Traffic-related air pollution and cardiovascular mortality in central Taiwan

Dai-Hua Tsaia, Jia-Lin Wangb, Kai-Jen Chuanga, c, Chang-Chuan Chan*a

a Institute of Occupational Medicine and Industrial Hygiene, College of Public Health, National Taiwan University, Taiwan

b Department of Chemistry, National Central University, Taoyuan County, Taiwan

c St. Mary’s Medicine Nursing and Management College, Yilan County, Taiwan

* Corresponding Author:

Prof. Chang-Chuan Chan

Institute of Occupational Medicine and Industrial Hygiene,

College of Public Health, National Taiwan University,

Rm. 722, No.17, Xu-Zhou Rd., Taipei 100, Taiwan

Tel/ Fax: 886-2-33228082 / 886-2-23222362

Email: ccchan@ntu.edu.tw
Abstract

This study aims to investigate the relationship between cardiovascular mortality and traffic-related air pollutants, including NO₂, CO, and six volatile organic compounds (VOCs), i.e. propane, iso-butane, propylene, benzene, m,p-xylene, and o-xylene. The concentrations of NO₂ and CO from 1993 to 2006 were measured at a fixed-site air monitoring station, and VOCs data from 2003 to 2006 were obtained from a photochemical assessment monitoring station (PAMS) in an urban area in central Taiwan. Outcome variables were daily mortality data of cardiovascular diseases (ICD-9-CM 410-411, 414, 430-437) from 1993 to 2006. Cardiovascular mortality averaged 1.5 cases, ranging between 0 and 9 cases per day. Daily air pollution levels ranged 0.5-80.5 ppb for NO₂ and 0.1-3.8 ppm for CO. From the subset data of 2003 to 2006, daily averaged values ranged 0.6-17.5 ppb for propane, 0.3-6.7 ppb for iso-butane, 0.3-6.7 ppb for propylene, 0.2-3.8 ppb for benzene, 0.3-26.0 ppb for m,p-xylene, and 0.02-7.6 ppb for o-xylene. Poisson Generalized additive models (GAM) were used to estimate the effects of elevated air pollutant levels on daily mortality adjusting for meteorological conditions and temporal trends. Single-pollutant models showed that cardiovascular mortality was significantly associated with CO and NO₂ lagged 0-2 days, and was also associated with each of the six VOCs lagged 0 day. The relative risk (RR) for an interquartile (IQR) increase in air pollution levels were: 1.027-1.031 for NO₂, 1.022-1.029 for CO, 1.065 for propane, 1.055 for iso-butane, 1.049 for propylene, 1.059 for benzene, 1.039 for m,p-xylene, and 1.047 for o-xylene, respectively. Such associations became insignificant in two-pollutant models. In conclusion, daily cardiovascular mortality is associated with acute exposure to traffic air pollution in urban environment.

Keywords: Traffic; Cardiovascular; Mortality; Volatile organic compound; Generalized additive model
Acknowledgment

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1. Introduction

Nitrogen dioxide (NO$_2$) and carbon monoxide (CO) are good indicators of traffic exhaust emissions because they contribute most of the total emission (Holman, 1999). Epidemiologic studies had reported that short term exposure to NO$_2$ and CO is associated with increased cardiovascular mortality, including cerebrovascular diseases [International Classification of Disease, 9$^{th}$ revision (ICD-9), 430-436], strokes (160-169 by ICD-10), and ischaemic heart disease (ICD-9, 410-414) (Forastiere et al., 2005; Hoek et al., 2002; Hong et al., 2002). Source-apportionment studies have identified volatile organic compounds (VOCs) as a major group of traffic-related air pollutants (Guo et al., 2007; Mukerjee et al., 2004). And several commuter studies have found that commuters around the world were exposed to similar traffic-related VOCs in urban environments (Chan et al., 1993; Chan et al., 1994; Chan et al., 1991; Guo et al., 2007; Jo and Park, 1999; Kuo et al., 2000; O'Donoghue et al., 2007). VOCs are an important class of outdoor air toxics because they are ubiquitous and associated with increased long-term health risks (Pratt et al., 2000). However, no study has yet addressed the relationship between cardiovascular mortality and traffic-related VOCs, possibly due to lack of long-term monitoring data of VOCs.

The association between ambient particulate matter with an aerodynamic diameter less than 10 micrometers (PM$_{10}$) and cardiovascular mortality has been conducted in many countries (Pope and Dockery, 1999; Samet et al., 2000). Specifically, cerebrovascular
mortality, ischemic heart disease and strokes were often stratified to investigate the
mechanism of the association with PM$_{10}$ (Hoek et al., 2001; Hong et al., 2002; Zeka et al.,
2005). Therefore, it is imperative to investigate its effects on cardiovascular mortality, though
PM$_{10}$ are known to be produced by diverse sources other than traffic emissions (Holman,
1999).

Since 2002, photochemical assessment monitoring sites (PAMS) established in Taiwan
routinely monitored C$_2$-C$_{12}$ VOCs with hourly resolution (Tsai et al., 2008). The VOC dataset
provided a special opportunity for us to analyze the association between daily variation in
VOC concentrations and cardiovascular mortality. The aim of this study was to investigate
whether daily cardiovascular mortality was associated with traffic-related air pollutants,
including NO$_2$, CO, VOCs as well as PM$_{10}$ in an urban environment with significant traffic
pollution.
2. Materials and methods

2.1 Study area

Taichung is the third largest city in Taiwan, with 163 square kilometers and a population of 1,044,392 inhabitants (population density: 6,407 people / km²). It is a densely populated metropolitan with a vehicle density of 5,724 vehicles/km². The estimated mobile emission was 14,905 tons/year for NOx, 65,966 tons/year for CO, 11,056 tons/year for VOCs and 1,284 tons/year for PM$_{10}$ (TEPA, 2006). There were 9,704 buses and trucks, 342,667 cars, and 580,643 motorcycles in Taichung city at the time of this study (DOT, 2006).

2.2 Mortality data

Data on daily deaths recorded from January 1, 1993 to December 31, 2006 within Taichung City were obtained from the Department of Health, which is in charge of the death registration system in Taiwan. ICD-9 was used as the disease classification code. Our outcome variables in this study include death from cerebrovascular diseases (ICD-9, 430-437), and ischaemic heart diseases (ICD-9, 410-411, 414). We obtained all the electronical records of daily death data from 1 January 1993 to 31 December 2006. The study period of daily mortality data was matched with the available period of daily environmental data for statistical analysis.

2.3 Environmental data

Hourly concentrations of NO$_2$, CO, and PM$_{10}$ were measured by one fixed-site
monitoring station (Chongming station) in downtown Taichung from 1 January 1993 to 31 December 2006, for a total of 5112 days. This station was operated by the Taiwan Environmental Protection Agency (TEPA) and placed on the roof of a three-story building in a school campus. The building is 15 meters above ground level and 200 meters away from main traffic roads. The station uses commercial monitoring instruments designated by the USEPA as an equivalent or reference method and manufactured by US Thermo Environmental Instruments, Inc. (Franklin, MA, USA). The instruments included: a) model 42 chemiluminescence NO-NO$_2$-NOx analyzer (RFNA-1289-074) for NOx, b) model 48 gas filter correlation ambient CO analyzer (RFCA-0981-054) for CO, and c) model 650 (formerly: Wedding & Associates) PM$_{10}$ Beta gauge automated particle monitor (EQPM-0391-081) for PM$_{10}$. The scheduled quality control procedures included daily zero and span checks, biweekly precision checks, quarterly multiple-point calibrations and data validation. Accordingly, TEPA contracted an independent quality assurance program and instrument performance audit to assure data quality.

Hourly data of VOCs from January 1, 2003 to December 31, 2006, a total of 1461 days, were obtained from a PAMS station, which was located next to the fixed-site air monitoring station, where 55 VOCs species of ozone precursors are being continuously measured. The selection of the target 55 VOCs was based on a list suggested by the U.S.EPA for PAMS located in ozone non-attainment areas.(USEPA, 1994) Commercial auto-2-D GC systems
(Perkin Elmer, USA) were used to monitor VOCs in these PAMS stations. The system employed a cryogen free sorbent trap with 2-D chromatography to cope with the wide volatility range of the target compounds. The PLOT column ($30\text{m} \times 0.25\text{mm}$; $\text{df}=8\text{m}$, Hewlett-Packard, San Fernando, CA, USA) separates lower boiling compounds from $C_2$-$C_5$; whereas the DB-1 column ($60\text{m} \times 0.25\text{mm}$; $\text{df}=1.0\text{m}$, J&W Scientific, Folsom, CA, USA) separates higher boiling ones from $C_6$-$C_{12}$. The system was equipped with a Deans switch to heart-cut the early eluting 25 $C_2$-$C_5$ species from the DB-1 column to the PLOT column before reaching the first flame ionization detector (FID). The remaining 30 $C_6$-$C_{12}$ compounds were cut to an uncoated column to be detected by the second FID. Overall, there were 29 alkanes, 9 alkenes, 1 alkyne, and 16 aromatics in the 55 VOCs. For each hourly analysis, ambient air is drawn via the air inlet into the trap for 45 minutes. Species confirmation and concentration calibration was carried out by a pressurized standard gas mixture containing 55 target compounds with concentrations ranging from 20 to 40 ppbC (ppb multiplied by carbon number) (purchased from Spectra gases, Branchburg, NJ, USA). In the early phase of the monitoring program, daily calibration checks were performed at midnight. As a result, measurements per day comprised of 23 sample injections plus one standard gas injection. In the second phase of monitoring (starting from 2004) when the instrument had shown sufficient stability, the calibration check was only performed every 5 days at each station. The retention times of the 55 compounds were used for species identification, and the
concentrations of the 55 species were calculated by their individual response factors obtained from the standard aliquots. GC/MS was also used to confirm species identification. In addition to the routine quality control and assurance check on the system and the data, the system was also validated by comparing it with a self-built auto-GC with subtle differences in design and construction. (Wang et al., 2004) Highly agreeable results were obtained from the parallel measurements sharing a common air inlet.

Among those 55 VOCs, six VOCs were selected as traffic-related indicators. They were propane from LPG vehicles (Blake and Rowland, 1995), iso-butane from evaporative losses from gasoline (Buzcu and Fraser, 2006), propylene, benzene, m,p-xylene, and o-xylene from vehicle exhausts (Elbir et al., 2007). All hourly concentrations were then averaged into daily values (0:00-23:00) to represent daily air pollution situations in the entire Taichung area.

2.4 Statistical methods

Descriptive statistical analyses of mortality data and environmental data are summarized into mean, standard deviation, maximum, minimum, and interquartile range (IQR) in Table 1. Pearson correlation analysis was used to analyze the correlation between all nine air pollutants considered in this study from 2003 to 2006 in central Taiwan. Generalized additive models (GAM) were used to analyze the association between air pollution and mortality by adjusting for non-linear relation between confounders and mortality cases (Hastie and
Smooth function in GAM was used to control temperature effects and time-trend variables in our models. The imputations were carried out using GAM with Poisson distribution. The general form of our base model is

$$\log(E(Y_t)) = \beta_0 + X_t + S_{\text{temp}} + S_{\text{month}}$$

$Y_t$ obeys Poisson distribution with parameter $E(Y_t)$ and represents mortality on day $t$ in the equation. $\beta_0$ is the model intercept, and $X_t$ is the pollutant variables on day $t$. $S_{\text{temp}}$ and $S_{\text{month}}$ are the filtered, smoothed functions of temperature and time trend. The assumption of the linearity between the log of mortality cases and air pollution was graphically analysed using the locally weighted running-line smoother (LOESS) to remove low-frequency variability (Cleveland, 1979; Cleveland and Devlin, 1988). The filtering parameter used to remove monthly variability was selected by minimizing autocorrelation of residuals, reviewing residual plots, and minimizing the Akaike Information Criterion (AIC) (Cakmak et al., 1998). AIC is essentially a measure of goodness of fit that penalized for model complexity.

Log-relative risks were estimated using GAM.

Single-pollutant models were built by adding each of the three air pollutants ($\text{NO}_2$, $\text{CO}$, and $\text{PM}_{10}$) lagged 0-2 days from 1993 to 2006 to the base model. Due to the limited availability of data on the traffic-related VOCs, the analysis of association between cardiovascular mortality and traffic-related VOCs, was limited to 1461 days, from 2003 to 2006. Single-pollutant models were built by adding each of the nine air pollutants ($\text{NO}_2$, $\text{CO}$,
PM\textsubscript{10}, propane, iso-butane, propylene, benzene, x,p-xylene, and o-xylene) lagged 0-2 days to the base model.

The air pollutants, whose relative risk (RRs) and 95% confidence intervals (CIs) for IQR change in pollution levels less than a significance level of 0.05 in the single-pollutant models, were considered for further analyses in our two-pollutant models. All data analyses were performed by SAS software procedure (version 9.1; SAS Institute Inc., Cary, NC) and a significance level of 5% was used.
3. Results

Table 1 summarizes the daily mortality and environmental conditions data used in the models. There were 7504 cardiovascular death cases in central Taiwan from 1 January 1993 to 31 December 2006, a total of 5112 days, with a mean age of 71 years at the time of death. Among cardiovascular death cases, about 70% were cerebrovascular disease cases, i.e. 5200 cases, and 2304 were ischaemic heart disease cases. Furthermore, among cerebrovascular deaths, half of them are stroke cases (53%). Daily mortality for cardiovascular diseases in central Taiwan averaged at 1.5 cardiovascular deaths per day. Daily ambient temperatures and air pollution levels varied considerably during this period in central Taiwan. During the study period, daily temperatures fluctuated between 10.3 and 34.1 °C and ambient air pollution levels differed from 0.5 to 80.5 ppb for NO₂, from 0.1 to 3.8 ppm for CO, and from 9.0 to 263.3 µg/m³ for PM₁₀. The IQR of the environmental data, which were used for calculating RRs in the GAM, were 8.3 °C for daily temperature, 13.4 ppb for NO₂, 0.5 ppm for CO, and 46.4 µg/m³ for PM₁₀, respectively. For a shorter subset data from 2003 to 2006, daily cardiovascular mortality was 1.5 deaths per day, which were not different from the full dataset of 1993 to 2006. There were only 2151 cardiovascular cases, including 829 ischaemic heart disease cases, and 1322 cerebrovascular disease cases (data not shown). The IQR of VOC data in this short period were 2.7 ppb for propane, 0.96 ppb for iso-butane, 0.85 ppb for propylene, 0.62 ppb for benzene, 1.62 ppb for m,p-xylene, and 0.59 ppb for o-xylene,
respectively.

Table 2 shows Pearson correlation coefficients between NO$_2$, CO, PM$_{10}$ and the six VOCs from 2003 to 2006. It should be noted that those VOCs were highly correlated with each other, ranging from 0.71 to 0.97. In addition, VOC were also highly correlated with gaseous traffic-related pollutants, i.e. NO$_2$ ($r$=0.73 - 0.81) and CO ($r$=0.72 - 0.84). However, VOCs were moderately correlated with PM$_{10}$ ($r$=0.47 - 0.65). The correlation coefficients between PM$_{10}$ v.s. NO$_2$, and PM$_{10}$ v.s. CO were 0.74 and 0.69, respectively. Furthermore, the correlation coefficients of those three air pollutants from 1993 to 2006 had similar coefficients (0.71 and 0.66).

Table 3 shows the association between cardiovascular mortality and NO$_2$, CO and PM$_{10}$ in the longer period, from 1993 to 2006. By adding individual air pollutants to single-pollutant models, we found NO$_2$, CO, and PM$_{10}$ at lagged 0 day and lagged 1 day were significantly associated with cardiovascular mortality. At lagged 2 days, NO$_2$ and CO were associated with cardiovascular mortality. Significantly associated with cerebrovascular mortality were NO$_2$ and CO at lagged 0 to 2 days, and PM$_{10}$ at lagged 1 day. However, none of the air pollutants were associated with either of the mortality subcategories for ischaemic heart diseases or strokes in single-pollutant models. Although the RRs for ischaemic heart diseases and strokes were not significant, it is apparent from table 3 that more RRs for strokes were greater than 1, with relatively few RRs for ischaemic heart diseases being greater than 1.
The three pollutants, NO₂, CO, and PM₁₀ were then combined to construct two-pollutant models. However, the results of these models did not show any significant associations, neither for cardiovascular mortality nor for cerebrovascular mortality. We did not apply two-pollutant models to analyze mortality for ischaemic heart diseases because no air pollutant was significantly associated with outcomes in single-pollutant models.

Single-pollutant models showed no significant association between cardiovascular mortality and NO₂, CO and PM₁₀ in the shorter period from 2003 to 2006, as shown in Fig. 1. By contrast, we found that propane, iso-butane, propylene, benzene, m,p-xylene and o-xylene at lagged 0 day were significantly associated with cardiovascular mortality in single-pollutant models. However, we did not find the association between cardiovascular mortality and six VOCs lagged 1 to 2 days. Moreover, none of the VOC pollutants showed any significant associations with cerebrovascular mortality, ischaemic heart diseases, and strokes at any lags. Each of NO₂, CO, and PM₁₀ was then combined with each the VOCs respectively to construct two-pollutant models. However, no associations were found among 18 models.
4. Discussion

This study supports that traffic-related pollutants, NO$_2$, CO, and six VOCs were associated with cardiovascular mortality. Our findings on NO$_2$ were consistent with a cohort study in Netherlands, which reported that traffic NO$_2$ caused cardiopulmonary mortality at lagged 0 day (Hoek et al., 2002). Another study done in Rome, Italy, reported that out-of-hospital coronary heart disease deaths were significantly associated with CO at lagged 0 (Forastiere et al., 2005). The role of those VOCs in the association with mortality can be explained in two aspects. The first reason is that VOCs could be a better indicator of traffic exposure. A previous commuter study in central Taiwan has identified similar VOCs, including benzene and xylene, during commutes (Kuo et al., 2000). A micro-environment study suggested that VOCs could better represent resident’s exposure to traffic air pollution than PM in urban environment (Fischer et al., 2000). It has also been proposed that VOCs were mostly associated with fine particles (Odabasi et al., 2005). The other possible reason is that some of those VOCs are regarded as hazardous air pollutants (HAPs) and can cause more adverse effects.

In addition, PM$_{10}$ was also associated with cardiovascular mortality. Short-term elevations in PM$_{10}$ are capable of evoking cardiac arrhythmias, worsening heart failure, or triggering acute atherosclerotic/ ischemic cardiovascular complication (Brook et al., 2004). We observed increased cardiovascular mortality for increasing concentrations of PM$_{10}$ at
lagged 0 and lagged 1 day. This finding was similar but slightly different from a large, multi-city study, which reported that heart disease showed association primarily with concentrations at lagged 1 and lagged 2 days (Zeka et al., 2005). The lagged 0 effect could be explained due to the higher average concentration of PM$_{10}$ in this study (66.9 ug/m$^3$), compared with the average concentrations in the studied cities (15.9 – 35.0 ug/m$^3$) in the US. Zeka and colleagues also reported that the studies used only a single day rather than a distributed lag exposure will lower the effect estimates. This explained that the effects of NO$_2$ and CO on cardiovascular mortality persisted in all three days (lagged 0 to 2 days) while the past studies showed the effects on the same event day (lagged 0 day) (Forastiere et al., 2005; Hoek et al., 2002). Moreover, NO$_2$ and CO have more lagged day (from lagged 0 to 2 days) effects on cardiovascular mortality and cerebrovascular mortality than PM$_{10}$ (only lagged 0 and lagged 1 day), suggesting that more traffic-related pollutants have stronger effects.

NO$_2$, CO and PM$_{10}$ were associated with cardiovascular mortality from the period 1993 to 2006, but no associations were found in the data analysis for a rather shorter period, i.e. from 2003 to 2006. The reason why we did not observe the effect from the latter data analysis may be that the study duration was too short to reflect the statistical power.

Our two-pollutant models, i.e. NO$_2$ with PM$_{10}$, and CO with PM$_{10}$, or PM$_{10}$ with any of the six VOCs did not show any significant associations with cardiovascular mortality. The possible reasons can be drawn as follows. First, it has been reported that the effects estimates
for the indicators for fine particles were more stable in two-pollutant models. Our data do not allow a conclusion about the effects of fine particles, and further large studies incorporating different particle size would be helpful. Second, the results suggest that factors other than particle indicators from the air pollution mixture are important as well. Gaseous air pollutants related to vehicle emissions were more significantly associated with cardiovascular and cerebrovascular mortality than other particulate air pollutants in urban environment (Chan et al., 2006).

In addition, the highly association among VOCs implies that those VOCs are from similar sources. It should be noted that PM$_{10}$ are more multi-sourced, resulting in poorer correlation in concentration with those traffic-related VOCs.

It should be noted that ischaemic heart diseases were not associated with any air pollutants in this study. Given that there are only 2304 and 2757 cases from ischaemic heart diseases and strokes, respectively, over the 5112 study days. The insufficient sample size could be the possible reason why no air pollutants were significantly associated mortality for either ischaemic heart disease or strokes in our study.

Past studies have shown that indoor VOCs and in-vehicle VOCs concentrations are usually greater than outdoor concentrations (Chan et al., 1991; Son et al., 2003). Since the majority of people spend most of their time indoors, our study results might still under-estimate the effects due to the relatively low VOC concentrations in the PAMS station.
We used air pollution levels from air monitoring stations to represent individuals’ exposures. Therefore, we could not avoid the measurement error of misclassifying our subjects’ exposures to air pollutants. We used one air monitoring station to represent the residents within 1 km circle area. Such exposure misclassification can bias our outcomes towards either null or positive results, as reported in previous studies (Zeger et al., 2000; Zeka and Schwartz, 2004). Furthermore, patients with cardiovascular diseases are mostly elderly people, and many of them are disabled and may spend more of their time indoors than outdoors.

Regardless these limitations, we conclude that urban air pollution can increase cardiovascular mortality among adults in central Taiwan. Exposure to traffic-related NO₂, CO and PM₁₀, even at levels below current standards, has been already reported to increase the risk of respiratory symptoms (Nordling et al., 2008) and cardiovascular mortality (Zeka et al., 2005). Our study findings not only confirm the effects of those pollutants but also indicate a more sensitive traffic-related pollutants i.e. VOCs. Among the measured air pollutants, we conclude that traffic-related NO₂, CO, and VOCs are strong predictors of cardiovascular mortality than other pollutants in urban environments. But more comprehensive exposure measurements of traffic emissions are still needed in future epidemiological studies in order to confirm our findings of the associations between different pollution components and the cardiovascular mortality increase. Further toxicological studies are also recommended to
elucidate the biological mechanisms and pathogenetic processes in the cardiovascular system induced by urban air pollution.
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specific mortality: effects of lags and modification by city characteristics. Occup
Table captions

Table 1 Summary of daily mortality for cardiovascular diseases and environmental conditions in central Taiwan from 1993-2006.

Table 2 Pearson correlation coefficients for air pollutants in central Taiwan from 1 January 1993 to 31 December 2006.

Table 3 Relative risk of mortality for cardiovascular, ischaemic heart disease, and cerebrovascular disease, for IQR changes in air pollutant levels for different lag in single-pollutant model in central Taiwan from 1 January 1993 to 31 December 2006.
Table 1

<table>
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<th>Mortality data</th>
<th>Day</th>
<th>Count</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
<th>IQR</th>
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<td>Cardiovascular mortality(^1) (ICD-9: 410-411, 414, 430-437)</td>
<td>5112</td>
<td>7504</td>
<td>1.5</td>
<td>1.2</td>
<td>0</td>
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<td>Ischaemic heart disease (ICD-9: 410-411, 414)</td>
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<td>2304</td>
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<td>Strokes (ICD-9: 430-434)</td>
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<td>NO(_2) (ppb)</td>
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<td>28.7</td>
<td>10.6</td>
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<td>80.5</td>
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<td>CO (ppm)</td>
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<td>0.1</td>
<td>3.8</td>
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<td>PM(_{10}) (ug/m(^3))</td>
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<th>Volatile organic compounds(^2)</th>
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<th>Value</th>
<th>Mean</th>
<th>SD</th>
<th>Min</th>
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<td>Propane (ppb)</td>
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<td>4.3</td>
<td>2.23</td>
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<td>17.49</td>
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<td>Propylene (ppb)</td>
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</table>

SD: standard deviation, Min: minimum, Max: Maximum, IQR: inter-quarter range

\(^1\) Acute myocardial infarction [International Classification of Disease, 9th revision-Clinical Modification (ICD-9) 410]; other acute and subacute forms of ischemic heart disease (ICD-9, 411); other forms of chronic ischemic heart disease (ICD-9, 414); subarachnoid haemorrhage (ICD-9, 430); other intracerebral haemorrhage (ICD-9, 431); other and unspecified intracranial haemorrhage (ICD-9, 432); occlusion and stenosis of pre-cerebral arteries (ICD-9, 433); occlusion of cerebral arteries (ICD-9, 434) transient cerebral ischaemia (ICD-9, 435); acute but ill-defined cerebrovascular disease (ICD-9, 436); and other and ill-defined cerebrovascular disease (ICD-9, 437).

\(^2\) Data period available from 2003 to 2006
<table>
<thead>
<tr>
<th></th>
<th>NO₂</th>
<th>CO</th>
<th>PM₁₀</th>
<th>propane</th>
<th>i-butane</th>
<th>propylene</th>
<th>benzene</th>
<th>m,p-xylene</th>
<th>o-xylene</th>
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<td>NO₂</td>
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<td>o-xylene</td>
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<td>(ICD-9: 410-411, 414, 430-437)</td>
<td>Lag 0: 1.031 (1.007, 1.056)*</td>
<td>1.025 (1.006, 1.046)*</td>
<td>1.042 (1.016, 1.069)*</td>
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<tr>
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<td>Lag 1: 1.030 (1.006, 1.055)*</td>
<td>1.029 (1.009, 1.049)*</td>
<td>1.038 (1.012, 1.065)*</td>
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<td>Lag 2: 1.027 (1.003, 1.052)*</td>
<td>1.022 (1.002, 1.043)*</td>
<td>1.019 (0.994, 1.045)</td>
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<td><strong>Ischaemic heart disease</strong></td>
<td>Lag 0: 0.984 (0.943, 1.027)</td>
<td>0.984 (0.949, 1.021)</td>
<td>0.978 (0.934, 1.024)</td>
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<td>(ICD-9: 410-411, 414)</td>
<td>Lag 1: 0.975 (0.934, 1.018)</td>
<td>0.986 (0.950, 1.022)</td>
<td>0.980 (0.935, 1.028)</td>
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<td>Lag 2: 0.994 (0.951, 1.038)</td>
<td>1.007 (0.970, 1.045)</td>
<td>1.024 (0.994, 1.052)</td>
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<td><strong>Cerebrovascular disease</strong></td>
<td>Lag 0: 1.030 (1.001, 1.060)*</td>
<td>1.025 (1.002, 1.049)*</td>
<td>1.027 (0.996, 1.059)</td>
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<td>(ICD-9: 430-437)</td>
<td>Lag 1: 1.032 (1.004, 1.062)*</td>
<td>1.028 (1.005, 1.052)*</td>
<td>1.032 (1.001, 1.064)*</td>
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<td>Lag 2: 1.032 (1.003, 1.062)*</td>
<td>1.029 (1.005, 1.054)*</td>
<td>1.024 (0.993, 1.056)</td>
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<tr>
<td><strong>Strokes</strong></td>
<td>Lag 0: 1.014 (0.974, 1.055)</td>
<td>1.018 (0.986, 1.051)</td>
<td>1.019 (0.978, 1.063)</td>
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<td>(ICD-9: 430-434)</td>
<td>Lag 1: 1.019 (0.980, 1.059)</td>
<td>1.022 (0.989, 1.055)</td>
<td>1.019 (0.977, 1.062)</td>
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<td>Lag 2: 1.015 (0.976, 1.055)</td>
<td>1.016 (0.983, 1.049)</td>
<td>1.007 (0.965, 1.050)</td>
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</table>
Fig. 1 Relative risk of cardiovascular mortality for IQR changes in air pollutant levels for lag 0 to 2 days in single-pollutant model in central Taiwan from 1 January 2003 to 31 December 2006