produced by delays between actual onset and admission [2,11]. Our aim was to study the role of air pollutants in the hours leading up to STEMI admission after adjusting for weather.

2. Material and methods

The study was performed as a case-crossover analysis [12] with methodology as follows [13].

2.1. Study population

We considered for inclusion all MI patients entered in SWEDEHEART at 1 of 10 acute hospitals in the greater Stockholm area (defined below) for whom date and time of admission were known. Patients with a final diagnosis other than STEMI were excluded (e.g. chest pain or angina pectoris, non-ST elevation MI (NSTEMI), Tako-Tsubo cardiomyopathy, type II myocardial infarction events etc.).

The rationale and organisation of SWEDEHEART has been described in detail elsewhere [10]. In brief, patients are included prospectively on admission to a dedicated cardiac unit for chest pain or MI at one of 65 hospitals in Sweden. The registry contains variables describing infarct type, co-morbidities, treatment on admission as well as in-hospital, and any complications. Data accuracy is approximately 96% as ascertained through a process of continuous monitoring [14].

Greater Stockholm is the largest conurbation in northern Europe, encompassing municipalities on lake Mälaren and the Baltic Sea coast of eastern Sweden (Supplementary Fig. 1).

The study was approved by the regional ethics committee in Stockholm, Sweden (Dnr 2012/60-13/2) and adheres to the Declaration of Helsinki. As this project was a national registry study, requirement for written informed consent was waived.

2.2. Data on air pollution and weather

Air pollution data were obtained from Swedish Environmental Research Institute (www.ivl.se) at hourly resolution for gaseous air pollutants: NO_2, sulphur dioxide (SO_2) and tropospheric ozone, as well as suspended particulate matter subdivided based on diameter: ≤2.5 μm ($PM_{2.5}$) vs. ≤10 μm (PM_{10}). Monitoring stations with hourly data were used as shown in Supplementary Fig. 1. In brief, all air pollution data were obtained from two inner city monitoring stations located within a radius of 100 m: particulate pollutants ($PM_{2.5}$ and PM_{10}) were obtained at *Hornsgatan*, which is considered a street level station, located at a height of 3 m (i.e. not 'kerb-side') along a street where an estimated 40,000 cars pass each weekday. Gaseous pollutants (SO_2, NO_2, ozone) were obtained at *Torkel Knutssonsgatan*, a roof-top station located 20 m above the street. Meteorological data were obtained as follows. Atmospheric pressure was available from the monitoring station at *Bromma Flygplats* which is located 14 m above sea level, close to Bromma Airport, 12 km North-West of the city centre of Stockholm. All other meteorological variables were obtained from the monitoring station at *Tullinge Flygfält*, located 45 m above sea level, 23 km south of the city centre of Stockholm (Supplementary Fig. 1).

2.3. Myocardial infarction data

Acute admissions in the catchment area described above occurred through emergency departments at public hospitals in operation during the study period. Of note, all hospitals offering emergency services for acute chest pain or MI in Stockholm are public. Admissions recorded in SWEDEHEART may therefore be considered an unselected population, and coverage satisfactory.

2.4. Statistics

The relationship between air pollutants and incident MI was analysed after adjusting for weather in a case-crossover design as originally described by Maclure [12]. In brief, this is a type of self-matched case-control study: for each person, exposure data are collected for the time period before the event ('hazard' period) and for a set of 'control' periods that were not associated with the event in question. The method studies the effects of an intermittent exposure on an acute outcome in a time-stratified fashion such that hazard periods are compared to control periods. Exposure was defined during each period as averages over 3-h windows, and bidirectional sampling was performed of control periods exactly 2 weeks before and after the hazard window (i.e. ±336 h; 2 controls per case). While case-crossover studies eliminate within-subject confounders by design, any impact of temporal trends is minimised as control periods are sampled both before and after the case period. Lags of up to 24 h prior to admission were analysed in conditional logistic regression with nesting on each set of case and control periods. Regression was performed with all predictors entered as continuous covariates, with results scaled to express the effect per 1-unit increment on the interquartile range (IQR) scale, with the exception of precipitation which was extremely skewed: 80.1% of all hours exhibited zero precipitation, and a dichotomous variable was therefore created based on any precipitation vs. none. Temperature was entered into models at a 21-h lag [5,15] as a restricted cubic spline [16] with 3 degrees of freedom (which minimised Akaike information criteria). Univariate analyses were performed by regressing STEMI risk on predictors at each individual lag. Multivariable single-pollutant models were built by also entering non-linear effects of potential confounders to univariate models: non-linearly analysed (i.e. spline transformed) temperature and relative humidity were chosen for consideration *pre-hoc*. [17] Missing data for pollution and weather variables were interpolated linearly. All analyses were done in R version 3.4.

2.5. Supplementary analyses

Three sensitivity analyses were performed to demonstrate robustness of results to (1) seasonal infections, which were entered as potential confounders (influenza and respiratory syncytial virus) using weekly count data on the number of cases diagnosed. These were available for the Greater Stockholm area period 22 Oct 2001 until the end of the study period (i.e. 1 June 2014) from the national Swedish microbial surveillance program administered by the Public Health Agency of Sweden (www.folkhalsomyndigheten.se). (2) Robustness to distance to weather and pollution stations was considered by excluding hospitals located outside of the Stockholm City area. (3) Lastly, selection of control periods was redesigned to use periods matched on clock-time but at +/−3 and +/−6 weeks from the case period, for a total of 4 controls for each case.

3. Results

Study period and accordingly also sample size were determined in the following manner: (i) SWEDEHEART had incomplete coverage prior to 2000, and $PM_{2.5}$ was available in Stockholm only after 1 Jan 2000 which was accordingly considered the earliest date possible for analysing hourly data. To accommodate bidirectional sampling, the final study period therefore began on 15 Jan 2000. (ii) Data were available for this project from SWEDEHEART until 1 June 2014 which was accordingly considered the end of the study period. (iii) SO_2 was available only until 31 Oct 2005 and analyses of this pollutant was therefore restricted to that period, i.e. ended on 15 Oct 2005. Main analyses therefore spanned a total of 117,240 h or 13.4 years, and SO_2 analyses spanned 50,400 h or 5.75 years. The population in the catchment area averaged 2,159,616 (Supplementary Table 1). There were 14,601 admissions for STEMI that met inclusion criteria for the main analysis (5611 for analysis of SO_2), corresponding to 1091 admissions annually or 51 admissions per 100,000 inhabitants per year (50.0 admissions for SO_2) (Supplementary Table 2). Patient characteristics are shown in Supplementary Table 3.

3.1. Air pollution and weather variables

Aggregate data on air pollution and weather variables are shown in Table 1. Counts for missing data are shown in Supplementary Table 4.

3.2. STEMI risk in relation to air pollution and weather

Associations for lags up to 24 h are shown in Fig. 1 after scaling to express effect per IQR increment, with statistical significance of estimates indicated as 95% confidence intervals. Two air pollutants exhibited a statistically significant association with STEMI risk: (i) NO_2 at a 15-h lag (odds ratio (95% confidence interval): 1.042) (1.011–1.073) ($P = 0.007$) and (ii) $PM_{2.5}$ at a 20-h lag: 1.027 (1.001–1.054) ($P = 0.042$). IQR intervals are tabulated in Table 1.

As shown in Fig. 1, atmospheric pressure and air temperature were both positively related to STEMI risk, and wind velocity negatively:

Table 1
Air pollution and weather in Greater Stockholm from 1 Jan 2000–1 June 2014, except *SO_2 which is shown from 1 Jan 2000–31 Oct 2005. All data are mean (SD).

Variable	Value	Interquartile range
Weather		
Atmospheric pressure, mm Hg	1012.0 (11.6)	15.1
Precipitation, cm	0.06 (0.27)	0.0
Relative humidity, %	80.9 (16.0)	20.5
Temperature, °C	5.8 (8.5)	12.0
Wind velocity, m/s	2.9 (1.8)	2.6
Pollution		
Gaseous pollutants		
NO_2, μg/m^3	15.0 (10.9)	12.9
Ozone, μg/m^3	51.1 (21.9)	30.2
SO_2,* μg/m^3	2.4 (2.8)	1.8
Particles		
$PM_{2.5}$, μg/m^3	13.5 (9.4)	9.4
PM_{10}, μg/m^3	38.4 (43.8)	26.5

strongest association for atmospheric pressure at 19 h: 1.028 (1.002–1.055) ($P = 0.034$), for air temperature at 21 h: 1.067 (1.047–10.87) ($P = 0.019$), and for wind velocity at 22 h: 0.964 (0.934–0.995) ($P = 0.022$). Raw data on levels of NO_2 and $PM_{2.5}$ at 15 h and 20 h, respectively, are tabulated in Supplementary Table 5.

3.3. Multivariable analysis

As shown in Table 2, multivariable analyses of NO_2 and $PM_{2.5}$ in single-pollutant models that adjusted for non-linear effects of temperature and relative humidity showed an independent association with STEMI risk of both NO_2 (1.065 (1.031–1.101); $P < 0.001$) and $PM_{2.5}$ (1.026 (1.001–1.054); $P = 0.046$). When additional adjustment was performed for atmospheric pressure, NO_2 retained a similar effect size with a high degree of statistical significance (1.057 (1.022–1.094); $P = 0.001$) but the estimate of $PM_{2.5}$ straddled the null (1.024 (0.997–1.052); $P = 0.083$).

3.4. Supplementary analyses

Supplementary analyses including sensitivity testing showed directionally similar albeit smaller effect estimates to the main analyses (Supplementary Table 6).

4. Discussion

We examined the association between the very short-term risk of STEMI and air pollutants after adjusting for weather in the greater Stockholm area. Key findings of this project were that, firstly, very short-term risk was most strongly related to exposure to the gaseous pollutant NO_2, with a weaker effect of fine particulate air pollution ($PM_{2.5}$). In models adjusting for air temperature, relative humidity and atmospheric pressure, NO_2 remained independently correlated to STEMI risk, but $PM_{2.5}$ was rendered non-significant.

4.1. Impact of air pollution on STEMI risk

The existence of very short-term effects of air pollutants on MI risk is supported by mechanistic studies which have demonstrated that inhalation of diesel exhaust is pro-ischaemic and pro-thrombotic in subjects with ischaemic heart disease within an hour of exposure [18]. Nonetheless, a majority of previous reports in this field have been based on administrative or clinical registry data with low temporal resolution, and studies have typically linked daily means for environmental variables with daily counts of MI events [4], studying lag times of several days or even weeks [5,19]. Accordingly, few published reports have achieved as high a temporal resolution as the present study.

The present report noted elevated STEMI risk within hours of exposure to the gaseous pollutant NO_2, as well as the particulate pollutant $PM_{2.5}$. Bhaskaran et al. investigated events in the nationwide MINAP registry in UK and found a relationship with NO_2 levels at a lag of 1–6 h: a 2% increase in risk was seen for each 10 μg/m^3 increment, i.e. a similar effect to that seen in the present study: we found a 4.2% increase in the odds of STEMI for a 12.9 μg/m^3 increment in NO_2 [7]. Short-term effects of particulate pollution on MI risk was studied by Peters et al.: [2] $PM_{2.5}$ was associated with an increased MI risk within an hour of exposure. As the analysis was truncated at 6 h, the longer lags where the present analysis found an association with $PM_{2.5}$ (highest at 20 h) were not included.

Interestingly, we noted a positive relationship between STEMI risk and atmospheric pressure which was sustained over the lags where statistically significant associations were seen with NO_2 and $PM_{2.5}$. While earlier reports have reported conflicting data regarding the association between MI risk and atmospheric pressure [20], a U-shaped association has been proposed [21] as has an increased risk the day after a *decrease* in pressure, which was suggested to be due to an *increase* in risk once pressure begins to rise again [22]. When atmospheric pressure was taken into account, we found that NO_2 was still independently associated with STEMI but not $PM_{2.5}$.

One possibility is that elevated levels of atmospheric pressure and $PM_{2.5}$ may both occur during times of stable and relatively less windy weather due to limited atmospheric mixing—an interpretation supported by positive relationships between STEMI risk and both temperature and atmospheric pressure and the inverse relationship seen with wind velocity. A majority of cases were from the city centre of Stockholm where exhaust fumes can be expected to play an important role in air pollution. Formation of so-called inversion layers characterised by cool air layers being trapped under a layer of warmer air acting as a 'lid' may offer an explanation for the pattern seen in the present report [23]. While most research in this area have studied the impact of inversion layers on respiratory health, an association with cardiovascular risk will require future study using other data sources [24,25].

The present report also suggested that different pollutants appear to exert maximal effects at different lags: the effect of NO_2 was highest at a lag of 15 h whereas the relatively weaker effect seen for $PM_{2.5}$ was noted at a longer lag of 20 h. Admittedly, the present analysis was very granular in its use of hourly intervals for air pollutants, and role of the exact lags may be difficult to know. Nonetheless, it does suggest that pollutants may, firstly, not have synchronised effects on risk post-exposure. While emissions are typically of NO, atmospheric conversion to NO_2 occurs in the presence of ozone and ultraviolet light. NO_2 together with nitrate aerosols and hydrocarbons form an important fraction of $PM_{2.5}$ [26]. The fact that the 2 air pollutants related to short-term STEMI risk in this report (NO_2 and $PM_{2.5}$) are thus closely related raises the possibility that the seemingly more rapid impact of NO_2 may reflect genuine differences in 'incubation time'. While both are known to cause airway inflammation, NO_2 is poorly water soluble which enables it to reach the distal airways rapidly, where it undergoes conversion to nitric and nitrous acids. It is conceivable that this facilitates a faster effect of NO_2 on endothelial function or more rapid oxidative microvascular stress, as opposed to the more heterogeneous pollutant $PM_{2.5}$ which is more mixed in terms of physical properties and chemical composition [27,28].

4.2. Limitations

Several limitations apply to this study. Firstly, central monitoring sites were used as a surrogate for population exposure which introduces questions of within-city heterogeneity and representativeness of measurements; wearable sensors may provide a means to obtaining a more direct estimate of exposure in future studies [29]. Secondly, studies of routinely acquired data on pollutant levels and weather variables need to take into account measurement error which can be a source of analytical noise. Thirdly, data in time series at hourly intervals will inevitably have an inherent temporal dependency structure, and repeated

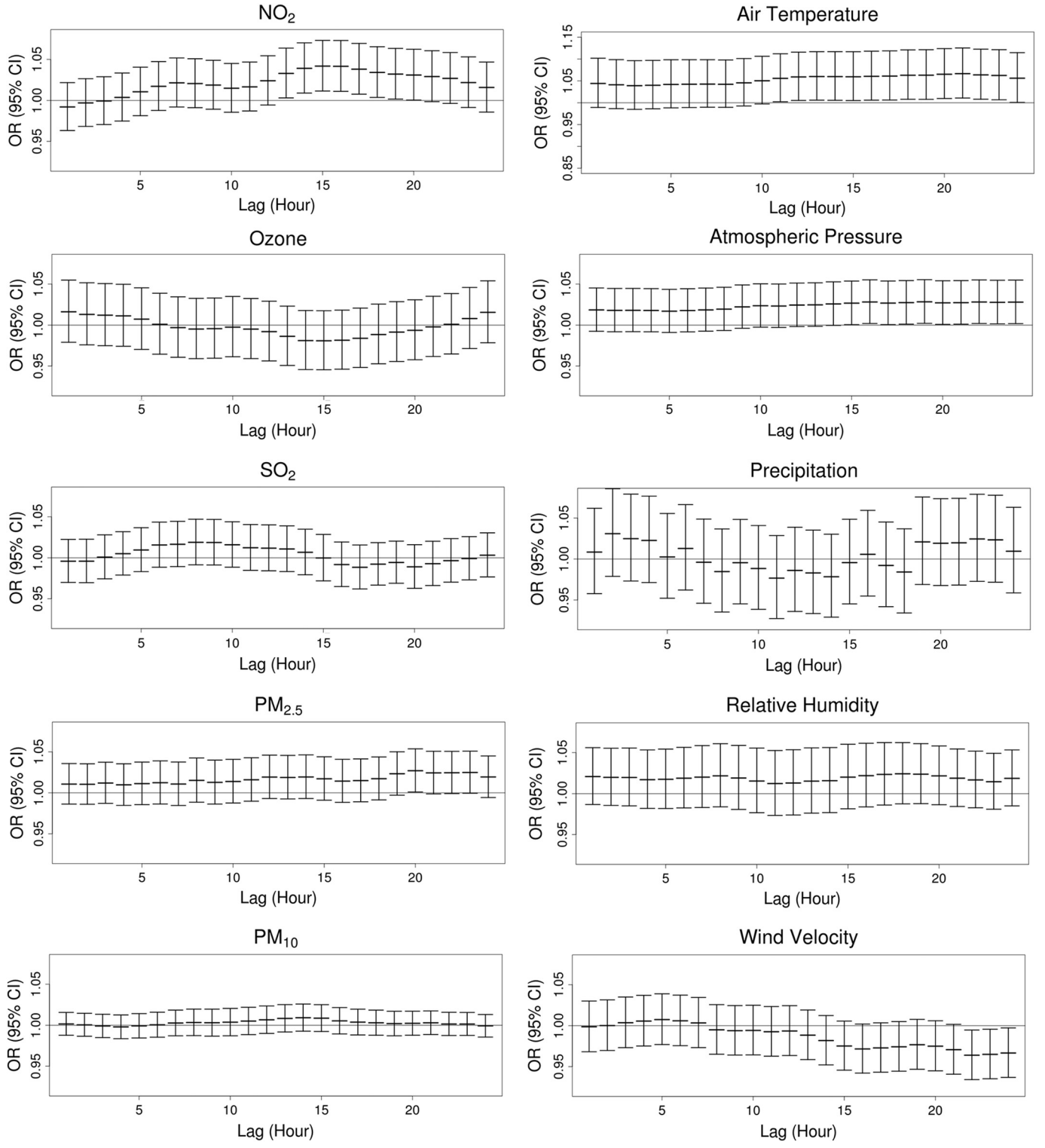

Fig. 1. Lag-response diagrams showing risk of STEMI as odds ratios with 95% confidence intervals for interquartile range increments in predictor variables, except precipitation which is modelled after dichotomising into any vs. none.

null-hypothesis significance testing at individual lags may act to inflate the risk of a type I error. This report therefore seeks to convey the important message that short-term effects of air pollution do exist, but that the patterns are likely to be more important than the exact lags per se. The sensitivity analysis using differently sampled controls illustrates this point: while the overall pattern remained, the effect of NO_2 at a 15-h lag was smaller. Repeating the analysis with differently sampled control weeks represents a form of out-of-sample test of validity, and, while a similar pattern was seen of a short-term effect of NO_2, its size at a 15-h lag was more modest than in the original analysis.

5. Conclusion

We found associations between STEMI risk and the gaseous pollutant NO_2 within hours of exposure which were independent of effects of temperature, relative humidity and atmospheric pressure. The

Table 2
STEMI risk associated with air pollutants in univariate, as well as multivariable single-pollutant models. Estimates in model 1 were adjusted for non-linear effects of temperature and relative humidity, and in model 2 also for atmospheric pressure. Odds ratios are estimated based on 1-unit increments in predictors scaled to the interquartile range scale.

Model	Lag	Odds ratio	*P*
Univariate analyses			
NO_2	15	1.042 (1.011–1.073)	0.007
$PM_{2.5}$	20	1.027 (1.001–1.054)	0.042
Multivariable, single-pollutant model 1			
NO_2	15	1.065 (1.031–1.101)	<0.001
$PM_{2.5}$	20	1.026 (1.001–1.054)	0.046
Multivariable, single-pollutant model 2			
NO_2	15	1.057 (1.022–1.094)	0.001
$PM_{2.5}$	20	1.024 (0.997–1.052)	0.083

particulate pollutant $PM_{2.5}$ was also related to STEMI risk in models that adjusted for temperature and humidity, but non-significant after taking into account atmospheric pressure. These data are complementary to earlier reports in this field, by showing the importance of very short-term fluctuations in air pollution for STEMI risk, which has not been widely acknowledged previously.

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Conflicts of interest

The authors report no relationships that could be construed as a conflict of interest.

Data availability

Datasets on air pollution and weather can be downloaded as described. Access to SWEDEHEART is available to collaborators with appropriate approvals including that of the registry steering committee and after ethics committee review. Concomitant data on seasonal infections from the national Swedish microbial surveillance program is available through the Public Health Agency of Sweden (www.folkhalsomyndigheten.se).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2018.10.069.

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