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Acute and Subacute Effects of Urban Air Pollution on Cardiopulmonary Emergencies and Mortality: Time Series Studies in Austrian Cities

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Abstract: Daily pollution data (collected in Graz over 16 years and in the Linz over 18 years) were used for time series studies (GAM and case-crossover) on the relationship with daily mortality (overall and specific causes of death). Diagnoses of patients who had been transported to hospitals in Linz were also available on a daily basis from eight years for time series analyses of cardiopulmonary emergencies. Increases in air pollutant levels over several days were followed by increases in mortality and the observed effects increased with the length of the exposure window considered, up to a maximum of 15 days. These mortality changes in Graz and Linz showed similar patterns like the ones found before in Vienna. A significant association of mortality could be demonstrated with NO₂, PM_{2.5} and PM₁₀ even in summer, when concentrations are lower and mainly related to motor traffic. Cardiorespiratory ambulance transports increased with NO₂/PM_{2.5}/PM₁₀ by 2.0/6.1/1.7% per 10 µg/m³ on the same day. Monitoring of NO₂ (related to motor traffic) and fine particulates at urban background stations predicts acute effects on cardiopulmonary emergencies and extended effects on cardiopulmonary mortality. Both components of urban air pollution are indicators of acute cardiopulmonary health risks, which need to be monitored and reduced, even below current standards.

Keywords: air pollution; particulate matter; nitrogen dioxide; emergencies; mortality; time series

1. Introduction

In most European cities PM_{10} and NO_2 are used for surveillance of air quality, under the assumption that these air pollutants predict health effects best. NO_2 is monitored as the surrogate of motor traffic exhaust, which is measured more easily than ultrafine particles. However, it is still unclear whether NO_2 only modifies effects of PM_{10} on mortality [1] or if it is a key indicator of motor traffic and its health effects [2–4]. Recently European cities have in addition monitored $PM_{2.5}$, because a limit was set for its annual concentration, however, a standard for daily $PM_{2.5}$ concentration is still missing in Europe. Some health effects were found more closely related to NO_2 [5,6] which is more variable with distance to road and correlated with black carbon and ultrafines, in particular from diesel soot.

In multicentre studies similar associations between daily PM_{10} exposures and cardiorespiratory mortality were found in Europe and the United States [7,8]. Mortality increase with PM_{10} , however, was higher in Canada [9], while in Europe most pronounced mortality effects were seen in warmer cities with higher NO_2 and a larger NO_2/PM_{10} ratio [10]. This pattern was less pronounced in the United States, where the proportion of diesel cars is lower.

In Austria 55% of passenger cars and nearly all vans and trucks use diesel engines, because of the relatively low price of diesel fuel. In the capital Vienna a $10 \mu\text{g}/\text{m}^3$ change in $PM_{2.5}$ was associated with a 2.6% change of daily mortality and a $10 \mu\text{g}/\text{m}^3$ change in NO_2 with a 2.9% change of daily mortality, lagged 0–14 days. The increase of mortality [11] and hospital admissions [12,13] with PM and NO_2 was seen in Vienna even in years without violation of EU standards (PM_{10} annual average $40 \mu\text{g}/\text{m}^3$; PM_{10} daily average $50 \mu\text{g}/\text{m}^3$ exceeded on not more than 35 days per year; NO_2 hourly average $200 \mu\text{g}/\text{m}^3$ with maximal 18 exceedances per year). The present study investigates the associations of TSP, PM_{10} , $PM_{2.5}$, NO_2 , SO_2 and O_3 concentrations with daily mortality in a longer time series, in Graz, the 2nd largest and in Linz, the 3rd largest Austrian city, sharing similar populations and car fleets. The goal was to verify the results from Vienna in smaller cities and to find the best indicators for the prediction of short-term effects of urban air pollution on specific mortality. In addition to previously used generalized additive models (GAM), a case-crossover approach is used for the evaluation of air pollutant effects outside the heating season, to enable conclusions on motor traffic. As an additional outcome, cardiopulmonary emergencies transported to hospitals were available for a shorter time period in the city of Linz. These cases are also analysed in a case-crossover approach.

2. Study Population and Methods

The city of Graz is situated south of the Alps in a semi alpine basin, with only very little industrial emissions. Nevertheless the EU standard for PM_{10} is exceeded regularly in winter, because of weak natural ventilation, long lasting inversions and stagnant weather conditions, when a locally aged aerosol forms from motor traffic and domestic heating [14,15]. Similar to Vienna, the city of Linz is located north of the Alps on the Danube, where air exchange is much better, resulting in lower ambient

air pollution in winter, despite of a heavy industry conglomerate (iron, steel, chemistry). Like in Vienna SO_2 had been reduced in both cities to low concentrations before 1990, leaving particulate matter (from traffic and heating) and NO_2 (dominated by diesel powered vehicles) as the key pollutants [16], with data available after quality checks from 1990 to 2005 in Graz and from 1990 to 2007 in Linz. We used daily concentrations of pollutants (averaged over all available stations) as measures of exposure.

In Graz the number of inhabitants is only 261,540 (15% in comparison to Vienna) and in Linz 189,367 (11% in comparison to Vienna). From power calculations [17] we therefore had to choose longer time series than in Vienna, starting 1990. Methods used for monitoring of air pollutants and for modelling of missing data have been described earlier [12,15]. In short, time series from monitoring stations were used, which showed the highest correlation with all other urban background stations. Missing data were replaced by linear regression from the station with the second highest correlation to all other stations. In Graz exposure estimation was based on routine data of six stations, with five urban background stations monitoring TSP and NO_2 since 1990. PM_{10} was monitored at these stations since 2000 and $\text{PM}_{2.5}$ in 2000–2001 at one station only. Before 2000 PM_{10} was modelled from station specific relations to TSP and meteorological parameters. $\text{PM}_{2.5}$ could not be modelled in Graz, because relations to measured values were available only for a single year and a single station.

In Linz exposure estimation for the period 1990–2007 was based on six stations, four of them monitoring TSP and NO_2 since 1990, PM_{10} since 2000 and one station monitoring $\text{PM}_{2.5}$ in 2000–2001 and since 2005. Earlier missing data on $\text{PM}_{2.5}$ were modelled from TSP. SO_2 , O_3 and meteorological data were available for the complete period of observation in both cities. In Linz no sentinel data on influenza were available like in Vienna and Graz. In Graz sentinel data on influenza (registered 1990–2005 weekly and during epidemics daily) were used to adjust for the confounder of influenza epidemics, defined by an increase of incidence to $>3,500$ cases per week. Associations of air pollutants with hospital admissions (together with diagnoses at discharge) had been analysed in both cities before [12,13]. The present time series on daily mortality incidence in Graz and Linz used the same ICD-classes, overdispersed Poisson GAMs and lags (lag 0–1 day and polynomial distributed lags 0–7 and 0–14 days), which had been used in the Vienna study [11]. From death certificates the main or underlying cause of death is usually based on hospital diagnoses and high autopsy rates [18], ensuring a high diagnostic quality. Total mortality was analysed as well as deaths from specific causes, namely cardiovascular and respiratory diseases. In addition total mortality in the elderly (aged 65+) was investigated because this group might be more sensitive to effects of air pollution [19].

The model contained the following confounders: long-term trend and seasonality, day of the week, period of influenza epidemics and meteorological variables. The analysis started from building a base model without the air pollution variables. Thereby it was determined, what best described variability of daily mortality using all confounders. Day of the week and periods of influenza epidemics were modelled with dummy variables, other confounders were modelled using spline functions [20]. First the appropriate number of knots for the cubic regression spline representing long-term trend and seasonal variations was sought by minimising the sum of partial autocorrelation over 40 days [9]. Next meteorological factors were sequentially entered to the model: temperature, atmospheric pressure, humidity and changes of temperature and pressure between consecutive days. Different lags (0–3) and varying degrees of freedom were considered. Choice of lag structure was based on the Akaike Information Criterion [21].

Several aspects of air pollution effect on mortality were taken into account. The acute effect was modelled using linear and logarithmic function of mean of current and previous days. In addition the concentration-effect was further modelled as a spline function. Cumulative effects and a possible harvesting effect were investigated through a 3rd degree of polynomial distributed lag model. We also contemplated non-linear pollutant effects for the distributed lag model [22,23], but because the pollutants' effects were fairly linear we abstained from these for the sake of interpretability. Difference in magnitude of air pollution effect by season (warm/cold) and level of other pollutants (high/low) were examined by creating appropriate dummy variables to be included in interaction terms. All computations were done using R 2.5.0 software applying the mgcv package for GAMs. For interactions between NO₂ and PM at lag 0–1 we used models with interaction of PM₁₀ and high NO₂ and NO₂ and high PM₁₀, defining “high” as the median or above.

In Linz morbidity data were also available from eight years: daily emergency transports to hospitals had been registered and diagnoses encoded in 2000–2007. This period in Linz was too short for stable results on mortality by GAM because of the small population and a high correlation between daily temperature and fine particulate concentration [17]. Therefore a case-crossover approach [24] was chosen, both for cardiopulmonary emergency transports and for cardiopulmonary and total deaths: exposures on case days were compared by conditional logistic regression with those on referent days occurring on a day with the most similar temperature (less than 1 °C difference) in the same season [25–28]. To eliminate time trends we chose a bi-directional approach and a time window of 7–21 days before and after the case (two referent days for each day of occurrence). This way temperature and season were eliminated as confounders and an overlap between periods of occurrence and referent periods was prevented. The time window of 7–21 days was narrow enough to find a day with equal temperature in the same season and it left a minimum distance of seven days to the occurrence day of cases at lag 0. This minimum distance was reduced to six days by analysing lags of one day, but it was not possible to analyse distributed lags of 0–7 days and 0–14 days without overlap of occurrence days and referent days. Therefore only short latencies could be analysed in the case-crossover study.

3. Results

Daily means of particulate matter and NO₂ registered in Graz at five background stations and in Linz at three background stations and one roadside station are shown in Table 1. The distributions of the key pollutants PM₁₀ and NO₂ were similar, with higher median of NO₂ in Linz (38 µg/m³) than in Graz (32 µg/m³), but lower 95th percentile in Linz (62 µg/m³) than in Graz (68 µg/m³). The median/95th percentile of PM₁₀ was slightly higher in Linz (32/86 µg/m³) compared to Graz (29/73 µg/m³). The median/95th percentile of TSP in Graz were 39/100 µg/m³ and the median/95th percentile of PM_{2.5} in Linz were 19/49 µg/m³.

SO₂ concentrations were low most of the time (except for the first two winters observed in Graz): in Graz the median/95th percentile was 7/30 µg/m³ and in Linz 5/19 µg/m³. Ozone was constantly higher during summer (especially during the last summer seasons observed), but had been monitored at elevated locations (Graz 348 m, 450 m and 661 m; Linz 265 m and 335 m), which were not representative for the exposures in domestic areas, where PM and NO₂ had been monitored. In Graz the median/95th percentile) was 49/96 µg/m³ and in Linz 41/86 µg/m³.

Table 1. Descriptive statistics of pollution concentrations ($\mu\text{g}/\text{m}^3$) and health effects (average per day).

(A) NO₂	Graz 1990–2005	Linz 1990–2007	Linz 2000–2007	Vienna 2000–2004
Arithm. mean	35.7	39.4	34.6	23.0
Std. dev.	17.0	13.3	13.1	10.7
Median	31.9	38.4	32.8	21.3
5 th percentile	15.1	20.6	16.5	9.0
25 th percentile	24.1	30.1	26.0	14.9
75 th percentile	43.3	47.2	41.1	29.1
95 th percentile	68.5	62.2	58.0	43.0
(B) TSP	Graz 1990–2005	Linz 1990–2007	Linz 2000–2007	Vienna 2000–2004
Arithm. mean	45.8	-	-	30.6
Std. dev.	26.8	-	-	16.1
Median	38.8	-	-	27
5 th percentile	16.6	-	-	12
25 th percentile	27.4	-	-	19
75 th percentile	56.5	-	-	39
95 th percentile	100.2	-	-	61
(C) PM10	Graz 1990–2005	Linz 1990–2007	Linz 2000–2007	Vienna 2000–2004
Arithm. mean	34.0	38.1	33.31	30.2
Std. dev.	19.3	26.6	12.9	19.0
Median	29.1	31.9	28.2	25.6
5 th percentile	12.4	11.1	10.5	9
25 th percentile	20.7	20.4	18.4	16.8
75 th percentile	42.0	48.3	42.8	39.0
95 th percentile	73.4	85.8	73.8	66.8
(D) PM2.5	Graz 1990–2005	Linz 1990–2007	Linz 2000–2007	Vienna 2000–2004
Arithm. mean	-	22.4	20.5	16.3
Std. dev.	-	15.0	12.9	10.5
Median	-	18.8	17.1	13.7
5 th percentile	-	7.1	7.1	5
25 th percentile	-	12.5	11.7	8.9
75 th percentile	-	27.9	25.6	20.6
95 th percentile	-	49.3	45.9	36.2
(E) End-points	Graz 1990–2005	Linz 1990–2007	Linz 2000–2007	Vienna 2000–2004
Total deaths	6.9	5.6	5.4	43.9
Cardiovasc.	3.6	2.6	2.4	23.2
Respiratory	0.3	0.4	0.4	2.3
Ambulance calls	-	-	15.7	-
Method	GAM	GAM	Case cross-over	GAM

Most deaths occurred on Mondays and least on Sundays. Mortality rates were highest in winter. When in sensitivity analysis degrees of freedom for the seasonal variation were increased the effect estimates for the pollutants were reduced, as was to be expected. But the impact of moderate changes in the degrees of freedom was only minimal (data not shown). Among the weather parameters

temperature had the strongest effect both on mortality (in a typical u-shaped form) and on the estimates of the pollutants' effects. Indeed in most of the full models SO_2 seemed to be protective, which had been kept in the model as a sort of negative control despite of its low concentrations, for some endpoints and lags even significantly so, at least in Vienna and Linz. This implausible finding was removed or even reversed when temperature was not included in the models. This could eventually indicate some over-adjustment by temperature and was one of the reasons to stratify by temperature in the case-crossover models applied for the shorter time-series in Linz.

Figure 1. Per cent change in daily mortality, with 95% confidence intervals, in Graz (G) 1990–2005 and Linz (L) 1990–2007 per $10 \mu\text{g}/\text{m}^3$ increase in the respective pollutant measure over the preceding 2, 8, and 15 days (lag 0–1, lag 0–7, lag 0–14). Corresponding data [11] of Vienna (V) are shown for comparison.

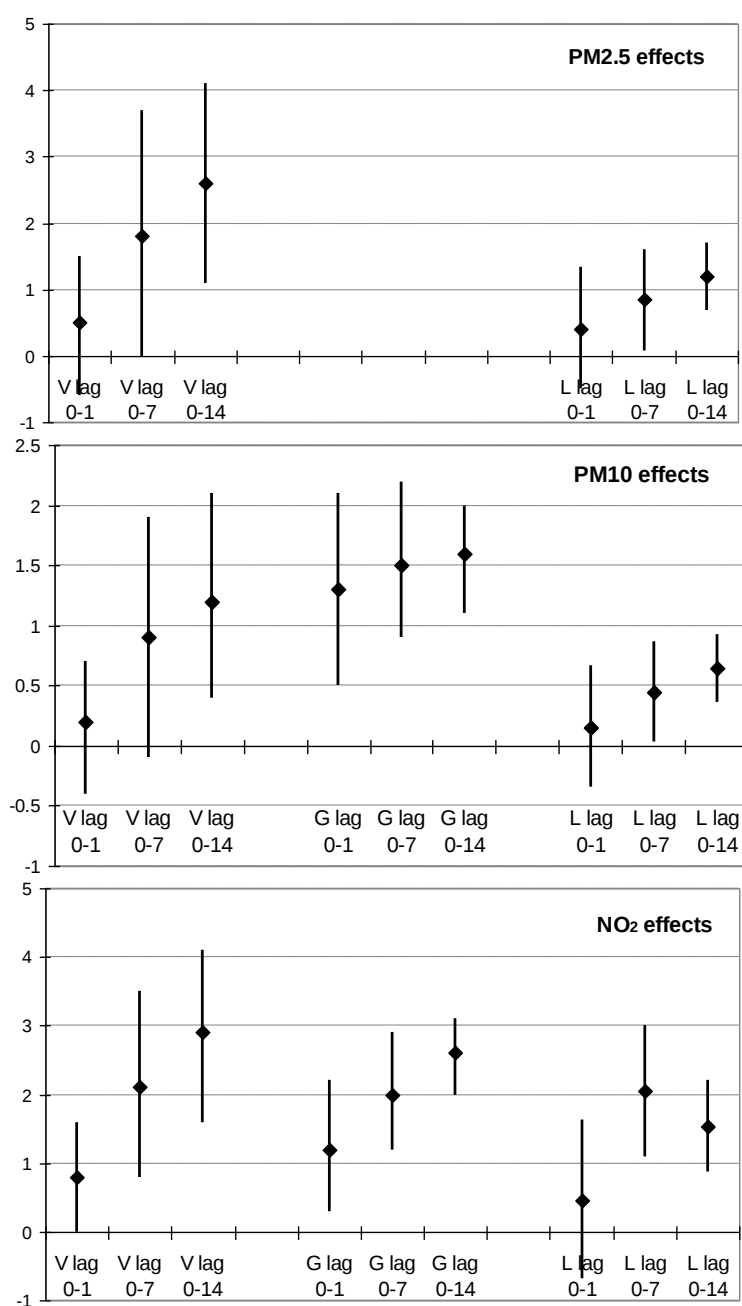


Figure 1 and Supplemental Table S1 give effect estimates for daily overall mortality. Results for special age groups and selected causes of death are also presented in Supplemental Table S1. Results from the Vienna study [11] are included for comparison. The temporal distribution of effect estimates is exemplified for several mortality end-points in Graz in Supplemental Figure S1. Simpler models measuring the average exposure over 8 or 15 days instead of calculating the additive effects of 8 or 15 days distributed lags gave rather similar cumulative results (data not shown).

In all three cities significant increases of mortality are seen with daily NO₂ and fine particle mass. No significant changes were found for SO₂ and O₃ (data not shown). After an increase of NO₂ or PM total mortality increases cumulatively with latency. Mortality increase was highest per µg/m³ for NO₂. In Graz a mortality increase of 2.6% (3.2% in males and 2.1% in females) was found per 10 µg/m³ of NO₂ for a lag of 0–14 days. Mortality increased less per µg/m³ of PM₁₀ and TSP, but significantly. Also in Linz significant increases of mortality for lags 0–7 and 0–14 days were observed, related to NO₂ (highest per µg/m³), PM_{2.5} and PM₁₀ (lowest per µg/m³). Except for a higher effect of NO₂ in Linz at lag 0–7 all pollutants show the highest effect at lag 0–14, indicating cumulative effects. Higher effects at longer lags are also seen in the results per interquartile range, with the highest estimate per IQR of NO₂. None of the interaction terms between NO₂ and PM₁₀ was found to be significant in Graz and in Linz (data not shown).

Missing data on PM₁₀ and PM_{2.5} had been inferred from other measured pollutants for periods of several years and these estimates were pooled with measured data. We did sensitivity analyses to justify this procedure by investigating the interaction of PM effects with a dummy variable for the periods in which PM₁₀ was calculated from TSP or PM_{2.5} from PM₁₀. Results for the main effects of the pollutant remained unchanged (both coefficient value and its significance), both dummy variable and interaction term were statistically not significant.

The mortality increase was significant in both cities for males and females, for the elderly and for cardiopulmonary diseases. Also for specific causes of death the highest estimates were found at a lag of 0–14 days, with highest mean estimates for respiratory deaths (COPD in particular), followed by cardiovascular deaths, which reached 2.4% per 10 µg/m³ of NO₂ in both cities at lag 0–14. In Graz at a lag of 0–7/0–14 days a mean increase of 2.9%/4.0% per 10 µg/m³ was estimated for deaths from ischemic heart diseases (IHD) related to NO₂ and 3.7%/4.3% related to PM₁₀. The corresponding figures for deaths from respiratory diseases (RD) were 8.1%/12.3% related to NO₂ and 3.4%/5.1% related to PM₁₀. Increases in Linz were lower, for RD in particular, with cardiovascular deaths more related to NO₂. However, all results for deaths from IHD and RD were also in Linz significantly related to NO₂ and PM₁₀ at longer latencies (IHD at lag 0–7 and 0–14, and RD at lag 0–14). Daily deaths from gastrointestinal diseases (used for control in the time series) did not show any associations with air pollutant concentrations in Graz and Linz (data not shown).

As a sensitivity analysis in Linz the period since 2000 (Figure 2) was studied separately when PM₁₀ was measured directly and no longer had to be calculated from TSP [15]. The key pollutants measured in Linz from 2000 to 2007 showed a similar distribution of concentrations like in Vienna from 2000 to 2004.

To exclude any effects from exposures to domestic heating (wood smoke), separate mortality analyses were run outside the heating seasons from June 1st to August 31st over eight years. Both approaches (case-crossover and GAM) estimate elevations of mortality after short latency (up to one

day). Despite of the shorter time period available for the case-crossover analysis and the limitation to summer months, confidence intervals were found narrower than in the full period analysed by GAM. Per $\mu\text{g}/\text{m}^3$ the effect on total mortality on the same day was highest for $\text{PM}_{2.5}$. But the case-crossover model yields significant elevations of mortality also for NO_2 and PM_{10} of the same and the preceding summer day.

Figure 2. Effect estimates in Linz 2000–2007: Per cent change in daily mortality, with 95% confidence interval, per 10 $\mu\text{g}/\text{m}^3$ increase in the respective pollutant measure. Full diamonds: Estimates from the years 2000–2007 on the same and on preceding day (lag 0 and lag 1), case-crossover model for the summer season (1st June to 31st August). For comparison Poisson GAM (open diamonds): estimates from the years 1990–2007 (full years) for the average of the same and preceding day (lag 0–1).

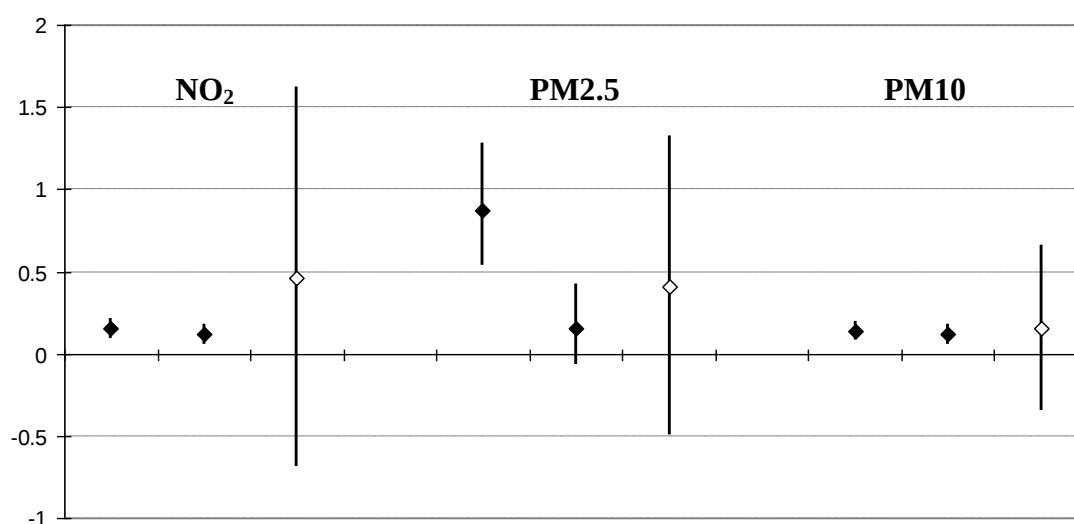


Table 2. Per cent increase in ambulance calls (with 95% confidence intervals) related to a 10 $\mu\text{g}/\text{m}^3$ increase in the respective pollutant measure. Linz (2000–2007).

	Lag 0 day	Lag 1 day	Lag 2 days
NO_2			
All cardiopulmonary	2.0 (0.8; 3.2)	−3.5 (−4.6; −2.4)	−
Cardiovascular	1.2 (−0.4; 2.8)	−4.2 (−5.7; −2.6)	−1.0 (−2.5; 0.6)
Respiratory	2.3 (−0.5; 5.1)	−3.2 (−5.9; −0.6)	0.3 (−2.4; 3.0)
Unclear cause	3.4 (1.2; 5.6)	−3.2 (−5.3; −1.0)	−0.1 (−2.3; 2.1)
$\text{PM}_{2.5}$			
All cardiopulmonary	6.1 (4.3; 7.8)	0.1 (−1.4; 1.7)	−
Cardiovascular	7.1 (4.7; 9.4)	0.3 (−1.8; 2.4)	2.5 (0.3; 4.7)
Respiratory	2.1 (−2.1; 6.3)	−1.6; −5.2; 2.0)	1.0 (−2.7; 4.7)
Unclear cause	3.7 (0.4; 7.0)	0.4 (−2.5; 3.4)	3.4 (0.3; 6.4)
PM_{10}			
All cardiopulmonary	1.7 (1.0; 2.3)	0.0 (−0.6; 0.6)	−
Cardiovascular	1.3 (0.9; 2.6)	−0.5 (−0.9; 0.7)	0.5 (0.1; 1.8)
Respiratory	2.7 (1.2; 4.2)	1.3 (−0.1; 2.7)	1.0 (−0.4; 2.5)
Unclear cause	1.6 (0.9; 3.3)	0.0 (−0.7; 1.6)	0.9 (0.2; 2.5)

In the case-crossover model significant elevations of cardiopulmonary emergencies (Table 2) were found related to NO₂, PM_{2.5} and PM₁₀, but not to O₃. Per µg/m³ morbidity effects at lag 0 were most pronounced for PM_{2.5}. No significant associations of cardiopulmonary emergencies or mortality were found with SO₂ (data not shown). At lag 1 NO₂, PM_{2.5} and PM₁₀ appeared to be protective indicating a sort of “harvesting” effect.

4. Discussion

The association of cardiopulmonary morbidity and mortality with PM, NO₂ and proximity to road are likely to share the same biological mechanisms: fine and ultrafine particles from combustion entering the lung, pulmonary reflexes over the autonomic nervous system affecting heart rate and arrhythmia, pulmonary inflammation, systemic inflammation from ultrafines entering the blood stream, endothelial dysfunction, reactive oxygen species, activation of leucocytes, platelets and other coagulation factors.

Health effects of air pollution are often classified into short- and long-term effects, although there is most probably a continuum of effects on the time scale, not yet completely understood. The results in Graz and Linz confirm earlier observations in Vienna [11], that prolonged mortality effects are higher than immediate effects, indicating that latent and cumulative effects outweigh harvesting (reduction of mortality after short increase due to premature deaths of most sensitive persons). Time series studies in Vienna had also found increases of respiratory hospital admissions related to PM_{2.5} [12] and of mortality from cardiovascular, cerebrovascular and respiratory diseases related to PM₁₀, PM_{2.5} and NO₂, most pronounced for a lag period of 15 days [12]. A time-series of daily hospital admissions in Vienna, Graz and Linz predicted the highest increases of cardiovascular admissions per µg/m³ for PM₁₀, followed by PM_{2.5} and PM₁₀, and also significant associations with daily NO₂ concentrations were found [13]. The present study in Linz supplements, that also cardiopulmonary emergency transports in ambulance cars (preceding hospital admissions) rise quickly with daily concentrations of PM_{2.5}, PM₁₀ and NO₂. In Vienna overall and cardiovascular mortality increased with latency and reached highest values per µg/m³ of NO₂, followed by PM_{2.5}. The same was found for overall mortality in Graz and Linz (Figure 1) and for cardiopulmonary mortality, indicating that legally binding limit values of daily PM₁₀ might be less important than limitation of fine and ultrafine particles associated with NO₂.

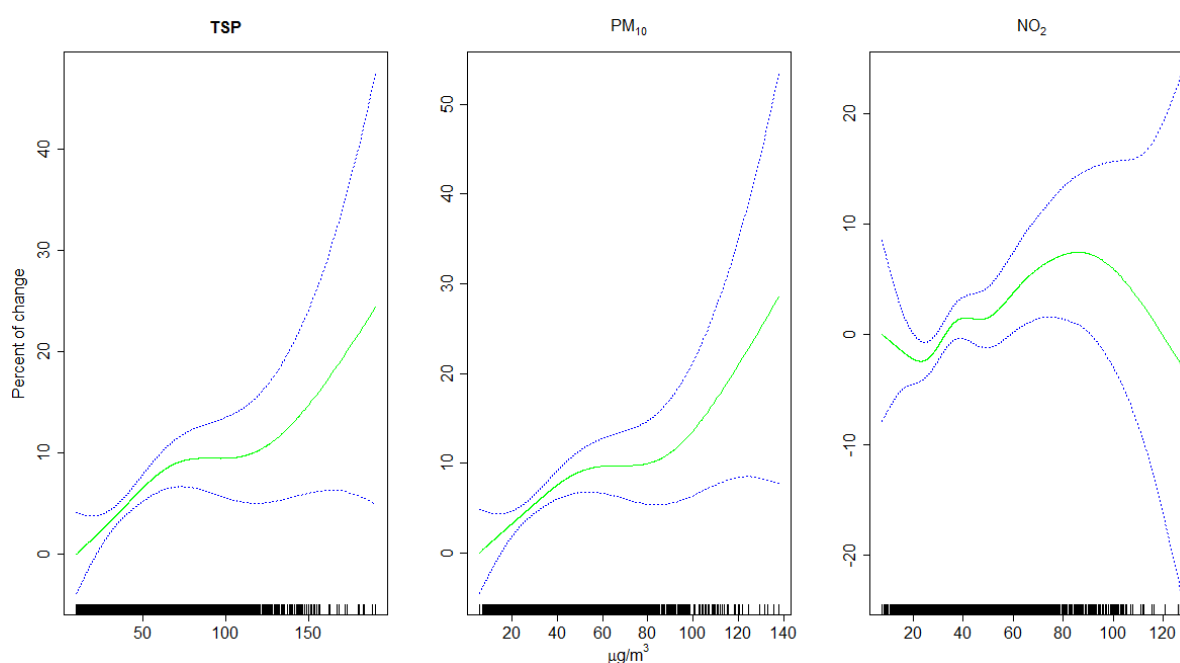
As an advantage of the time-series approach [29,30] the same population is used as exposed (on high pollution days) and as “control” (on low pollution days). Therefore no confounding by individual characteristics is probable. In contrast to cohort studies, time series are not confounded by risks spatially correlated to air pollution like traffic noise [31]. Daily means of urban air pollution are rather dependent on meteorological factors and day of the week (controlled in the present study) than on daily means of traffic noise, the socio-economic status or the daily dose of tobacco smoke (unrelated in time to ambient air pollution).

Differences between the three largest Austrian cities have to be examined in cohort studies, but we think that the present study found more similarities than differences of results. Differences in attribution of underlying cause of death to pulmonary or cardiovascular disease in elderly persons could be influenced by local diagnostic and coding peculiarities; however, overall mortality is mainly influenced by the age distribution of the population, which was found to be very similar in Graz, Linz

and Vienna [18]. Interactions of outdoor pollution with effects of active and passive smoking cannot be excluded; however, the same regulations apply for tobacco control in the three cities and smoking rates in 2006–2007 were similar [32]: Graz 20.9% (males 28.2%, females 14.2%) Linz 22.5% (males 23.2%, females 21.9%), Vienna 24.6% (males 30.3%, females 19.6%). A speculative explanation for the smaller effect of PM₁₀ on mortality in Linz is the short distance of one of the stations in Linz to a tunnel portal of a busy road, where higher concentrations were measured than in domestic areas, possibly leading to an overestimation of mean exposure and an underestimation of the slope in the concentration-response relationship.

In Linz a very quick rise of cardiopulmonary emergencies was observed with NO₂, PM_{2.5} and PM₁₀, even in summer (without heating) at levels below current standards. Together with the results on mortality this acute increase of cardiopulmonary morbidity indicates that urban air pollution is still a matter of concern and confirms the demand to lower PM_{2.5} standards and to introduce a limit value for the daily mean of PM_{2.5} in the European Union [33].

Figure 3. Per cent change in daily mortality, with 95% confidence intervals, in Graz (1990–2005), related to pollutants' concentration over the same and the preceding day (lag 0–1). Degrees of freedom: 3.1 (TSP), 3.3 (PM₁₀), 5.4 (NO₂).



In Graz a significant increase of total mortality was seen already at short latencies with TSP and PM₁₀, below a daily PM₁₀ concentration of 50 µg/m³, which is the EU limit for the daily mean. This increase of mortality was steep at concentrations below the current standard of PM₁₀ and flattened above (Figure 3). At very high concentrations we observed a second increase; however, the estimate from rare events is unreliable, as can be seen from the widening confidence intervals at high concentrations. What could be concluded from the concentration-response function is the absence of a threshold, as found by other investigators [8,34], but questions also the present regulations: Figure 3 seems to contradict the current methods of surveillance of daily PM₁₀ concentrations with reporting of the number of exceedances of 50 µg/m³ per year: From short-term mortality effects in Graz it seems to

be of low relevance if the PM_{10} concentration is a little below or above the standard of $50 \mu\text{g}/\text{m}^3$. On the other hand considerable health benefits are expected from reductions of pollutants also at locations where and at times when concentrations are very much below the current standard. For NO_2 the concentration-response function shows an increase of short-term mortality in the concentration range of about 25 to $90 \mu\text{g}/\text{m}^3$. The estimate gets unreliable at very low and especially at very high concentrations, but from Figure 3 no indication for a threshold higher than 25–30 $\mu\text{g}/\text{m}^3$ can be seen. On the other hand, also these effects level off, when daily means of about $35 \mu\text{g}/\text{m}^3$ are exceeded, but this flattening of the curve is only seen up to about $50 \mu\text{g}/\text{m}^3$, which is frequently exceeded due to motor traffic at urban background stations in domestic areas. Most pronounced are the prolonged effects of NO_2 on mortality, which can hardly be explained by the gas itself. More likely NO_2 is a surrogate for ultrafine particles from motor traffic and diesel exhaust in particular. Therefore this study supports demands for a better surveillance of traffic-related pollutants. Emission concentrations of ultrafine particles are regulated already in the EU [35].

The results of the present study contribute to the “suggestive evidence of a causal relationship” between exposure to traffic-related air pollution and total (mainly cardiovascular) mortality [8]: NO_2 indicates combustion and motor traffic. The contribution of motor traffic to PM increases with decreasing particle size. Therefore the effects on mortality, most pronounced per $\mu\text{g}/\text{m}^3$ of NO_2 and increasing with decreasing particle size of PM in Graz, Linz and Vienna, indicate health effects of motor traffic. In all three cities NO_2 is mainly of local origin, also in summer [36]. By contrast, in Linz and Vienna about 60% of PM_{10} is imported [37], while in Graz PM_{10} is also mainly of local origin, with a high traffic share [38]. Even in Linz the contribution of the industry to NO_2 exposure of the population is small and in Graz it is negligible. In contrast to studies in more industrialized areas [39], we therefore think that the results on NO_2 -related health effects in this study allow conclusions on health effects of motor traffic and diesel engines in particular. Up to now studies on proximity to roads [40,41] are unable to say, which part of health effects are related to the irritant gas NO_2 itself, to accompanying ultrafine particles or to traffic noise, which had also been suggested as a risk factor for cardiovascular diseases [42], or to other spatially correlated risk factors. For PM also Goodman *et al.* [43] observed that extended effects of air pollution exceed its acute effects. To our knowledge there are no studies on subacute mortality and NO_2 , however, in cohort studies higher cumulative effects had been found after much longer latencies for both PM [39,40] and NO_2 [3,4].

Data on PM_{10} were not available before 2000 and had to be modelled from TSP and meteorology. Nevertheless PM_{10} effects were found to exceed those of TSP, as expected. $PM_{2.5}$ had been monitored in Graz and Linz in 2000–2001 and in Linz since 2005. Despite of the necessity to model the $PM_{2.5}$ concentrations in Linz from PM_{10} in 2002–2004 and from TSP in earlier years, $PM_{2.5}$ was found to predict cardiopulmonary emergencies and mortality (Figure 1) very well.

Separate time series over the summer months did not reveal a significant effect of ozone on mortality, but significant elevations related to NO_2 , $PM_{2.5}$ and PM_{10} . This underlines the importance of motor traffic exhaust for acute effects on mortality in urban environments. The absence of mortality effects related to ozone was also observed in Vienna [11] and might be due to the sampling strategy in all three cities, which was different from other pollutants: Ozone was not monitored in densely populated areas like PM_{10} , NO_2 and SO_2 , but at elevations and downwind of urban precursor emissions, where few people lived. We cannot exclude small effects of O_3 on respiratory mortality in

summer months, detectable only in larger cities. A longer time series from Vienna with sufficient power could recently show ozone effects on mortality [43] using data of a central station.

SO₂ effects were not seen in Graz, Linz and Vienna during the available person-years of observation, however, already before the begin of this study, during the 1980s, Austria had achieved the highest SO₂ reduction among the signatory states of the Helsinki Protocol [12] and these low concentrations over most of the observation period, could explain the lack of association of SO₂ with mortality.

For Vienna emissions of NO₂ were estimated to decrease [44] from 2010 to 2020 by 513 tons only (road traffic NO₂ from 1,590 to 1,090 tons per year). During the same decade a reduction of PM₁₀ by 240 tons was estimated (road traffic from 770 to 570 tons per year). This does not give hope for a disappearance of related health effects in the near future.

5. Conclusions

The concentrations of NO₂ and small particles predicted health effects of urban air pollution in the three largest cities of Austria very well. Therefore we see no necessity at present to supplement surveillance by other indicators of combustion and motor traffic such as black carbon [45,46] or ultrafine particles [47]. Changing motor technology (direct injection, *etc.*) and fuel-additives, however, could possibly require additional indicators of ambient air pollution in the future.

From the results in Graz and Linz we conclude, that also smaller cities face acute health effects of air pollution from local motor traffic. This study supports the conclusion [12,33] that more stringent standards need to be adopted in Europe to protect public health from ambient PM_{2.5} and it adds, that also further reductions of NO₂ might be necessary on local, national and EU level, accompanied by epidemiological studies.

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Conflicts of Interest

The authors declare no conflict of interest.

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Supplemental Materials

Table S1. Lag-structure and degrees of freedom in the models. (A) Per cent increase in mortality (with 95% confidence intervals) related to a 10 $\mu\text{g}/\text{m}^3$ increase in pollution—Vienna. (B) Per cent increase in mortality (with 95% confidence intervals) related to a 10 $\mu\text{g}/\text{m}^3$ increase in pollution—Graz. (C) Per cent increase in mortality (with 95% confidence intervals) related to a 10 $\mu\text{g}/\text{m}^3$ increase in pollution—Linz (1990–2007).

(A) Vienna	Lag 0–1 day	Lag 0–7 days	Lag 0–14 days
	NO₂		
All deaths	0.8 (0.0; 1.6)	2.1 (0.8; 3.5)	2.9 (1.6; 4.1)
Cardiovascular	0.7 (−0.4; 1.8)	3.1 (1.2; 5.0)	4.6 (2.9; 6.3)
Ischemic heart	0.4 (−1.2; 1.9)	2.8 (0.3; 5.3)	4.5 (2.4; 6.6)
Other heart	1.6 (−1.1; 4.5)	4.6 (0.4; 9.1)	3.9 (0.4; 7.6)
Cerebrovascular	1.0 (−1.7; 3.9)	2.4 (−1.8; 6.7)	4.4 (0.8; 8.2)
Respiratory	2.3 (−0.6; 5.2)	5.7 (1.1; 10.6)	6.7 (2.7; 10.8)
COPD	1.9 (−2.4; 6.4)	9.9 (3.0; 17.3)	8.9 (3.0; 15.2)
	TSP		
All deaths	0.3 (−0.3; 0.9)	0.9 (−0.0; 1.9)	0.8 (0.0; 1.6)
Cardiovascular	0.3 (−0.5; 1.2)	1.6 (0.3; 3.0)	1.7 (0.7; 2.8)
Ischemic heart	0.8 (−0.4; 2.1)	2.6 (0.8; 4.5)	3.4 (1.9; 4.9)
Other heart	0.3 (−1.8; 2.4)	0.9 (−2.2; 4.1)	−1.3 (−3.7; 1.2)
Cerebrovascular	0.1 (−2.0; 2.3)	0.9 (−2.3; 4.1)	1.7 (−0.9; 4.3)
Respiratory	2.3 (0.0; 4.7)	3.7 (0.1; 7.4)	2.7 (−0.1; 5.6)
COPD	3.6 (0.2; 7.1)	8.5 (3.1; 14.2)	5.0 (0.7; 9.4)
	PM_{2.5}		
All deaths	0.5 (−0.6; 1.5)	1.8 (−0.0; 3.7)	2.6 (1.1; 4.1)
Cardiovascular	0.4 (−1.0; 1.8)	2.7 (0.3; 5.2)	3.8 (1.9; 5.8)
Ischemic heart	1.0 (−1.0; 3.1)	3.8 (0.6; 7.1)	5.5 (3.0; 8.1)
Other heart	0.9 (−2.6; 4.6)	3.0 (−2.2; 8.5)	1.5 (−2.5; 5.7)
Cerebrovascular	0.4 (−3.2; 4.1)	2.7 (−2.7; 8.4)	4.9 (0.7; 9.3)
Respiratory	3.9 (0.1; 7.8)	7.0 (0.9; 13.4)	6.4 (1.9; 11.2)
COPD	6.4 (0.6; 12.5)	14.0 (4.9; 24.0)	9.0 (1.9; 16.6)
	PM₁₀		
All deaths	0.2 (−0.4; 0.7)	0.9 (−0.1; 1.9)	1.2 (0.4; 2.1)
Cardiovascular	−0.2 (−0.6; 1.0)	1.4 (0.1; 2.8)	2.0 (0.9; 3.1)
Ischemic heart	0.4 (−0.7; 1.6)	2.1 (0.4; 3.9)	3.2 (1.8; 4.6)
Other heart	0.4 (−1.5; 2.4)	1.3 (−1.6; 4.3)	−0.1 (−2.4; 2.2)
Cerebrovascular	0.3 (−1.6; 2.4)	1.4 (−1.6; 4.4)	2.6 (0.3; 5.0)
Respiratory	2.1 (0.0; 4.2)	3.5 (0.1; 6.9)	3.0 (0.5; 5.5)
COPD	3.5 (0.4; 6.7)	7.9 (3.0; 12.9)	5.1 (1.3; 9.1)

Table S1. Cont.

(B) Graz	Lag 0–1 day	Lag 0–7 days	Lag 0–14 days
	NO₂		
All deaths	1.2 (0.3; 2.2)	2.0 (1.2; 2.9)	2.6 (2.0; 3.1)
65+	1.6 (−0.6; 3.8)	2.0 (1.1; 2.9)	1.9 (1.3; 2.5)
Before 65	1.2 (0.1; 2.3)	2.6 (0.7; 4.5)	2.5 (1.2; 3.9)
Male	1.3 (0; 2.7)	2.4 (1.2; 3.7)	3.2 (2.3; 4.1)
Female	1.1 (−0.2; 2.4)	1.8 (0.7; 2.9)	2.1 (1.3; 2.9)
Cardiovascular	0.3 (−1.1; 1.7)	1.9 (0.7; 3.0)	2.4 (1.6; 3.2)
Ischemic heart	0.5 (−1.6; 2.7)	2.9 (1.1; 4.8)	4.0 (2.7; 5.4)
COPD	7.8 (2.2; 13.7)	10.8 (5.9; 16.0)	13.9 (10.3; 17.5)
Respiratory	6.2 (1.7; 11.0)	8.1 (4.1; 12.2)	12.3 (9.5; 15.2)
	TSP		
All deaths	0.9 (0.4; 1.5)	1.2 (0.7; 1.7)	1.2 (0.9; 1.5)
65+	1.1 (−0.1; 2.3)	1.2 (0.7; 1.7)	0.1 (−0.3; 0.4)
Before 65	0.9 (0.3; 1.5)	1.4 (0.3; 2.4)	0.9 (0.2; 1.6)
Male	1.1 (0.3; 1.9)	1.5 (0.8; 2.2)	1.5 (1.1; 2.0)
Female	0.8 (0; 1.5)	1.1 (0.5; 1.7)	1.1 (0.6; 1.5)
Cardiovascular	1.2 (0.5; 2.0)	1.9 (1.2; 2.5)	0.9 (0.4; 1.3)
Ischemic heart	1.3 (0.1; 2.6)	2.6 (1.5; 3.6)	3.1 (2.4; 3.8)
COPD	2.9 (−0.1; 6.0)	3.7 (0.9; 6.4)	4.0 (2.1; 5.9)
Respiratory	1.9 (−0.6; 4.5)	2.4 (0.2; 4.7)	3.7 (2.2; 5.3)
	PM₁₀		
All deaths	1.3 (0.5; 2.1)	1.5 (0.9; 2.2)	1.6 (1.1; 2.0)
65+	1.3 (−0.4; 3.1)	1.5 (0.8; 2.2)	0.3 (−0.1; 0.8)
Before 65	1.3 (0.4; 2.2)	1.8 (0.4; 3.3)	1.5 (0.5; 2.5)
Male	1.4 (0.2; 2.5)	1.8 (0.8; 2.8)	2.0 (1.4; 2.7)
Female	1.2 (0.1; 2.3)	1.5 (0.6; 2.3)	1.3 (0.8; 1.9)
Cardiovascular	1.6 (0.6; 2.7)	2.5 (1.6; 3.4)	1.0 (0.4; 1.7)
Ischemic heart	2.0 (0.2; 3.8)	3.7 (2.3; 5.2)	4.3 (3.2; 5.3)
COPD	3.5 (−0.8; 7.9)	4.3 (0.6; 8.2)	4.6 (2.1; 7.2)
Respiratory	2.8 (−0.8; 6.4)	3.4 (0.4; 6.5)	5.6 (3.5; 7.8)
(C) Linz	Lag 0–1 day	Lag 0–7 days	Lag 0–14 days
	NO₂		
All deaths	0.5 (−0.7; 1.6)	2.0 (1.1; 3.0)	1.5 (0.9; 2.2)
Cardiovascular	0.1 (−1.5; 1.7)	2.3 (0.9; 3.7)	2.4 (1.5; 3.4)
Ischemic heart	1.0 (−1.4; 3.4)	3.8 (1.7; 5.9)	2.5 (1.0; 3.9)
COPD	1.1 (−4.1; 6.7)	1.1 (−3.6; 6.0)	1.5 (0.8; 2.3)
Respiratory	1.9 (−2.3; 6.3)	2.4 (−1.1; 6.0)	2.5 (1.5; 3.5)
65 +	0.4 (−0.9; 1.7)	2.0 (0.9; 3.1)	1.5 (0.7; 2.2)
Male	1.2 (−0.3; 2.7)	2.0 (0.6; 3.4)	1.8 (0.8; 2.8)
Female	0.0 (1.5; 1.5)	1.9 (0.6; 3.1)	1.9 (1.0; 2.8)

Table S1. *Cont.*

	PM_{2.5}		
All deaths	0.4 (−0.5; 1.3)	0.8 (0.1; 1.6)	1.2 (0.7; 1.7)
Cardiovascular	0.5 (−0.7; 5.9)	1.8 (0.7; 2.9)	3.3 (2.5; 4.1)
Ischemic heart	0.6 (−1.4; 2.6)	2.1 (0.4; 3.8)	2.0 (0.9; 3.2)
COPD	1.3 (−2.9; 5.6)	0.3 (−3.5; 4.4)	1.3 (0.8; 1.9)
Respiratory	2.5 (−0.7; 5.9)	2.1 (−0.8; 5.0)	3.6 (2.8; 4.3)
65 +	0.7 (−0.4; 1.7)	1.2 (0.3; 2.0)	1.4 (0.9; 2.0)
Male	0.4 (−0.8; 1.6)	0.5 (−0.6; 1.6)	0.8 (0.1; 1.6)
Female	0.0 (−1.2; 1.2)	0.6 (−0.4; 1.7)	0.7 (0.0; 1.4)
	PM₁₀		
All deaths	0.2 (−0.3; 0.7)	0.4 (0.0; 0.9)	0.6 (0.4; 0.9)
Cardiovascular	0.3 (−0.4; 1.0)	1.1 (0.5; 1.7)	2.0 (1.5; 2.4)
Ischemic heart	0.4 (−0.7; 1.5)	1.3 (0.4; 2.3)	1.3 (0.7; 2.0)
COPD	0.4 (−2.0; 2.8)	0.0 (−2.2; 2.2)	0.8 (0.5; 1.1)
Respiratory	1.1 (−0.8; 2.9)	1.0 (−0.6; 2.6)	2.1 (1.7; 2.5)
65 +	0.3 (−0.3; 0.8)	0.7 (0.2; 1.1)	0.9 (0.6; 1.2)
Male	0.1 (−0.6; 0.8)	0.2 (−0.4; 0.8)	0.3 (−0.1; 0.7)
Female	0.1 (−0.5; 0.8)	0.6 (0.0; 1.2)	0.7 (0.4; 1.1)

Figure S1. Temporal patterns of air pollution effects in Graz. The title of each graph indicates: cause of death, pollutant (pm.calc. indicating that PM₁₀ was calculated from TSP), pdl (indicating that those are PDL models), and 73/143 (7 and 14 days lags, 3rd degree polynomial). X axis: day, Y axis: change (in%) of daily number of deaths associated with 10 mg/m³ increase.

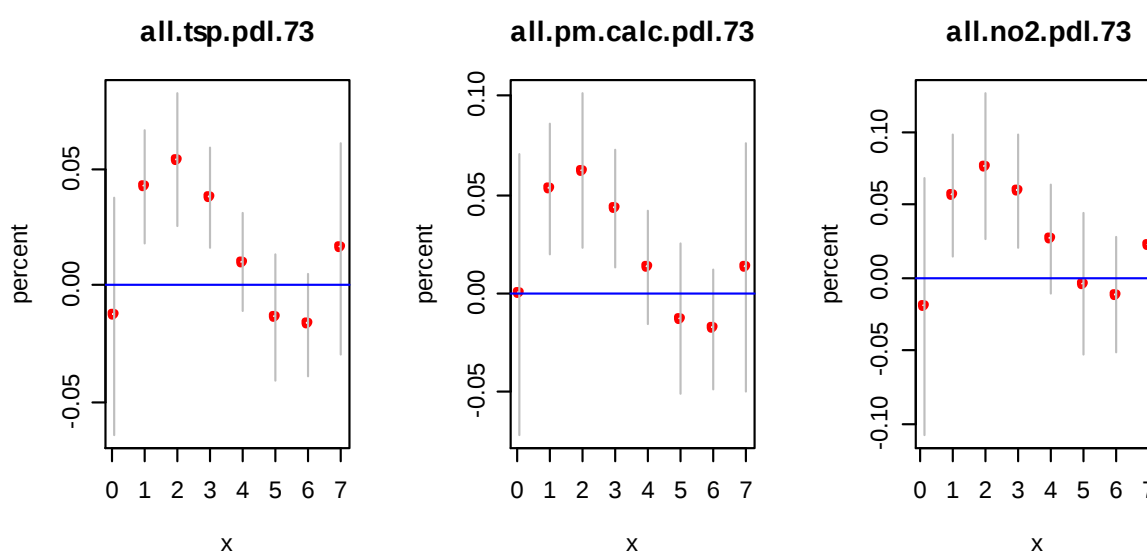


Figure S1. Cont.

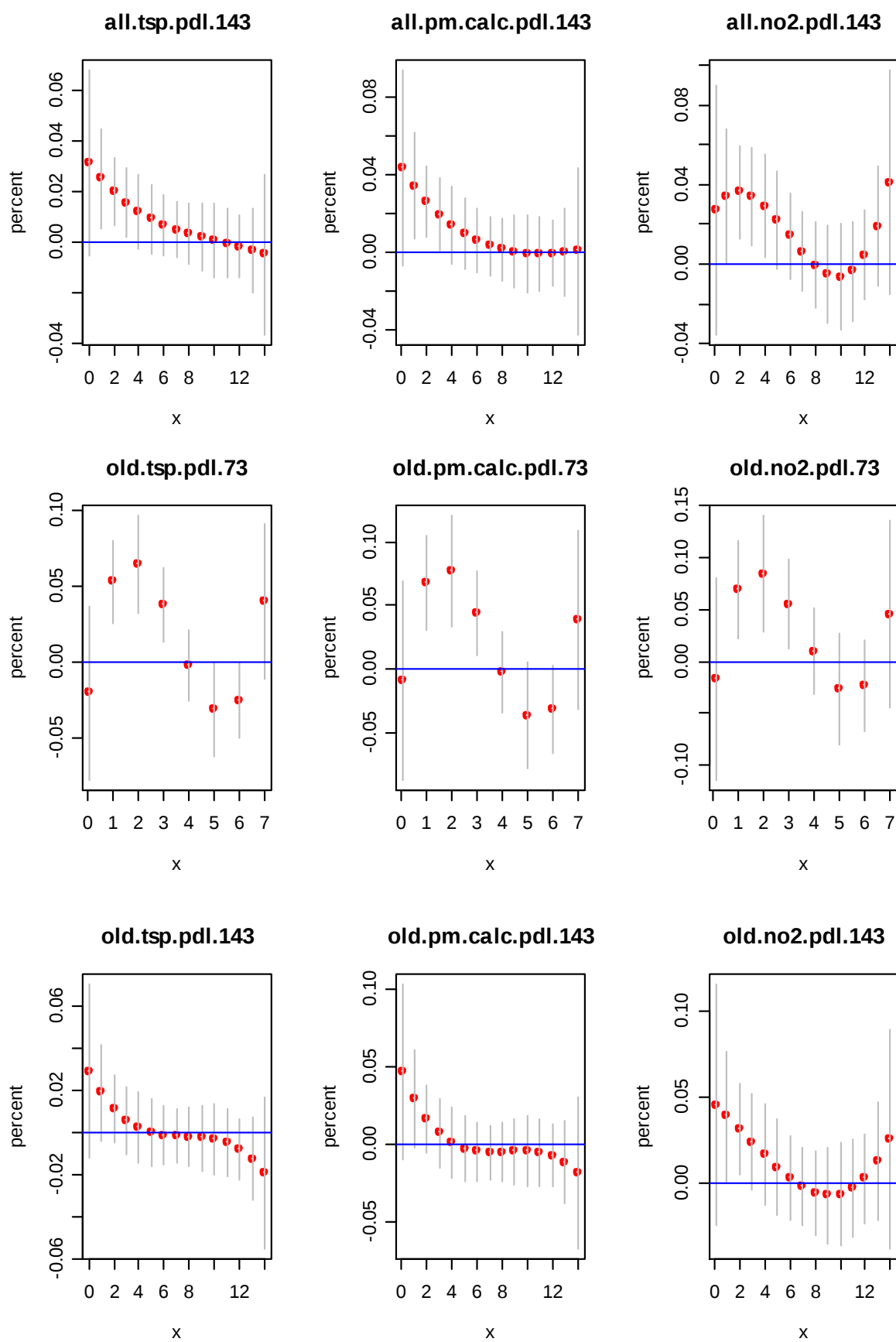


Figure S1. Cont.

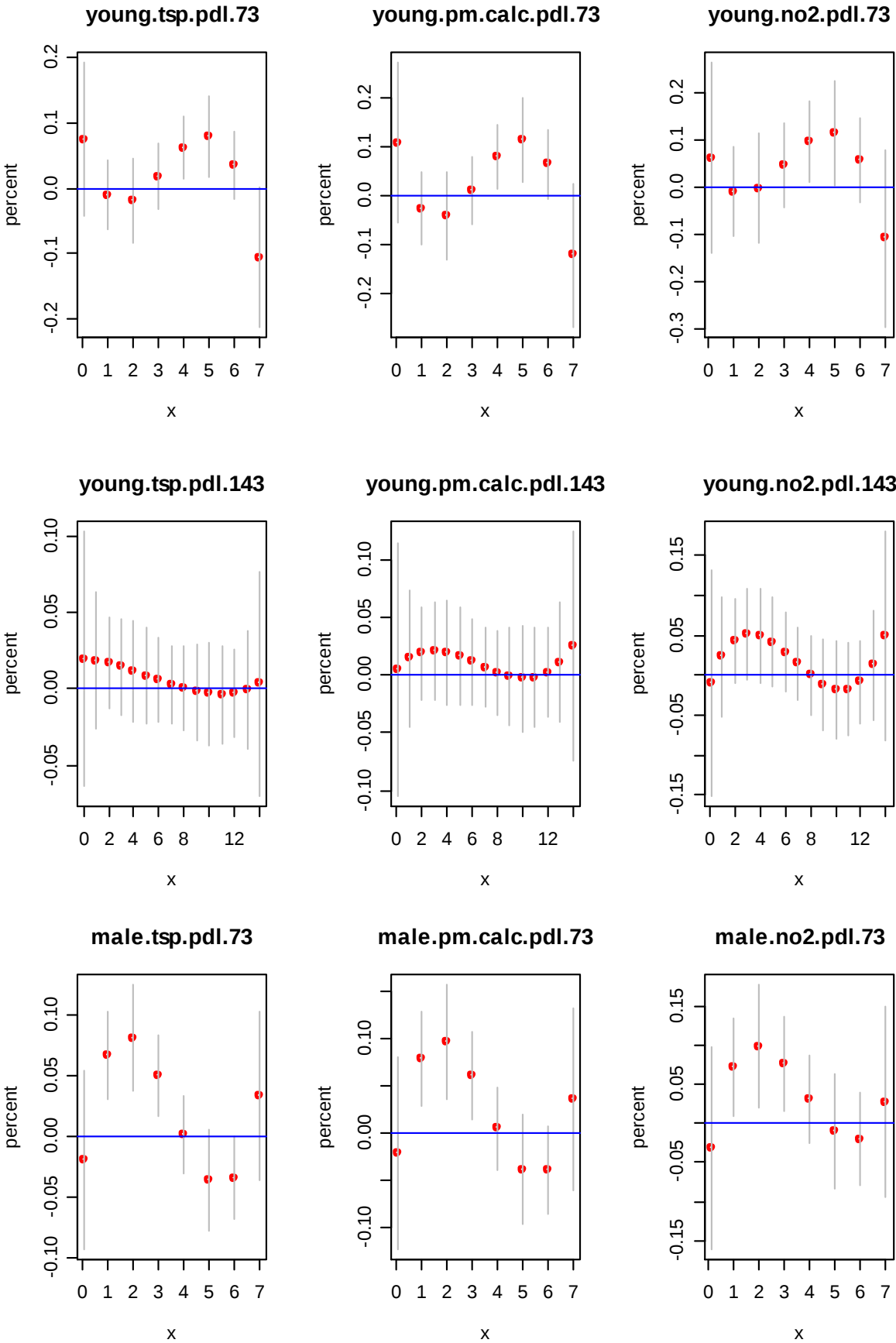


Figure S1. Cont.

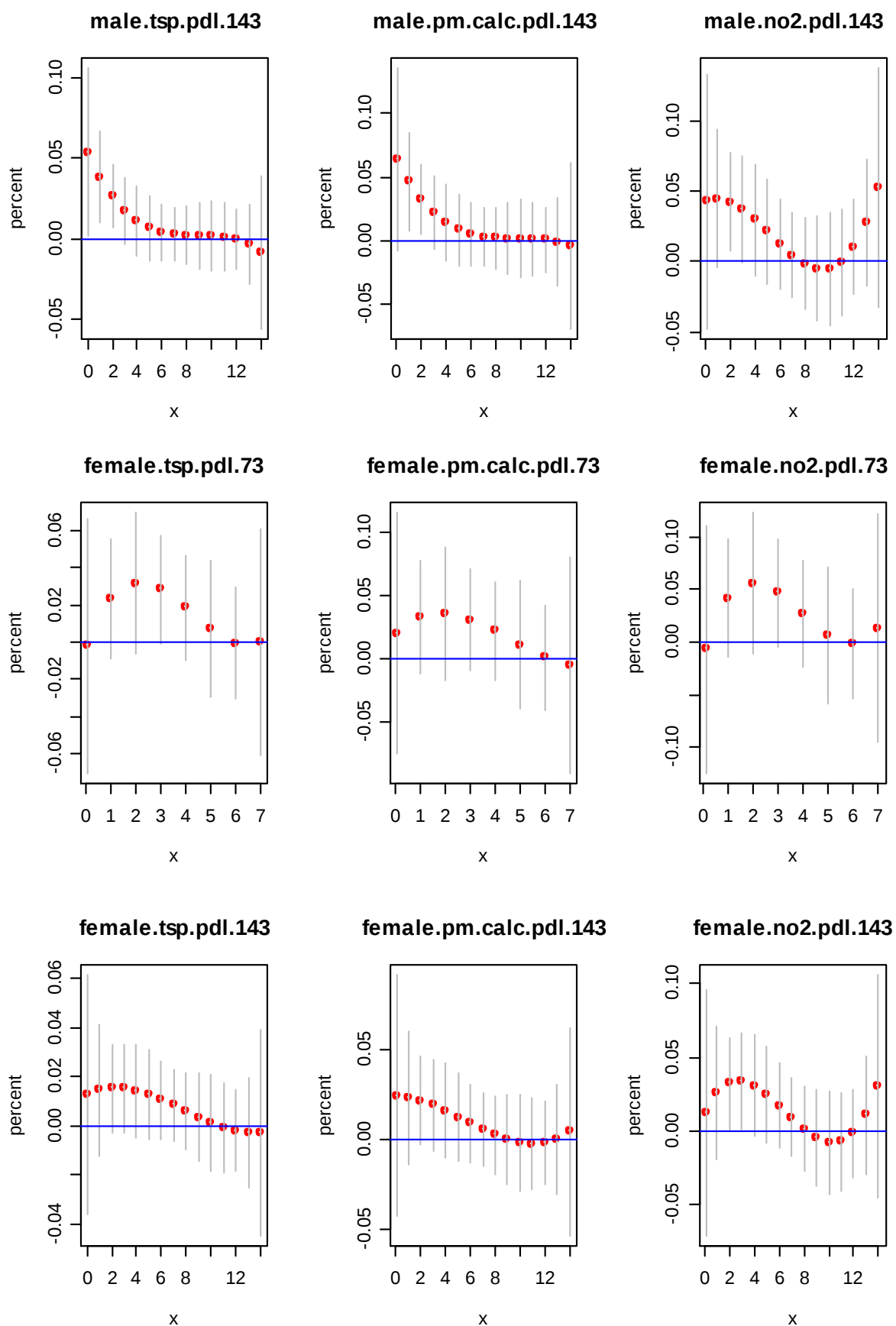


Figure S1. Cont.

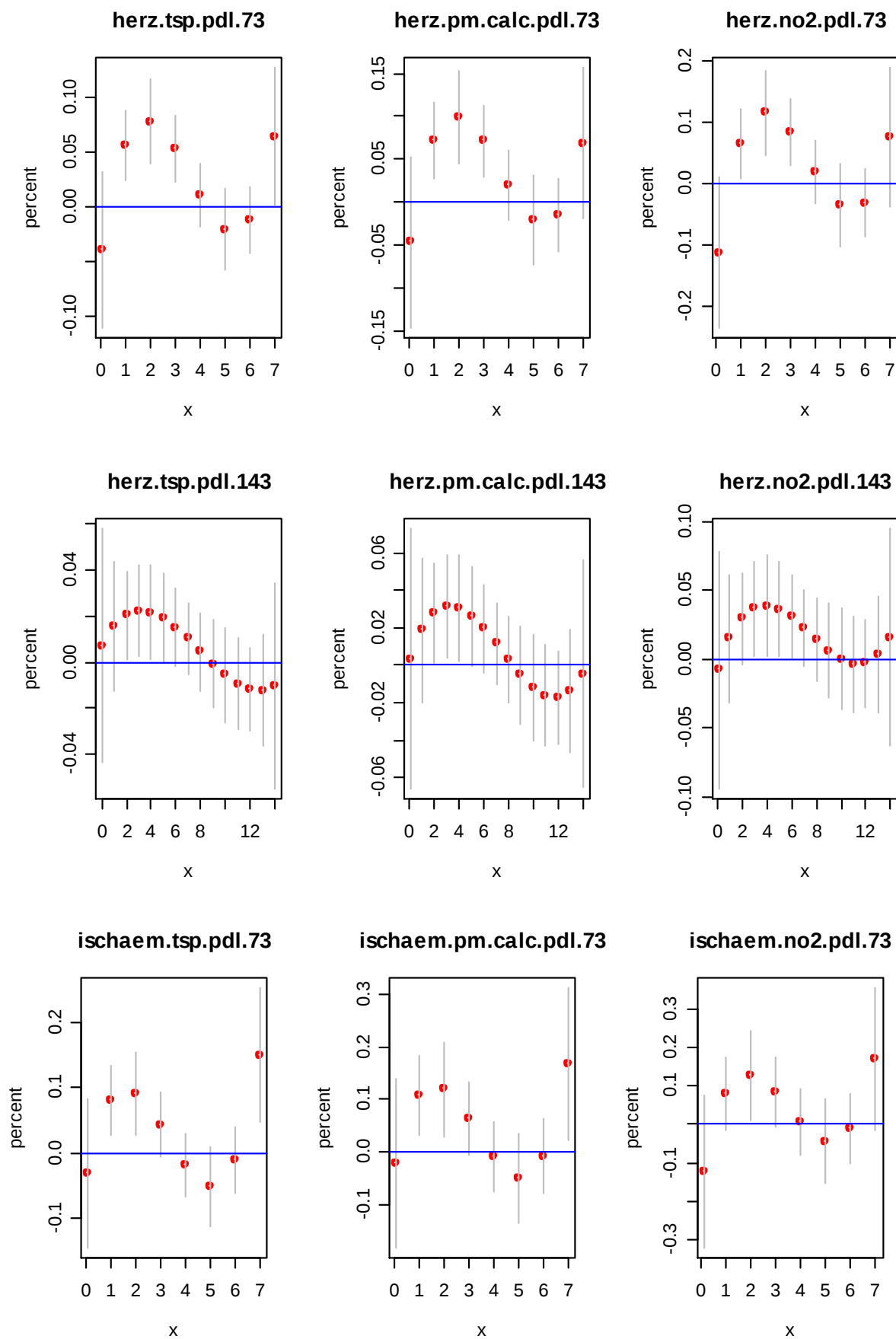


Figure S1. Cont.

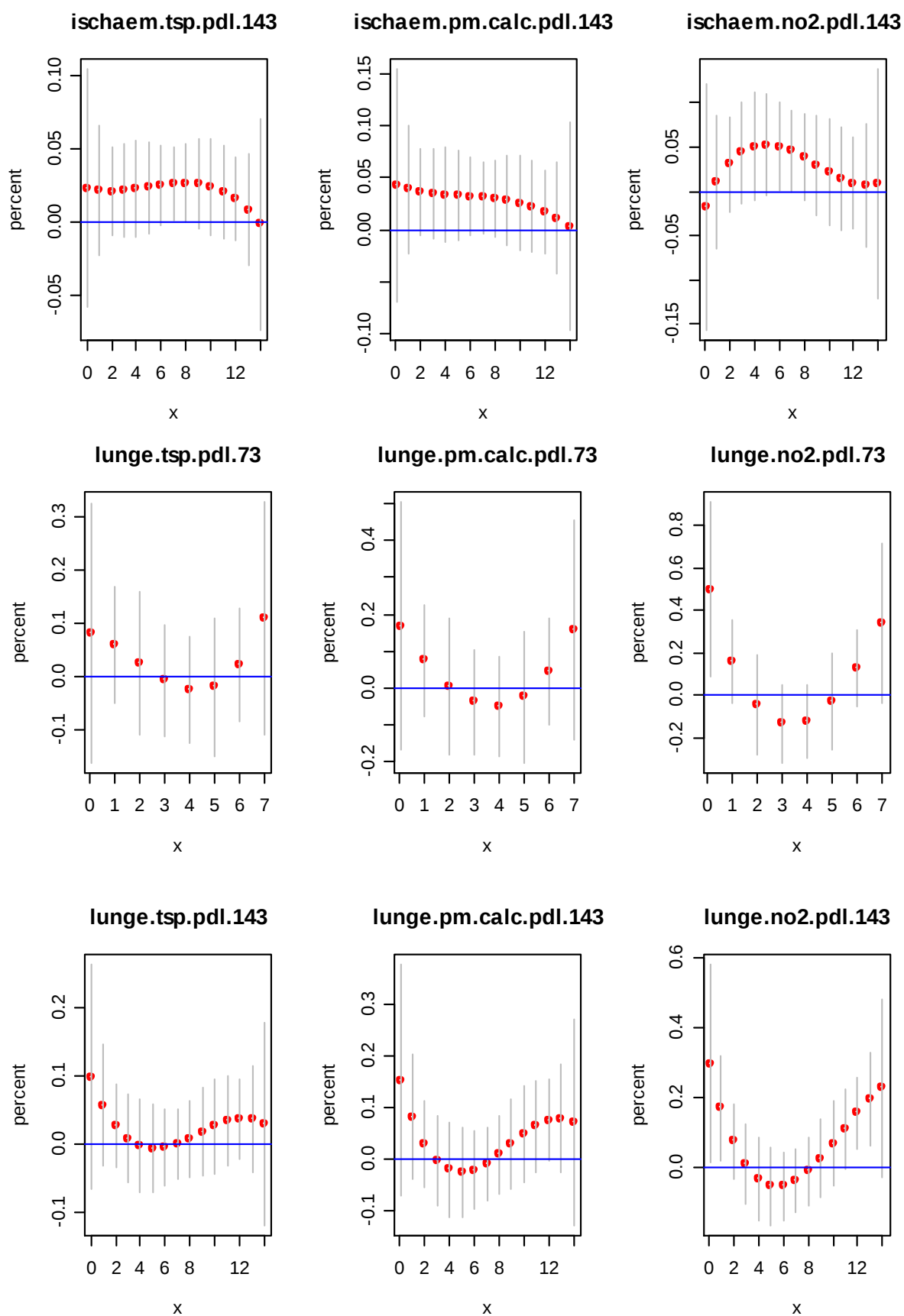


Figure S1. Cont.

