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Effects of long-range transported air pollution from vegetation fires on daily mortality and hospital admissions in the Helsinki metropolitan area, Finland

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ABSTRACT

Introduction: Fine particulate matter ($PM_{2.5}$) emissions from vegetation fires can be transported over long distances and may cause significant air pollution episodes far from the fires. However, epidemiological evidence on health effects of vegetation-fire originated air pollution is limited, particularly for mortality and cardiovascular outcomes.

Objective: We examined association between short-term exposure to long-range transported $PM_{2.5}$ from vegetation fires and daily mortality due to non-accidental, cardiovascular, and respiratory causes and daily hospital admissions due to cardiovascular and respiratory causes in the Helsinki metropolitan area, Finland.

Methods: Days significantly affected by smoke from vegetation fires between 2001 and 2010 were identified using air quality measurements at an urban background and a regional background monitoring station, and modelled data on surface concentrations of vegetation-fire smoke. Associations between daily $PM_{2.5}$ concentration and health outcomes on i) smoke-affected days and ii) all other days (i.e. non-smoke days) were analysed using Poisson time series regression. All statistical models were adjusted for daily temperature and relative humidity, influenza, pollen, and public holidays.

Results: On smoke-affected days, 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a borderline statistically significant increase in cardiovascular mortality among total population at a lag of three days (12.4%, 95% CI −0.2% to 26.5%), and among the elderly ($\geq$65 years) following same-day exposure (13.8%, 95% CI −0.6% to 30.4%) and at a lag of three days (11.8%, 95% CI −2.2% to 27.7%). Smoke day $PM_{2.5}$ was not associated with non-accidental mortality or hospital admissions due to cardiovascular causes. However, there was an indication of a positive association with hospital admissions due to respiratory causes among the elderly, and admissions due to chronic obstructive pulmonary disease or asthma among the total population. In contrast, on non-smoke days $PM_{2.5}$ was generally not associated with the health outcomes, apart from suggestive small positive effects on non-accidental mortality at a lag of one day among the elderly and hospital admissions due to all respiratory causes following same-day exposure among the total population.

Conclusions: Our research provides suggestive evidence for an association of exposure to long-range transported $PM_{2.5}$ from vegetation fires with increased cardiovascular mortality, and to a lesser extent with increased hospital admissions due to respiratory causes. Hence, vegetation-fire originated air pollution may have adverse effects on public health over a distance of hundreds to thousands of kilometres from the fires.

1. Introduction

Exposure to smoke from vegetation fires (wildfires, prescribed forest fires, and open-field burning related to agricultural practices) is a serious and recurring problem in many parts of the world. There is a need to better understand health effects of air

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pollution episodes caused by the fires, which is further emphasised by the growing risk of wildfires in many regions in the future as climate change proceeds (Flannigan et al., 2009; Moritz et al., 2012). Severe fire events can increase short-term air pollution in nearby areas manyfold (Heil and Goldammer, 2001; Liu et al., 2015; van Donkelaar et al., 2011). Moreover, the deterioration of air quality is not locally or even regionally limited. If the meteorological conditions are favourable, the fine particulate matter ($PM_{2.5}$, particles with aerodynamic diameter < 2.5 μm) emissions can be transported in the atmosphere for hundreds or even thousands of kilometres (Niemi et al., 2009; Sapkota et al., 2005; Witham and Manning, 2007), resulting in population exposure and health effects over wide areas. Globally, vegetation-fire smoke has been estimated to cause over 300,000 premature deaths annually (Johnston et al., 2012).

Of the various pollutants released from biomass burning, particulate matter is considered the most harmful to public health (Naeher et al., 2007). Numerous epidemiological studies have shown exposure to urban $PM_{2.5}$ to be associated with respiratory and cardiovascular morbidity and mortality (Brook et al., 2010; Pope and Dockery, 2006). However, epidemiological evidence on the health effects of particulate matter originating from vegetation fires is scarcer. This is mainly because of the more recent rise in attention as well as the challenges in exposure assessment related to these relatively short duration events. Studies have consistently shown exposure to vegetation-fire smoke to be positively associated with respiratory morbidity outcomes, such as physician and emergency department visits, hospital admissions, and increased medication use (Dennekamp and Abramson, 2011; Liu et al., 2015; Youssouf et al., 2014). However, there has been less research on mortality and cardiovascular effects, and the evidence from the existing studies is still inconclusive.

In Finland ambient air pollution levels are relatively low compared to other regions in Europe, and a major portion of the urban $PM_{2.5}$ is derived from long-range transport of emissions from other countries (Vallius et al., 2003). Episodically, the long-range transported pollution can multiply the normal daily $PM_{2.5}$ concentrations in vast areas in southern Finland (Niemi et al., 2005; Niemi et al., 2009; Saarikoski et al., 2007), resulting in the exceedance of the World Health Organization (WHO) health-based daily guideline value of 25 μg/m^3 (WHO, 2006). Strongest air pollution episodes are typically related to emissions from vegetation fires in eastern European countries (Russia, Belarus, Ukraine, Estonia, Latvia and Lithuania) at a distance of hundreds to thousands of kilometres from southern Finland (Niemi et al., 2009). For example, in August 2002 a two-week episode due to the Eastern European fires resulted in an additional population-weighted average daily $PM_{2.5}$ exposure of 15.7 μg/m^3 and has been suggested to have caused 17 premature deaths (Hänninen et al., 2009). The objective of our study is to provide epidemiological data on the mortality and morbidity effects related to short-term exposure to long-range transported vegetation-fire smoke in southern Finland.

2. Materials and methods

Association between short-term exposure to long-range transported $PM_{2.5}$ originating from vegetation fires and mortality and hospital admissions was studied in the Helsinki metropolitan area in Finland. The study area consists of four cities (Helsinki, Espoo, Vantaa, and Kauniainen), has a total population around one million, and covers a land surface area of 770 km^2. Study period included years from 2001 to 2010.

2.1. Health outcomes

Daily data on mortality counts for non-accidental, cardiovascular and respiratory causes were obtained from Statistics Finland and on acute hospital admission counts for cardiovascular and respiratory causes from the national inpatient registry maintained by the National Institute for Health and Welfare. Both datasets were coded according to the International Classification of Diseases 10th revision (ICD-10). For mortality we used the underlying cause of death and for hospital admissions the primary diagnosis. In cases where the same person was admitted to a hospital more than once during the study period, only re-admissions that occurred > 28 days after the previous hospital discharge (same primary diagnosis) were considered. Non-accidental mortality included all deaths excluding injuries, poisoning, and external causes (ICD-10 codes A00-R99). Cardiovascular outcomes for mortality and hospital admissions included all cardiovascular diseases (ICD-10 codes I00-I99). Respiratory outcome for mortality included all respiratory diseases excluding pneumoconiosis associated with tuberculosis (ICD10 codes J00-J64, J66-J99). Respiratory outcomes examined for hospital admissions were all respiratory diseases (ICD-10 codes J00-J99) and chronic obstructive pulmonary disease (COPD) or asthma (ICD-10 codes J40-J45, J47). Health effects were studied for all ages and for the age-group ≥ 65 years.

2.2. Exposure assessment

We studied the association between outdoor $PM_{2.5}$ and mortality and morbidity outcomes on i) days strongly affected by vegetation-fire smoke and ii) all other days (referred to as non-smoke days). Study-area wide daily average exposure to $PM_{2.5}$ (μg/m^3) from 2001 to 2010 was defined based on hourly measurements at a monitoring station representing urban background pollution levels. The monitoring station belongs to a municipal air quality monitoring network maintained by the Helsinki Region Environmental Services Authority.

Days affected by vegetation-fire smoke were identified based on air quality measurements at the urban background monitoring station and at another municipal monitoring station representing regional background pollution levels, as well as on an estimated contribution of long-range transported vegetation-fire smoke to the daily air pollution. Contribution of smoke was evaluated based on the Navy Aerosol Analysis and Prediction System (NAAPS). The NAAPS, run by the US Naval Research Laboratory, is a global forecast model that predicts smoke aerosol concentrations in the troposphere (model description and results available from www.nrlmry.navy.mil/aerosol). Predicted surface smoke concentrations are provided on a 1° x 1° grid (i.e. about 110 km longitude and 55 km latitude resolution in southern Finland) at 6-h intervals. Days were determined as smoke affected when the three following preconditions were fulfilled simultaneously: i) the 24-h average $PM_{2.5}$ concentration at the urban background monitoring site was ≥ 25 μg/m^3, ii) the 24-h average $PM_{2.5}$ or PM_{10} concentration at the regional background site was ≥ 20 μg/m^3, and iii) the NAAPS model indicated abundant smoke (surface smoke concentration maximum over 4 μg/m^3) or some smoke (surface smoke concentration 1–4 μg/m^3) due to long-range transport from vegetation fires. The validity of the NAAPS model data in identifying vegetation-fire originated episodes in southern Finland has been proved based on chemical composition analyses of particles on days influenced by long-range transported smoke (Niemi et al., 2009). Measurements at the regional background monitoring station were not available for ten smoke days in 2005. For these days, air quality data from an urban monitoring station was used to confirm the high $PM_{2.5}$ concentrations detected at the urban background monitoring station.

The primary cut point of 25 μg/m^3 for daily $PM_{2.5}$ at the urban background monitoring station was chosen for identification of days with high air pollution because it corresponds with the WHO health-based guideline value for daily average $PM_{2.5}$. The 25 μg/m^3 cut point is nearly three times the average daily background level of $PM_{2.5}$ measured in 2001–2010 at the urban background monitoring station. Hence, the majority of the pollution during the smoke days can be assumed not to be caused by local emissions sources. Furthermore, to increase the number of observations and the statistical power of the analyses, days with high levels of air pollution directly preceding or following the identified smoke days were also included in the analyses if the 24-h average $PM_{2.5}$ concentration at the urban background monitoring site was ≥ 20 μg/m^3.

To test the sensitivity of our effect estimates to the definition of days affected by vegetation-fire smoke, we also used two alternative ways of defining a smoke day: 1) including days for which the NAAPS model indicated abundant smoke due to long-range transport from vegetation fires, as well as days with high levels of air pollution directly preceding or following these (see above), and 2) including only days for which the NAAPS model indicated abundant or some smoke, excluding the preceding or following days with high levels of air pollution. For the identified smoke days, the preconditions on the measured PM concentration levels at the urban background and regional background monitoring stations described above were also met.

2.3. Potential confounders

Meteorological data for daily average temperature and relative humidity in Helsinki city centre were obtained from the Finnish Meteorological Institute. Weekly influenza counts were provided by the National Institute for Health and Welfare. The weekly count was assumed to represent influenza status of all days of the week. Because the distribution of influenza counts was skewed, a dummy variable was created using the 80th percentile (55 cases per week) as the cut point. Pollen data was provided by the Aerobiology Unit of the University of Turku. Daily pollen counts were measured at one site in Helsinki from the beginning of March to the end of August, which is the annual pollen period in Finland. The daily sum of the four most allergenic species (birch, mugwort, alder, and grass) was calculated. Because the distribution of the sum variable was highly skewed with almost two thirds of the days with no pollen at all, a dummy variable was created using 100 grains/m^3 as the cut point (the 95% percentile). For ozone, maximum 8-hour moving average concentration (μg/m^3) at the urban background monitoring station was used. Mass concentration of inhalable coarse particles ($PM_{2.5–10}$) was estimated by subtracting daily $PM_{2.5}$ from daily PM_{10} measured at the same monitoring station.

2.4. Statistical analyses

Associations between outdoor $PM_{2.5}$ concentrations on smoke and non-smoke days and the health outcomes were estimated using Poisson time series regression. Time trends and seasonality were controlled in the models with a triple interaction of year, month, and day of the week. This corresponds to a time-stratified case-crossover analysis where the stratification is made by selecting referent days that fall on the same day of the week and in the same month and in the same year as the index day, and where all the covariates are common (Levy et al., 2001; Lu and Zeger, 2007). An overdispersion parameter, estimated as the deviance divided by the degrees of freedom, was also included in the model. Statistical analyses were conducted in SAS for Windows 9.3 TS1M0.

All effect estimates were adjusted for temperature, relative humidity, influenza, pollen, and public holidays. The core model, which included the interaction term for year, month, and day of the week, was built step-by-step. First, the most appropriate exposure lag and the shape of the association with temperature was defined and selected based on the minimum Akaike's information criteria (AIC). For the exposure lag, 0 (same day) to 3 days lags were tested. To determine the shape of the association with temperature, we tested linear and quadratic terms as well as two and three days averaged temperatures. Second, the same procedure was repeated for relative humidity. Lastly, the dummy variables for public holidays, influenza, and pollen were included in all of the models even though they did not always reduce the AIC. We examined lags of 0–4 days between exposure and health outcomes. Effect estimates are reported as % change in risk per 10 μg/m^3 $PM_{2.5}$.

As a sensitivity analysis, the models were rerun after excluding potential influential observations and outliers, which were identified according to the model diagnostic statistics (Cook's D, studentized deviance, leverage). Sensitivity of the effect estimates for potential confounding air pollutants was also tested by adjusting the core model separately for daily ozone and inhalable coarse particle concentrations. The effect of pollen count was also examined dividing it into four categories (no pollen at all, 1–19, 20–99, 100+ grains/m^3).

3. Results

In 2001–2010, there were overall 65,362 deaths from non-accidental causes, 26,642 deaths from cardiovascular causes, and 4287 deaths from respiratory causes in the Helsinki metropolitan area. The total number of hospital admissions was 82,827 for cardiovascular causes, 68,450 for respiratory causes, and 13,982 for COPD/asthma. Descriptive statistics for daily deaths and hospital admissions for different age groups, and during smoke and non-smoke days are shown in Table 1.

During the ten-year study period, there were altogether 20 air pollution episodes related to long-range transported vegetation-

Table 1
Summary statistics for daily mortality and hospital admission counts in the Helsinki metropolitan area in 2001–2010.

	Smoke days (N=72)		**Non-smoke days** (N=3580)	
	Median	Range	Median	Range
Deaths				
Non-accidental				
All ages	17	10–28	18	4–36
≥65 years	14	6–25	14	1–29
Cardiovascular				
All ages	8	3–14	7	0–18
≥65 years	6	3–12	6	0–16
Respiratory				
All ages	1	0–4	1	0–8
Hospital admissions				
Cardiovascular				
All ages	22	6–37	23	5–66
≥65 years	16	3–26	16	3–54
Respiratory				
All ages	20	7–38	18	2–88
≥65 years	9.5	1–20	8	0–45
COPD/asthma				
All ages	4	0–11	3	0–22

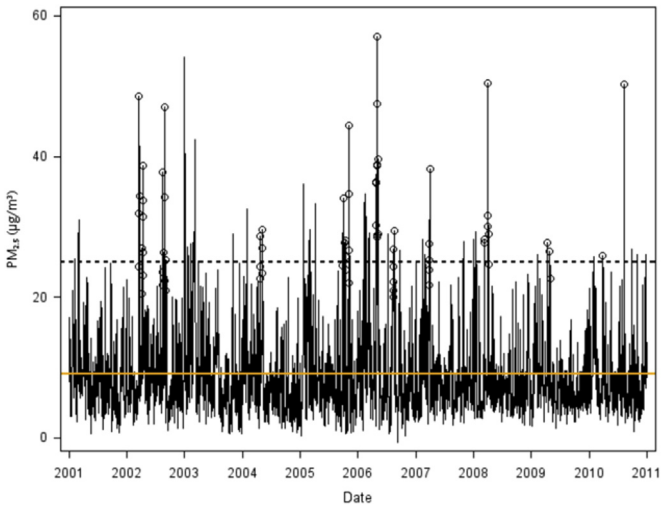

Fig. 1. Daily average $PM_{2.5}$ concentration measured at the urban background monitoring station in the Helsinki metropolitan area in 2001–2010. Air pollution episodes related to long-range transported smoke from vegetation fires are depicted with circles. Solid line represents long-term average $PM_{2.5}$ concentration, and dashed line the World Health Organization health-based guideline value for daily average $PM_{2.5}$.

fire smoke, 11 of which were classified to the 'abundant smoke' and 9 of which to the 'some smoke' -categories based on the NAAPS data. Duration of the episodes ranged from 1 to 11 days. The total number of smoke days included in the statistical analyses was 72. Of these, 44 occurred in March-May and 28 in August-November. Time series data for daily $PM_{2.5}$ concentrations and air pollution episodes related to long-range transported smoke from vegetation fires in the Helsinki metropolitan area are presented in Fig. 1. There were also a few high daily concentration peaks without smoke from vegetation fires, mainly caused by long-range transport of particles from the ordinary anthropogenic sources of Eastern Europe (Niemi et al., 2009).

Statistics for air pollutant concentrations, meteorological conditions, and other potential confounders for smoke and non-smoke days are presented in Table 2. During the identified smoke days, the average $PM_{2.5}$ concentration was over three times higher compared to non-smoke days. Correlations between daily $PM_{2.5}$ and inhalable coarse particles, ozone, temperature, and relative humidity were weak or very weak, both on smoke days (Spearman's rank order correlation coefficients −0.01, 0.33, −0.10, −0.09 for coarse particles, ozone, temperature, and relative humidity, respectively) and non-smoke days (correlation coefficients 0.03, 0.04, −0.03, 0.13, respectively).

3.1. Mortality

No association was detected between outdoor $PM_{2.5}$ concentration and non-accidental mortality on days affected by vegetation-fire smoke among all ages or those aged over 65 (Table 3). On non-smoke days, $PM_{2.5}$ was not associated with non-accidental mortality among all ages. However, in the ≥65 age-group there was a suggestive positive association at a lag of 1 day (1.9% per 10 µg/m³ increase in $PM_{2.5}$, 95% confidence interval −0.1% to 3.9%, $p=0.07$), which remained after excluding potential outliers from the analysis (Supplementary Table S1) and when using alternative definitions for a smoke day (Supplementary Table S3).

On smoke-affected days, outdoor $PM_{2.5}$ concentration was positively and nearly statistically significantly associated with mortality due to cardiovascular causes among all ages at a lag of three days (12.4%, 95% CI −0.2% to 26.5%, $p=0.054$, Table 3). Association with same day exposure also tended to be positive. The effect estimate at lag 3 increased when outliers were excluded from the analysis (18.9%, 95% CI −0.4% to 42%, $p=0.055$, Supplementary Table S1). In the age-group ≥65, association between smoke day $PM_{2.5}$ and cardiovascular mortality was borderline statistically significant both at lag 0 (13.8%, 95% CI −0.6% to 30.4%, $p=0.06$) and at lag 3 (11.8%, 95% CI −2.2% to 27.7%, $p=0.10$). After excluding outliers, the effect estimates at lags 0 and 3 increased and a suggestive positive effect also appeared at lag 4. The effect estimates both among all ages and the age-group ≥65 remained similar when using alternative definitions for a smoke day (Supplementary Table S3). On non-smoke days, association between $PM_{2.5}$ and cardiovascular mortality was not detected.

In case of respiratory mortality, the core models did not converge due to the small number of observations. Therefore, results for this outcome are not presented.

3.2. Hospital admissions

No association was detected between outdoor $PM_{2.5}$ and hospital admissions due to cardiovascular causes on smoke or non-

Table 2
Summary statistics for air pollutant concentrations and meteorological variables in the Helsinki metropolitan area in 2001–2010.

	N	Mean	Min	25th percentile	50th percentile	75th percentile	Max
Air pollution							
$PM_{2.5}$ (24 h average, µg/m³)							
Smoke day	72	30.0	20.0	24.3	27.7	34.2	57.0
Non-smoke day	3580	8.6	0	4.9	7.2	10.8	54.1
$PM_{2.5-10}$ (24 h average, µg/m³)							
Smoke day	72	18.0	0	7.0	11.8	24.5	88.6
Non-smoke day	3580	6.5	0	3.1	5	7.8	87.9
O_3 (8 h moving average, µg/m³)							
Smoke day	72	89.7	35.4	66.1	88.8	108.8	139.8
Non-smoke day	3579	61.9	1.5	47.9	61.3	75.0	151.8
Meteorological variables							
Temperature (24 h average, °C)							
Smoke day	72	11.2	1.3	6.3	9.1	17.2	24.6
Non-smoke day	3580	6.3	−22.6	0.1	6.2	13.9	25.7
Relative humidity (24 h average, %)							
Smoke day	72	70	39	56	71	83	96
Non-smoke day	3580	80	36	73	83	89	100

Table 3
Percentage change (95% confidence interval) in mortality for a 10 μg/m^3 increase in $PM_{2.5}$ on smoke-affected and non-smoke days.

	Smoke day (N=72)		**Non-smoke day** (N=3580)	
	% Change	**95% CI**	**% Change**	**95% CI**
All non-accidental				
All ages				
Lag 0	0.2	(−7.6, 8.6)	0.2	(−1.5, 2.0)
Lag 1	−4.0	(−11.5, 4.2)	1.3	(−0.5, 3.1)
Lag 2	2.5	(−5.6, 11.3)	1.0	(−0.9, 2.8)
Lag 3	1.2	(−6.6, 9.7)	0.2	(−1.7, 2.1)
Lag 4	1.0	(−6.9, 9.6)	-0.5	(−2.4, 1.3)
≥65 years				
Lag 0	2.4	(−6.5, 12.0)	0.6	(−1.4, 2.6)
Lag 1	−3.4	(−11.9, 5.8)	1.9	(−0.1, 3.9)
Lag 2	2.7	(−6.4, 12.8)	1.6	(−0.5, 3.8)
Lag 3	0.1	(−8.6, 9.7)	1.0	(−1.2, 3.2)
Lag 4	−1.4	(−10.2, 8.2)	0.0	(−2.1, 2.1)
All cardiovascular				
All ages				
Lag 0	8.0	(−4.7, 22.4)	−1.1	(−3.9, 1.7)
Lag 1	−4.0	(−15.4, 9.0)	0.5	(−2.4, 3.5)
Lag 2	4.3	(−8.3, 18.7)	0.7	(−2.2, 3.7)
Lag 3	12.4	(−0.2, 26.5)	−1.1	(−3.9, 1.7)
Lag 4	3.1	(−9.3, 17.2)	−1.8	(−4.6, 1.0)
≥65 years				
Lag 0	13.8	(−0.6, 30.4)	−1.0	(−4.0, 2.1)
Lag 1	−2.4	(−15.0, 12.1)	1.7	(−1.4, 4.8)
Lag 2	5.7	(−8.3, 21.8)	1.7	(−1.6, 5.0)
Lag 3	11.8	(−2.2, 27.7)	−0.6	(−3.9, 2.7)
Lag 4	3.7	(−9.9, 19.3)	−1.6	(−4.7, 1.6)

Table 4
Percentage change (95% confidence interval) in hospital admissions for a 10 μg/m^3 increase in $PM_{2.5}$ on smoke-affected and non-smoke days.

	Smoke day (N=72)		**Non-smoke day** (N=3580)	
	% Change	**95% CI**	**% Change**	**95% CI**
All cardiovascular				
All ages				
Lag 0	−3.5	(−10.5, 4.0)	0.2	(−1.5, 2.0)
Lag 1	4.7	(−2.6, 12.6)	0.8	(−0.9, 2.5)
Lag 2	2.6	(−4.8, 10.5)	0.0	(−1.6, 1.7)
Lag 3	2.0	(−5.4, 10.0)	0.7	(−0.9, 2.3)
Lag 4	−0.7	(−8.2, 7.4)	0.5	(−1.1, 2.1)
≥65 years				
Lag 0	1.5	(−6.9, 10.6)	−0.4	(−2.2, 1.5)
Lag 1	4.1	(−4.4, 13.4)	−0.2	(−2.1, 1.7)
Lag 2	5.1	(−3.8, 14.8)	−1.3	(−3.1, 0.6)
Lag 3	-0.2	(−8.8, 9.3)	0.5	(−1.4, 2.4)
Lag 4	-5.4	(−13.8, 3.9)	−0.2	(−2.0, 1.7)
All respiratory				
All ages				
Lag 0	1.0	(−7.3, 10.1)	1.3	(−0.7, 3.3)
Lag 1	1.7	(−6.7, 10.8)	0.7	(−1.2, 2.7)
Lag 2	3.0	(−5.5, 12.3)	−0.9	(−2.8, 1.0)
Lag 3	0.3	(−8.2, 9.4)	−0.7	(−2.6, 1.2)
Lag 4	0.5	(−8.3, 10.2)	−0.4	(−2.3, 1.5)
≥65 years				
Lag 0	10.5	(−2.2, 24.8)	−0.4	(−3.2, 2.4)
Lag 1	4.6	(−7.6, 18.5)	−0.9	(−3.6, 2.0)
Lag 2	6.3	(−5.8, 19.9)	−0.6	(−3.3, 2.2)
Lag 3	-2.8	(−14.2, 10.0)	−0.5	(−3.2, 2.2)
Lag 4	2.3	(−10.3, 16.6)	−0.4	(−3.0, 2.3)
COPD/asthma				
All ages				
Lag 0	11.3	(−5.7, 31.4)	0.2	(−3.7, 4.2)
Lag 1	6.4	(−10.0, 25.7)	0.7	(−3.2, 4.8)
Lag 2	15.8	(−2.1, 37.0)	−0.4	(−4.5, 3.8)
Lag 3	−4.9	(−20.7, 14.1)	−0.2	(−4.3, 3.9)
Lag 4	−2.6	(−19.8, 18.2)	−1.5	(−5.3, 2.5)

smoke days, neither among all ages nor the elderly (Table 4). However, when the definition for a smoke day was restricted to episodes for which the NAAPS model indicated abundant smoke from vegetation fires, there was a suggestive positive association between smoke day $PM_{2.5}$ and cardiovascular hospital admissions at lag 1 among all ages (7.1%, 95% CI −1.2% to 16.0%, $p=0.10$, Supplementary Table S4).

On smoke days, outdoor $PM_{2.5}$ was not associated with hospital admissions due to all respiratory causes among all ages. However, there was an indication of a positive association in the age-group ≥65, especially at lag 0 (10.5%, 95% CI −2.2% to 24.8%, $p=0.11$, Table 4). The effect estimate at lag 0 remained high after excluding outliers from the analysis (Supplementary Table S2). When restricting the definition for a smoke day only to the days for which the NAAPS model indicated abundant or some smoke (excluding preceding or following days with high levels of air pollution), the effect at lag 0 decreased considerably while the effect at lag 1 increased (Supplementary Table S4). However, it should be noted that the statistical power of the sensitivity analyses concerning the alternative ways of defining smoke days is limited by the lower number of smoke day observations (N=48). On non-smoke days, effect estimate for all respiratory hospital admissions was positive although not statistically significant among all ages at lag 0. The effect strengthened after exclusion of outliers (1.6%, 95% CI −0.3% to 3.6%, $p=0.10$) and when restricting the definition for a smoke day to episodes for which the NAAPS model indicated abundant smoke (1.7%, 95% CI −0.2% to 3.6%, $p=0.07$). In the age-group ≥65, no association with all respiratory hospital admissions was detected on non-smoke days.

In case of hospital admissions due to COPD/asthma, effect estimates for smoke day $PM_{2.5}$ were consistently positive among all ages at lags 0–2, the association being strongest at lag 2 (15.8%, 95% CI −2.1% to 37%, $p=0.09$, Table 4). Excluding outliers increased the effect estimate at lag 0 (19.1%, 95% CI −3.8% to 47.5%, $p=0.11$, Supplementary Table S2). The effect estimates at lags 0–2 remained high when restricting the definition for a smoke day to episodes for which the NAAPS model indicated abundant smoke (Supplementary Table S4). However, when restricting the definition for a smoke day only to the days for which the NAAPS model indicated abundant or some smoke, the positive effect at lags 0 and 1 disappeared. On non-smoke days, no association was observed between outdoor $PM_{2.5}$ and COPD/asthma hospital admissions. For the age-group ≥65 the core model did not reach convergence and, hence, the results are not presented.

For both mortality and hospital admissions, adjustment of the models for daily inhalable coarse particle or ozone concentrations or using a pollen variable with four categories instead of a dummy variable did not significantly change the $PM_{2.5}$ effect estimates on smoke or non-smoke days (Supplementary Tables S1 and S2).

4. Discussion

In the current study, we found suggestive evidence for an association between short-term exposure to long-range transported $PM_{2.5}$ from vegetation fires and increased cardiovascular mortality in the Helsinki metropolitan area for both total population and the elderly. While $PM_{2.5}$ on smoke days had no association with non-accidental mortality or hospital admissions due to cardiovascular causes, there was indication of an association with increased hospital admissions due to all respiratory causes among the elderly and COPD/asthma among all ages.

So far, there has been a limited amount of research on mortality related to vegetation-fire originated air pollution. Although we did not detect an association between $PM_{2.5}$ on smoke days and non-accidental mortality, some previous studies have reported increases in non-accidental mortality following exposure to vegetation-fire smoke (Analitis et al., 2012; Johnston et al., 2011; Sastry, 2002). However, our research does suggest an association with increased risk of cardiovascular deaths, and similar findings have been reported from other parts of the world (Analitis et al., 2012; Faustini et al., 2015; Johnston et al., 2011; Nunes et al., 2013; Sastry, 2002). The adverse effect of vegetation-fire smoke on cardiovascular mortality is also supported by two recent studies conducted in Melbourne, Australia, which found positive associations between vegetation-fire smoke exposure and out-of-hospital cardiac arrests (Dennekamp et al., 2015; Haikerwal et al., 2015).

In the current study, the effect of smoke day $PM_{2.5}$ on cardiovascular mortality was detected at exposure lags of zero (same-day exposure) and three days. In studies on urban PM in general, associations have been typically reported between 0 and 2 days lagged exposure and cardiovascular mortality, but associations have also been found at longer lags (Atkinson et al., 2014). It has been suggested that latent and cumulative effects of short-term PM exposures can extend over periods of weeks rather than days (Neuberger et al., 2013; Zanobetti et al., 2003). At the same time, even sub-daily PM levels have been associated with cardiovascular effects (Burgan et al., 2010). In a study by Peters et al. (2001), the risk of myocardial infarction was elevated both within a few hours and one day following $PM_{2.5}$ exposure.

Separate pathways have been proposed for the immediate and delayed effects of airborne particulate matter on cardiovascular health (Brook, 2008; Brook et al., 2010). Deposition of PM on lungs can directly stimulate lung nerve reflexes and result in autonomic nervous system imbalance, which can lead to elevated blood pressure, arrhythmias, vasoconstriction and, subsequently, cardiovascular events within minutes to hours. Another important and more delayed pathway is the induction of pulmonary oxidative stress and inflammation, leading to the release of pro-oxidative and pro-inflammatory mediators to the circulatory system. Resulting systemic inflammation can impair vascular endothelial function, enhance atherosclerotic plaque vulnerability and vascular thrombosis, and may lead to cardiovascular events within hours to days after exposure.

The confidence intervals of our effect estimates are wide and, hence, the size of the cardiovascular mortality effect should be interpreted with caution. Few studies have attempted to quantify cardiovascular mortality due to vegetation-fire smoke exposure per unit increase in PM concentration. However, Faustini et al. (2015), who conducted a multi-city research in Southern Europe with a study-setting similar to ours, reported an average increase of 3.42% (95% CI 0.64–6.28%) per 10 $\mu g/m^3$ PM_{10} in cardiovascular mortality among total population during smoke-affected days (lag of 0–5 days). The effect estimates of the current study (central estimate for total population 12.4% per 10 $\mu g/m^3$ $PM_{2.5}$ at lag 3) and Faustini et al. are higher than those commonly observed for short-term exposure to particulate matter air pollution in urban environments (0.4% to 1% per 10 $\mu g/m^3$ $PM_{2.5}$, Brook et al., 2010). Hence, greater harmfulness of vegetation-fire originated PM to the cardiovascular system compared to urban PM cannot be ruled out.

Harmfulness of the vegetation-fire originated PM follows from the source – combustion processes. Urban particulate matter is a heterogeneous mixture of particles from various sources which differ with respect to toxicity. However, combustion originating particles contain a large number of organic compounds with pro-oxidative properties (WHO, 2006). Toxicity of organic compounds may though be reduced during long-range transport of vegetation fire smoke (Jalava et al., 2006). Concerning cardiovascular mortality, estimates (per mass unit) for short-term effects have been found to be higher for black carbon, an indicator of combustion particles, than for (total) $PM_{2.5}$ (Janssen et al., 2011).

In contrast to cardiovascular mortality, no relationship was observed between smoke day $PM_{2.5}$ and hospital admissions due to all cardiovascular causes. Our findings are in line with several earlier studies (Hanigan et al., 2008; Henderson et al., 2011; Martin et al., 2013; Morgan et al., 2010), although some studies have found limited evidence for increased cardiovascular hospital admissions (Delfino et al., 2009; Johnston et al., 2007). One possible explanation for the absence of effect could be that a large part of acute cardiac events are very sudden and result in death before the patients reach a hospital, which is supported by the recent findings on the increased risk of out-of-hospital cardiac arrests following exposure to vegetation-fire smoke (Dennekamp et al., 2015; Haikerwal et al., 2015). Nevertheless, evidence for the occurrence of sub-lethal cardiovascular effects is provided by studies reporting statistically significant increases in hospital admissions due to ischemic heart disease (Haikerwal et al., 2015; Johnston et al., 2007), as well as emergency department visits due to various cardiovascular outcomes (Haikerwal et al., 2015; Johnston et al., 2014; Rappold et al., 2011; Resnick et al., 2015).

Due to the limited number of smoke day observations and the low number of daily deaths from respiratory causes, we were unable to study the relationship between smoke day $PM_{2.5}$ and respiratory mortality. However, we did find some indication of a positive association with hospital admissions due to all respiratory causes among the elderly and hospital admissions due to COPD/asthma among all ages. These potential effects are supported by several earlier studies, which have reported increases in hospital admissions for all respiratory causes, asthma, and COPD following exposure to vegetation-fire originated air pollution (Delfino et al., 2009; Hanigan et al., 2008; Henderson et al., 2011; Johnston et al., 2007; Martin et al., 2013; Morgan et al., 2010; Mott et al., 2005). The absence of statistically significant associations in the current study could be explained by a lack of statistical power related to the relatively small study population, the limited number of smoke day observations, and the rather moderate levels of $PM_{2.5}$ on smoke days in absolute terms (median and maximum daily average concentrations 27.7 and 57 $\mu g/m^3$, respectively).

On non-smoke days we found a suggestive positive association between outdoor $PM_{2.5}$ and non-accidental mortality at a lag of one day among the elderly, but no associations were detected with mortality or hospital admissions due to cardiovascular causes. The results on the cardiovascular outcomes are in line with our earlier study on the effects of daily urban PM among the elderly (aged 65 or older) in the same study area in 1998–2004 (Halonen et al., 2009). However, in case of respiratory hospital admissions results of the two studies are inconsistent, as in the previous study statistically significant associations were detected between urban $PM_{2.5}$ and hospital admissions due to all respiratory causes. In contrast, in the current study there was a suggestive small positive effect of same-day non-smoke $PM_{2.5}$ on all respiratory hospital admissions among the total population, but no sign of an effect among the elderly. We have no clear explanation for the dissimilar findings. However, the absence of respiratory health effects in the current study could be partly explained by the somewhat lower daily background $PM_{2.5}$ concentrations and the decrease in the daily number of respiratory hospital admissions in the study area in 2000–2010 compared to 1998–2004, likely due to improvements in the treatment of respiratory diseases. The dissimilar findings could also be partly explained by the use of different air quality monitoring stations to assess daily $PM_{2.5}$ exposure, as well as differences in the applied statistical methods.

Strengths of our research include the long study period and the exposure assessment based on $PM_{2.5}$ rather than PM_{10}, which has been used as an exposure indicator in most epidemiological

studies on health effects of vegetation-fire smoke. Strong smoke episodes in Finland are rare and their occurrence varies considerably from year to year (Niemi et al., 2009). Therefore, a long time period is required to ensure a sufficient number of observations for a meaningful statistical analysis. In regard to smoke exposure, $PM_{2.5}$ is a preferable indicator compared to PM_{10} because the majority of combustion-originated PM is in the fine particulate size-range, especially regarding the long-range transported emissions. In addition, because we studied the effects related to long-range transported smoke, the possible confounding effect of psychological factors on cardiovascular effects can be ruled out. Psychological stress has been suggested to play an important role in cardiovascular mortality related to severe local fire events (Analitis et al., 2012).

A limitation of the study is the lack of knowledge on the exact contribution of vegetation fires to $PM_{2.5}$ on the smoke-affected days. The use of air quality data from both urban background and regional background measurement stations confirms that the episodes were mainly caused by long-range transport and not local emission sources. During smoke days the daily $PM_{2.5}$ concentrations were also on average three times higher compared to the normal background level and, hence, the contribution of the long-range transport can be assumed to have been significant. Moreover, the validity of the NAAPS model data in identifying vegetation-fire originated episodes in southern Finland has been demonstrated based on chemical composition analyses of particles on days influenced by long-range transported smoke, and the chemical analyses have indicated substantial contribution from biomass burning during these episodes (Niemi et al., 2009). However, because strong smoke episodes in southern Finland are typically caused by air masses arriving from Eastern Europe, which contain regions with high anthropogenic emissions, air quality on smoke days is also likely to be somewhat affected by long-range transported air pollution from anthropogenic sources (Niemi et al., 2009). The mixing of smoke with anthropogenic pollutants might have been significant especially in the weaker phases of the smoke episodes.

5. Conclusions

Vegetation fires cause severe and recurrent air pollution problems in many parts of the world. Because the pollutants can be transported in the atmosphere over long distances, they can expose populations over wide areas. Moreover, in many regions exposure to vegetation-fire smoke can be expected to grow in the future as the risk of wildfires increases due to climate change. Our study suggests that the long-range transported fine particulate matter from vegetation fires may have adverse effects on public health over a distance of hundreds to thousands of kilometres from the fires. It also provides further evidence for the positive association between biomass-burning originated $PM_{2.5}$ and cardiovascular mortality. The severe and widespread adverse effects of vegetation-fire smoke on public health should be considered when evaluating the socio-economic impacts of wildfires and prescribed open biomass burning. Measures should also be taken to reduce the health risks related to the air pollution episodes. This can be achieved by providing timely warnings and advising people to stay indoors, keeping windows closed, and avoiding physical activities in polluted outdoor environments. Exposure to smoke can also be reduced by using air cleaners to improve indoor air quality, which is recommended particularly in care facilities for vulnerable people, such as the elderly, the children, and those with a pre-existing respiratory or heart disease.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2016.08.003.

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