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Mortality and Morbidity Attributed to Aerosol and Gaseous Emissions from Biomass Use for Space Heating

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ABSTRACT

Over the last years in Greece, the intense use of biomass for space heating resulted in increased ambient and indoor air pollution and enhanced population exposure to particulate matter and genotoxicants in particle and gaseous form such as polyaromatic hydrocarbons (PAHs). This study deals with the precise assessment of exposure to these pollutants and the related health and monetary impact. To these goals, measured and modelled data of outdoor and indoor PM_{10} and $PM_{2.5}$ were fed into an integrated exposure assessment modelling framework that takes into account indoor air quality, time-activity patterns of the exposed population and activity-based inhalation rates. Chemical analysis on the sampled PM allowed us to estimate the contribution of biomass burning to PM mass concentration and the associated increase in toxicity (expressed in terms of PAHs content). Health impacts were assessed adapting well-established exposure-response functions coupled with mechanistic exposure models. Monetary cost of these impacts was calculated based on the valuation of the willingness-to-pay/accept to avoid/compensate for the loss of welfare associated with them. PM from biomass burning is finer and more genotoxic than PM from traffic or other urban sources. Total exposure to PM and PAHs due to biomass use was significantly increased and the estimated health burden was increased by more than 40%, while the associated monetary cost rises to ca. €200 m.

Keywords: PM; Mortality; Morbidity; Biomass combustion, PAHs; Lung cancer.

INTRODUCTION

Particulate matter (Reid *et al.*, 2005) remains one of the most harmful airborne pollutants of increased scientific and regulatory interest. PM is considered ubiquitous, since it is emitted from both biogenic and anthropogenic sources (mainly multiple combustion sources), or generated by atmospheric reactions formatting secondary aerosol. Source complexity, transport phenomena and the atmospheric chemistry result in particulate matter of different size and chemical composition (Galindo *et al.*, 2011). Particles are found in indoor environments as well (Karakitsios *et al.*, 2014); indoor concentrations, are affected by outdoor levels, as well as the presence of indoor emitting sources such as smoking, cooking, wood-burning appliances like wood stoves and fireplaces, especially if the smoke leaks or backdrafts into the home. As a result, personal exposure to PM is estimated based on all types of environments and microenvironments (including different transportation means) encountered by the exposed individuals on a daily basis.

Specific human activities result in significantly increased PM levels in the ambient and in indoor air. A typical example is the irrational use of biomass burning for space heating in Greece. Although the use of biomass for space heating was introduced in 2011 as a CO_2-neutral means to foster global warming mitigation, a series of austerity measures combined with the excessive rise in the taxation of light heating diesel, resulted in irrational use of biomass for residential heating in the winter of 2012–2013. This was followed by a significant increase of ambient and indoor air levels and, consequently of population exposure to PM_{10} and $PM_{2.5}$ (Sarigiannis *et al.*, 2014). A major reason for the significant increase of PM emissions was the lack of technological readiness of Greek households to use biomass for space heating; the vast majority of the appliances included use of open fireplaces or woodstoves. Similar phenomena have occurred in other southern European countries as well. In Portugal, it was estimated that, in 2010, on average, 37% of the population used biomass burning for space heating (Gonçalves *et al.*, 2012). In Italy, in Po Valley, it was found that the fraction of OC apportioned to biomass burning by factor analysis varied between 20 and 29% (Paglione *et al.*, 2014). However,

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in other southern European areas such as Barcelona, the biomass burning contribution has been found to be lower e.g., 2.5% of $PM_{2.5}$ mass in Barcelona was attributed to biomass burning on an annual basis (Reche *et al.*, 2012), reflecting differences in annual space heating needs and socioeconomic status.

As a result, residential wood combustion is now recognized as a major particle source in many developed countries, and the number of studies investigating the negative health effects associated with wood smoke exposure is increasing (Naeher *et al.*, 2007). Combustion appliances in use today provide highly variable combustion conditions resulting in large variations in the physicochemical characteristics of the emitted particles. These variations are likely to influence the biological effects induced by the wood smoke particles. The use of wood or charcoal for heating or cooking during female adolescence was recently associated with chronic obstructive pulmonary disease later in life, providing further support for an association between wood smoke exposure and adverse respiratory effects. However, in order to better understand the effects of biomass burning on public health we need to take into account i) the actual human exposure as the integral of outdoor and indoor interactions and ii) differences in toxicity arising from the different uses of space heating means (Sarigiannis *et al.*, 2015).

Based on the above, in this study we aim to quantify the effects of biomass burning on i) outdoor and indoor air quality, ii) actual personal exposure to noxious combustion products and iii) the potential health and monetary effects in a large Greek urban environment (Thessaloniki). In order to address the observed data paucity related to population exposure, a comprehensive methodology seamlessly integrating measured and modeled data was developed to produce high quality estimates of personal exposure and of the respective health and monetary impact estimates.

METHODS

Study Design

To better identify the contribution of biomass burning to elevated PM and PAHs exposure and the respective impact on public health, a seven-month long monitoring and modeling study was designed including:

- Measurements of ambient air particles of aerodynamic diameter of 2.5 and 10 μm in two sampling locations, representative for the urban background levels and a traffic site as well.
- Measurements of indoor PM in 30 houses, including periods when an open fireplace was used or not for space heating. Details on the residential location characteristics are presented in Table 1.
- Chemical analysis of levoglucosan (a well-established tracer of biomass burning) and PAHs in the particle phase.
- Development of an indoor air pollution dispersion model that takes into account the interaction of outdoor penetration and the contribution of indoor sources.
- Assessment of personal exposure and actual population intake of PMx and PAHs in both the particle and gaseous phases, accounting for individual activity patterns and the related effect of activity intensity in inhalation rate and pollutants intake.
- Deposition of particles across the human respiratory tract, aiming at estimating the actual amount of particles and toxic burden people are exposed to.
- Application of well-established exposure-response functions for mortality and morbidity (cardiovascular and respiratory hospital admissions) based on PMx exposure and development of a mechanistic methodology for estimating exposure to PAHs and the attributable lung cancer risk.
- Translation of potential health impact into monetary costs.

Our 7 month study included a wide variety of meteorological conditions. To better understand the differences between the cold period of the year (during which space heating was used) and the warm period of the year, the measurement dataset was divided as follows:

a) From October 5 to November 10 (2012) and from March 10 to April 15 (2013) the period was considered as warm. PM levels measured in this period were considered as representative for the rest of the warm period, based on the evaluation of older PM time series in Thessaloniki.
b) From November 10 (2012) to March 9 (2013) the period was considered as cold; the low ambient air temperatures induced the use of space heating in dwellings.

PM Sampling

$PM_{2.5}$ and PM_{10} samples were collected using low-flow air samplers (ENCO PM, TCR TECORA, Italy). The samplers used sampling heads meeting the EN 12341 (PM_{10}) and EN 14907 ($PM_{2.5}$) standards, and operated at a flow-rate of 38 L min^{-1}, with a collection time of 24 h per sample. Samples were collected on PTFE membranes filters with PMP supporting ring (PALL Life Sciences, Ø 47 mm, pore size 2 μm, USA). Teflon filters were analyzed gravimetrically for particle mass concentrations using an electronic microbalance with a sensitivity of ± 1 μg after 24-h equilibration at a temperature between 20°C and 23°C and a relative humidity (RH) between 30 and 40%. Each filter was weighed at least three times before and after sampling, and the net mass was obtained by subtracting the average of the pre-sampling weights from the average of the post-sampling weights. Differences among replicate weightings were < 5 μg for the blanks and the samples. Prior to the start of the sampling campaign, the flow rate of the $PM_{2.5}$ and PM_{10} samplers was calibrated. Field blank filters were also collected and used to correct for background concentrations or influences from handling and transport.

Chemical Analysis

Levoglucosan content was determined according to the following procedure. A quarter of each filter was spiked with a known amount of a surrogate standard and extracted with a solution of dichloromethane:methanol 2:1 (Merck 99.9%) in an ultrasonic bath. The extract was filtered and evaporated until dryness. Then dichloromethane, pyridine (Sigma-Aldrich) and bis(trimethylsilyl)trifluoroacetamide (BSTFA) containing 1% trimethylchlorosilane (Supelco)

Table 1. Residential location characteristics.

	Building type	Ventilation (ACH)[a]	Air conditioning	Indoor PM and PAHs sources	Fireplace usage
1	Apartment 76 m^2 (2nd floor)	0.3–2.5	Yes	Cooking	No
2	Maisonette 104 m^2 (3 floors)	0.5–3.5	Yes	Cooking, candles, fireplace	Yes
3	Apartment 98 m^2 (3rd floor)	0.5–1.5	Yes	Cooking, fireplace	Yes
4	Maisonette 149 m2 (3 floors)	0.5–3.5	Yes	Cooking, fireplace	Yes
5	Apartment 54 m^2 (2nd floor)	0.5–2.5	Yes	Cooking	No
6	Apartment 72 m^2 (5th floor)	0.3–3.5	No	Cooking	No
7	Apartment 77 m^2 (1st floor)	0.5–2.5	No	Cooking	No
8	Apartment 43 m^2 (4th floor)	0.5–1.5	Yes	Cooking	No
9	Maisonette 108 m^2 (2 floors)	0.5–4.5	No	Cooking, fireplace	Yes
10	Apartment 88 m^2 (5th floor)	0.5–2.5	Yes	Cooking, fireplace	Yes
11	Maisonette 92 m^2 (2 floors)	0.3–2.0	Yes	Cooking, fireplace	Yes
12	Apartment 52 m^2 (2nd floor)	0.5–2.5	Yes	Cooking, candles	No
13	Maisonette 138 m^2 (3 floors)	0.5–1.5	Yes	Cooking, candles, fireplace	Yes
14	Apartment 114 m^2 (6th floor)	0.5–3.5	Yes	Cooking	No
15	Maisonette 112 m^2 (3 floors)	0.5–2.5	Yes	Cooking, fireplace	Yes
16	Apartment 42 m^2 (1st floor)	0.3–1.5	No	-	No
17	Maisonette 112 m^2 (3 floors)	0.5–3.5	Yes	Cooking, fireplace	Yes
18	Apartment 59 m^2 (3rd floor)	0.5–1.5	Yes	Cooking	No
19	Maisonette 164 m^2 (3 floors)	0.5–4.5	No	Cooking, fireplace	Yes
20	Apartment 103 m^2 (2nd floor)	0.5–3.5	Yes	Cooking, fireplace	Yes
21	Apartment 74 m^2 (3rd floor)	0.3–2.5	No	Cooking	No
22	Apartment 65 m^2 (1st floor)	0.5–2.5	Yes	Cooking	No
23	Maisonette 122 m^2 (3 floors)	0.5–1.5	No	Cooking, fireplace	Yes
24	Apartment 83 m^2 (6th floor)	0.5–3.5	Yes	Cooking	No
25	Apartment 92 m^2 (3rd floor)	0.5–3.0	Yes	Cooking	No
26	Apartment 50 m^2 (2nd floor)	0.3–2.5	Yes	Cooking	No
27	Apartment 88 m^2 (2nd floor)	0.5–3.5	Yes	Cooking	No
28	Maisonette 101 m^2 (2 floors)	0.5–4.5	Yes	Cooking, candles, fireplace	Yes
29	Apartment 119 m^2 (4th floor)	0.3–3.5	Yes	Cooking, fireplace	Yes
30	Apartment 71 m^2 (5th floor)	0.5–2.5	Yes	Cooking	No

[a] Air changes per hour (ACH) was defined after the inspection of the residential location, taking into account the number of openings (windows and doors), ventilation habits, renovation status and energy class.

were added and the reaction was conducted at a heater. A known amount of an internal standard was added prior to the analysis. Analysis was performed by an 7890A Agilent gas chromatographer coupled with a 5975C Agilent inert MSD mass spectrometer operated in the SCAN mode. One μL of each sample was injected into the GC in splitless mode where the inlet temperature was kept at 270°C. A fused silica capillary column (30 m × 250 μm × 0.25 μm i.d., HP-5MS Agilent) was used with helium as carrier gas. The GC oven started with an initial temperature of 100°C, held for 2 min and then ramped to 300°C with a temperature increase of 20 °C min^{-1}.

The concentrations of the PAHs in the particulate matter sampled were determined according to the following method. Half of each filter was spiked with a known amount of surrogate standards and extracted with dichloromethane (Merck, 99.8%) in an ultrasonic bath. Hexane (Merck 99.9%) was added and the extract was filtered and concentrated. Then hexane and a known amount of deuterated internal standards were added before a final stage of concentration to 0.5 mL. Analysis was performed by a 7890A Agilent gas chromatographer coupled with a 5975C Agilent inert MSD mass spectrometer operated in the SIM mode. Two μL of each sample was injected into the GC in splitless mode where the inlet temperature was kept at 280°C. A fused silica capillary column (30 m × 250 μm × 0.25 μm i.d., HP-5MS Agilent) was used for the separation of the fifteen PAHs with helium as carrier gas. The GC oven temperature was 60°C for 1 min, increased with a rate of 10 °C min^{-1} to 120°C, then increased with a rate of 5 °C min^{-1} to 240°C and then increased to 300°C (rate of 6 °C min^{-1}) and held for 20 min. Identification of PAHs was accomplished by using the 16 EPA priority PAHs standards (Sigma-Aldrich, including naphthalene, 2-methylnaphthylene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-cd]pyrene, dibenzo[a,h]anthracene and benzo[g,h,i]perylene), plus benzo[e]pyrene (Supelco), benzo[a]pyrene (Supelco) and perylene (Supelco). Overall the analysis method has been developed for analysis of 19 PAHs. Among them, 16 PAHs have been found above the limit of detection (LOD); naphthalene, 2-methylnaphthylene, acenaphthylene were below LOD, since they are found only in the gaseous phase.

Modelling Indoor Air Quality and Exposure

Indoor Concentrations

Assessment of indoor concentrations was based on the computational tool INTERA (Sarigiannis *et al.*, 2012a). This platform consists of a multi-zonal mass-balance model that accounts for the main processes related to particle fate, such as emissions, deposition, indoor/outdoor air exchange and outdoor-to-indoor infiltration. Default values for the parameters of background rate of emissions of European dwellings (Hänninen *et al.*, 2004) and of penetration rates and deposition were used (Özkaynak *et al.*, 1996), further enhanced by additional measurements for the validation of the model (Sarigiannis *et al.*, 2014).

Personal Exposure

The calculation of PM exposure depends on the indoor/outdoor concentration data for two clusters of exposed individuals. Moreover, exposure to PM was affected by the use of an open fireplace or not indoors. Personal exposure to and intake of PM and PAH were estimated taking into account the average concentration to which a person is exposed to these pollutants over a given period of time (Sarigiannis *et al.*, 2014), including indoor and outdoor activities. Personalized parameters affecting actual intake such as inhalation rates, which in turn are based on the respective activities were also taken into account (Sarigiannis *et al.*, 2012b; Sarigiannis *et al.*, 2014).

Health Impact Assessment

To estimate the associated health effects of PM exposure, we used the widely established epidemiological concentrations-response functions for outdoor PM, as determined by the HRAPIE team under WHO coordination (2013). For the PM attributed mortality and morbidity (respiratory and cardiovascular hospital admissions) health endpoints used in this study; differences in toxicity depending on PM composition were not taken into account (WHO, 2007). To better capture the marginal change in mortality and morbidity associated to biomass burning originated PM we used fine particles ($PM_{2.5}$) as the best exposure metric (Sarigiannis *et al.*, 2014). The concentration-response functions (CRFs) for the health endpoints of interest are given in Table 2. The PM_{10} CRF estimates were converted to estimates of $PM_{2.5}$ using an established methodology developed in the frame of the HEIMTSA project (IOM, 2011), based on the initial concept described by WHO (2004), since original $PM_{2.5}$ functions are not available for these endpoints. Relative risk was calculated for the average concentration for the period of interest. Then the attributable fraction was derived, and finally health impact was estimated by multiplying this with the background rate of disease and the respective population of interest.

Extending the currently existing paradigm of assessing PM attributed health effects we attempted to capture differences in toxicity (in terms of PAH content) as well as size distribution of biomass emitted particles. A comprehensive methodology has been followed for assessing the lung cancer risk attributed to PAHs adsorbed on PM (Sarigiannis *et al.*, 2015), that takes into account:

Table 2. Concentration response Functions (CRFs) and monetary valuation for the several endpoints addressed in the study.

Health endpoint	CRF	Reference	Background rate	Reference
Mortality (all causes)	6.2% (95% CI: 4%, 8.3%) change per 10 μg m^{-3} $PM_{2.5}$	(WHO, 2013)	Life table data (WHO, 2014)	(WHO, 2013)
Cardiac hospital admissions	0.6% (95% CI: 0.3%, 0.9%) change per 10 μg m^{-3} PM_{10}	(Hurley *et al.*, 2005; IOM, 2011)	723 emergency cardiac admissions per 100,000 population, all ages, per year (Hurley *et al.*, 2005):	(Hurley *et al.*, 2005; IOM, 2011)
Respiratory hospital admissions	0.9% (95% CI: 0.7%, 1.0%) change per 10 μg m^{-3} PM_{10}	(Hurley *et al.*, 2005; IOM, 2011)	617 emergency respiratory hospital admissions per 100,000 population, all ages, per year (Hurley *et al.*, 2005):	(Hurley *et al.*, 2005; IOM, 2011)
Mortality/morbidity indices	Low	Medium	High	Reference
Mortality (all cause) based on VSL	1,120,000	1,650,000	5,600,000	(Alberini *et al.*, 2006)
Cardiovascular hospital admissions	2,990	2,990	8,074	(Navrud, 2001; Holland *et al.*, 2004)
Respiratory hospital admissions	2,990	2,990	8.074	(Navrud, 2001; Holland *et al.*, 2004)
Lung cancer	480	451,000	2,800,00	(Alberini *et al.*, 2006)

- The overall toxicity of the mixtures of PAHs expressed as per the respective Toxic Equivalency Concentration (TEQ), which is determined using relative Toxic Equivalent Factors (TEFs), taking the *TEF* for B[a]P as basis and thus equal to 1 (Nisbet and LaGoy, 1992).
- Translation of the inhalation unit risk of B[a]P ($IUR_{B[a]P}$) value of 1.1×10^{-3} m^3 μg^{-1} (CEPA, 2004) into a slope factor (SF), assuming that $IUR_{B[a]P}$ refers to a human of 70 kg inhaling 20 m^3 of ambient air per day; this results in a *SF* equal to 3.85×10^{-6} (kg day) ng^{-1} B[a]P.
- Estimation of the size fractioned PM mass deposited across different HRT regions employing the MPPD HRT deposition model (de Winter-Sorkina and Cassee, 2002).
- Calculation of the actual TEQ dose retained by HRT by combining the TEQ levels of the different sized fractioned PM and the way they deposit across HRT.

Aiming at estimating the overall cancer risk from PAHs in the ambient air, the mass of PAHs in the gaseous phase was also estimated. This calculation was based on the partition coefficient K_p between gaseous and particles phase based on the Pankow model (Pankow, 1994):

$$K_p = \frac{N_s \cdot \alpha_{TSP} \cdot T \cdot e^{\frac{(Q_1 - Q_v)}{R \cdot T}}}{1600 \cdot p_L^0} \quad (1)$$

where N_s (cm^{-2}) is the available surface for adsorption, a_{tsp} (m^2 g^{-1}) is the special surface of aerosols, Q_1 (kJ mol^{-1}) is the enthalpy of adsorption from the surface, R is the ideal gas constant, T *is the temperature* (°K), and p_L^0 is the vapor pressure at 25°C. Concentration in the gaseous phase is then estimated by the following relationship:

$$K_p = \frac{F / TSP}{A} \quad (2)$$

where F is the concentration of PAHs in the particles phase, *TSP* is the total suspended particles (in practice all the amount of PAHs is adsorbed in PM_{10}; thus for this calculation the TSP of interest is considered equal to PM_{10}) and A is the concentrations of PAHs in gaseous phase. This calculation was done for each of the PAHs analyzed using EPISuite v4.11 (EPA, 2012). TEQ for PAHs in the gaseous phase was estimated as above and the corresponding slope factor was used for estimating lung cancer. Exposure was estimated following the same methodological scheme, but all of the PAHs in the gaseous phase were assumed to be taken in through inhalation.

Monetary Valuation

Evaluation of monetary impacts on human health accounts for changes in welfare, including i) resource costs, ii) opportunity costs and iii) dis-utility costs, which in turn are reflected in a valuation of the willingness-to-pay/accept (WTP/WTA), to avoid/compensate for the loss of associated welfare. Taking into account the several uncertainties that may arise in estimating the changes in welfare, the monetary valuation of the health effects estimated in the current study are given in Table 1.

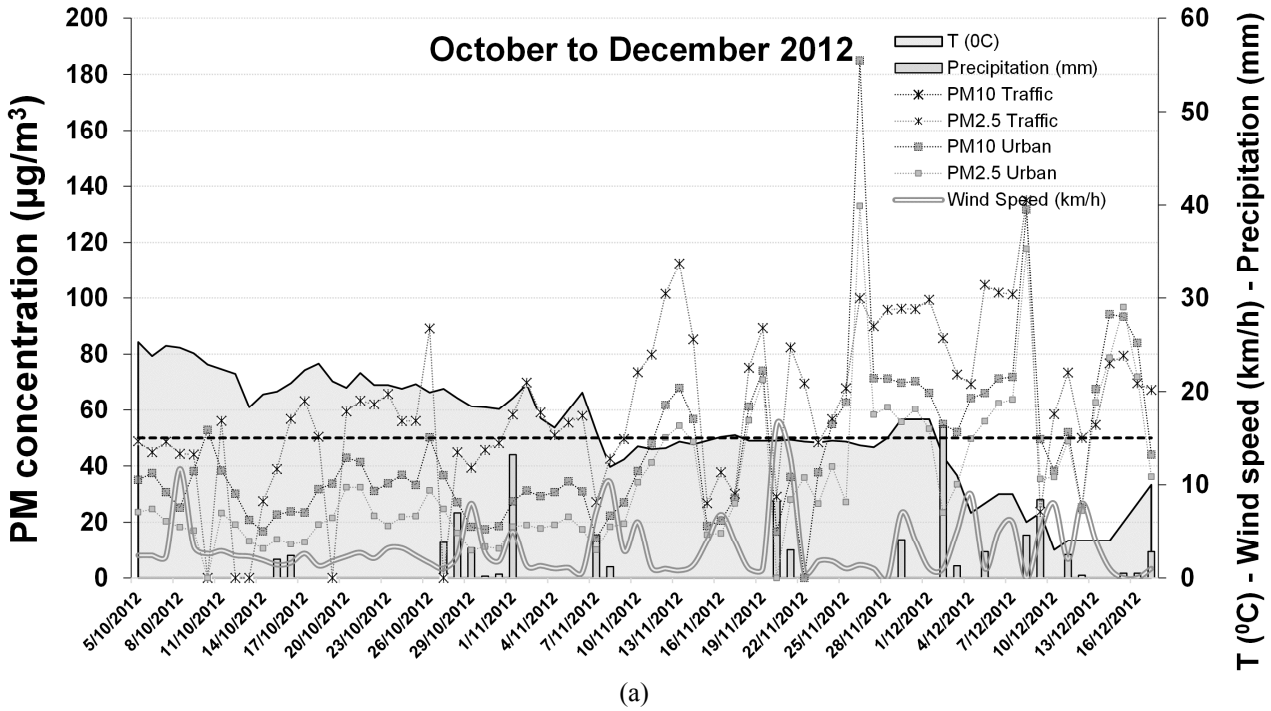
Probabilistic Framework Applied in the Study

Indoor air and exposure distributions were computed probabilistically following a Bayesian methodology employing Monte Carlo simulation of 50,000 iterations. The INTERA computational platform (Sarigiannis *et al.*, 2012a) was used for this, based on the distributions of measured ambient air concentrations, and air exchange rate, penetration coefficient, PM deposition and emission rates found in the literature (Sarigiannis *et al.*, 2014). Exposure distributions were used as input for health and monetary impact assessment. Similarly, literature-derived distributions of the CRFs and the monetary valuation functions used for each health endpoint considered in the study served as priors. For the CRFs, a normal distribution was fitted to the mean value and the respective 5% and 95% confidence intervals (these are given in Table 1). For monetary valuation functions, a log-normal distribution was fitted to the mean, min and max values (given in Table 1). Monte Carlo sampling in this case involved a large number of samples (500,000 iterations) executed on Crystal Ball (Oracle, 2011) based on the distributions of the input parameters. The Bayesian probabilistic framework followed in the study helped to increase the statistical robustness of the results, supporting thus the generalization of our findings to urban settings beyond the ones where the measurement campaign was executed.

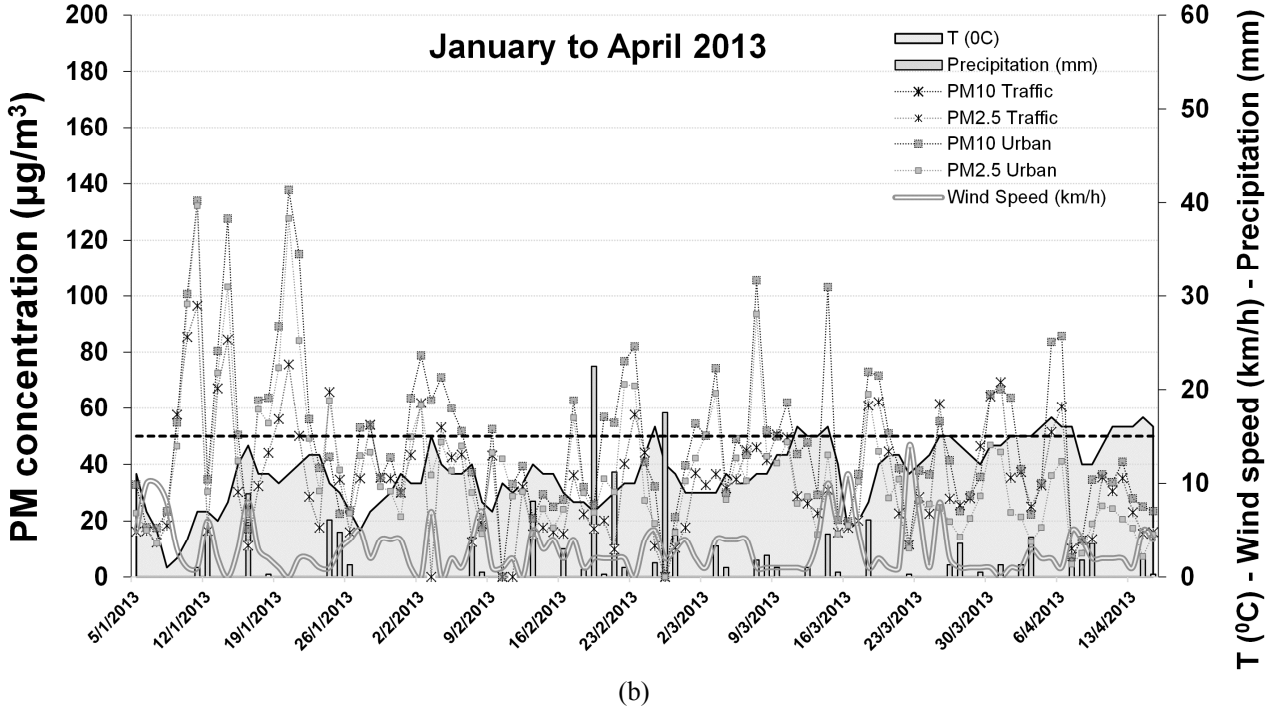
RESULTS

Ambient Air PM Levels and Biomass Contribution

Inter-day variability of the measurements campaign (for both sampling sites) that started in October 5 2012, is illustrated in Figs. 1(a)–1(b). Both $PM_{2.5}$ and PM_{10} concentrations tend to rise significantly during the transition from the warm to the cold period. The latter is marked by the lower temperatures starting in November 10, 2012 and finishing in March 10, 2013. During the warm period the concentrations at the traffic station (53.1 and 29.5 μg m^{-3} for PM_{10} and $PM_{2.5}$ respectively) were constantly higher than the concentrations at the background station (30.6 and 19.4 μg m^{-3}), because of the high traffic load, which is the dominant emission source in the urban area under study. This is the combined result from direct tailpipe emissions and re-suspension processes (movement of vehicles, loading and unloading operations). However, during the wintertime, PM concentrations increase rapidly at the urban background station contrary to the ones at the traffic station, due to the contribution of biomass burning as shown by elevated levoglucosan concentrations. Most days the thresholds (for both PM_{10} and $PM_{2.5}$) proposed by the 2008/50/EC guideline were exceeded. This phenomenon appeared in the Metropolitan area of Thessaloniki and spread rapidly as the mean ambient temperature fell below 10°C. The amount of PM_{10} mass that was produced by biomass burning was determined using the empirical function proposed by Caseiro *et al.* (2009). Higher levels of levoglucosan (almost twice) were found in PMx at the urban background sampling



(a)



(b)

Fig. 1. Ambient air PM_{10} and $PM_{2.5}$ concentrations and meteo data at the traffic and the urban background measurement sites for the periods a) October to December and b) January to April.

site compared to the traffic sampling site. The contribution of biomass combustion to PM mass concentration varied significantly with an average value of 15% and maximum values up to 40% at the traffic station and 30% and maximum values up to 70% at the urban background station. The highest contribution of biomass attributed PM was also related to extreme pollution incidents (mean daily concentration of PM_{10} close to 150 µg m^{-3}).

PAH Levels

Chemical analysis of PAHs and the respective translation into TEQ revealed that the toxic burden (in terms of PAH content) is significantly differentiated between the warm and the cold season. More in detail, the significant increase of PM levels in the urban and traffic site (by almost two times for PM_{10} and 3 times for $PM_{2.5}$), results to an even higher increase of TEQ content (by 4 and 5.5 times

respectively). More in detail, the average concentrations of TEQ in the urban background site is equal to 26.9 and 29.1 ng m^{-3} for $PM_{2.5}$ and PM_{10} respectively, compared to 6.5 and 6.8 ng m^{-3} during the warm period (Fig. 2(a)). Estimated TEQ in the gaseous phase, accounted for almost 50% of TEQ in particles in the urban background site and about 60% in the traffic background site. This is explained by the fact that PAHs of higher TEF, are characterized by stronger adsorption in the particles phase, e.g., B[a]P concentration in the gaseous phase accounts for 10 to 15% to the one found in particles. The detailed concentration levels for the 16 PAHs found on PM are illustrated in Figs. 2(b) and 2(c) for the urban background and the traffic site respectively.

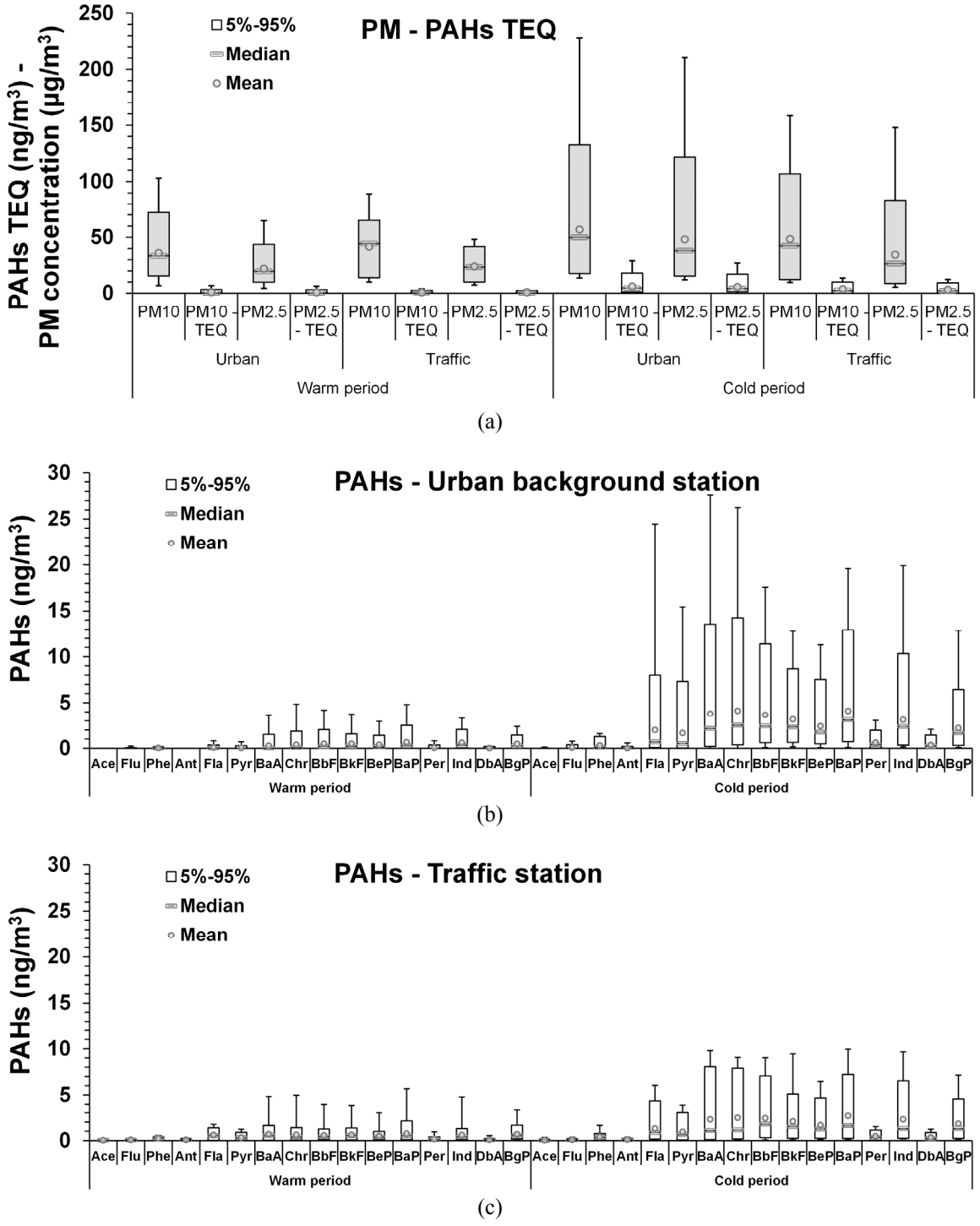


Fig. 2. (a) PMx and TEQ descriptive metrics for the two sampling sites between the cold and the warm period of measurements (b) PAHs concentration during the warm and cold period in the urban background location (c) PAHs concentration during the warm and cold period in the urban background location.

Indoor Air Concentrations - Measurements and Modeling Results

Indoor PM concentrations are nearly equal to the outdoor ones if no strong emission source (e.g., smoking indoors or combustion) is present given the aeration habits of the local population. However, in case of existence of fireplace PM concentrations tend to rise significantly. During fireplace operation PMx concentrations significantly increased up to 100 μg m^{-3} and the particles were found to disperse rapidly and uniformly within the house (Sarigiannis *et al.*, 2014). When the fireplaces burned for 3–5 hours daily, average daily concentrations tended to be similar or higher than the ones outside compared to houses without fireplace, with an average daily increase of about 10 μg m^{-3} and 14 μg m^{-3} for $PM_{2.5}$ and PM_{10} respectively. Daily duration of fireplace usage, type of wood used and burning as well as housing conditions such as residential volume and insulation explain the variance in indoor PM levels. Variance in these parameters is better reflected in the modelled concentrations, which are otherwise in very close agreement with the measured and the modeled concentrations. Personal exposure variability highly depends on indoor pollution levels, especially when there is an additional indoor source of PM and gaseous PAHs such as an open fireplace. It should be borne in mind that humans spend ca. 80–90% of their time indoors. For individuals exposed to indoor biomass burning, average daily exposure rises up to 38.5 and 35.8 μg m^{-3} for PM_{10} and $PM_{2.5}$ respectively. The increase of exposure to PM of smaller aerodynamic diameter is more significant, with exposure levels taking values of 27.4 and 17.5 μg m^{-3} for PM_{10} and $PM_{2.5}$ respectively.

Health Impact Assessment and Associated Monetary Valuation

The estimated mortality and morbidity are shown in Fig. 3(a). Almost 170 additional deaths are attributed to PM during the cold period compared to the cold period, although this refers to a period half of duration (4 months compared to 8 months). Similarly, an additional number of 100 respiratory and cardiovascular hospital admissions is expected during the cold period. Among the health endpoints of interest, the highest increase is related to the lung cancer estimated risk; although the overall incidence rate remains low (lifetime individual risk around 10^{-6}), this was actually increased up to almost 6 times compared to the warm period.

The associated monetary costs (Fig. 3(b)) of the observed health effects are dominated by mortality costs, resulting in an increase of ca. €200 m (potentially up to a billion) in the cold period of the year compared to the warm period. Increase in morbidity costs due to cardiovascular and respiratory hospital admissions are in the range of €250,000, while lung cancer risks costs (associated with exposure to PAHs in both the particle and the gaseous phases) are lower, mainly due to the low incidence rate.

DISCUSSION

The current study describes a comprehensive methodological framework for assessing exposure to PM and PAHs in the gaseous and particle phases due to biomass burning for space heating in cities and estimating the related public health impact. From the study it was found that widespread use of biomass burning results in elevated ambient and indoor air concentrations and consequently exposure to PM and PAHs. Using an integrative exposure model allowed us to avoid oversimplifications such as directly associating ambient air PM to health effects or accounting only for the gaseous form of PAHs; taking into account indoor/outdoor interactions and the parameters affecting exposure, we were able to properly account for the respective health effects. Health effects would be overestimated if epidemiologically established concentration response functions were simply applied to ambient air PM levels, while risks of exposed individuals using open fireplaces as a space heating source might be underestimated. This contribution is not only associated to open fireplaces but also to all incomplete combustion apparatus that use wood and wood products. The latter comprise a very important source of both in- and outdoor PM. Although several sources contribute to indoor air particles, during the operation of open fireplace, PM levels have been found to rise up to 100 μg m^{-3}. These results are comparable albeit lower to the ones presented by Guo *et al.* (2008) where peak levels of 167 (for PM_{10}) and 155 (for $PM_{2.5}$) μg m^{-3}, PM_{10} and $PM_{2.5}$ increase up to four and nine times respectively than during normal occupancy conditions. What has to be noted is the very significant increase in the number of fine and ultrafine particles, which are deemed to have the most hazardous effects to human health due to their ability to reach the lower part of the respiratory tract (bronchioles) and translocate in the body via systemic circulation (Brook, 2008); this is even more evident if an HRT deposition model is employed. Moreover, peaks of exposure (and potential intake) are related to specific activities performed outdoors (e.g., commuting by any transportation means) or indoors (use of open fireplace). Thus, cost-effective policy interventions aiming at public health protection from ambient air particulate episodes have to be targeted on curbing human exposure rather than simply ambient air concentrations. Time differences between peaks of ambient air PM and the actual PM intake during the day have to be accounted for when assessing the actual public health impact of PM exposure. Thus, a comprehensive risk assessment/health impact evaluation should take into account the complex outdoor/indoor air interactions, as well as exposure and intake dynamics taking stock of time–activity and intensity of activity patterns of the population. The latter are important modifiers when it comes to assessing population exposure to airborne pollutants.

The results of mortality and morbidity are similar to the ones from conceptually similar studies such as the one by Halouza *et al.* (2012) in Austria; more in detail, the use of a more refined framework for assessing the actual human exposure resulted in more realistic assessment of the overall health effects. On the contrary, the lack of a mechanistic description that takes into account actual HRT deposition results in underestimated cardiovascular effects as identified in the intervention study in Australia by Johnston *et al.*

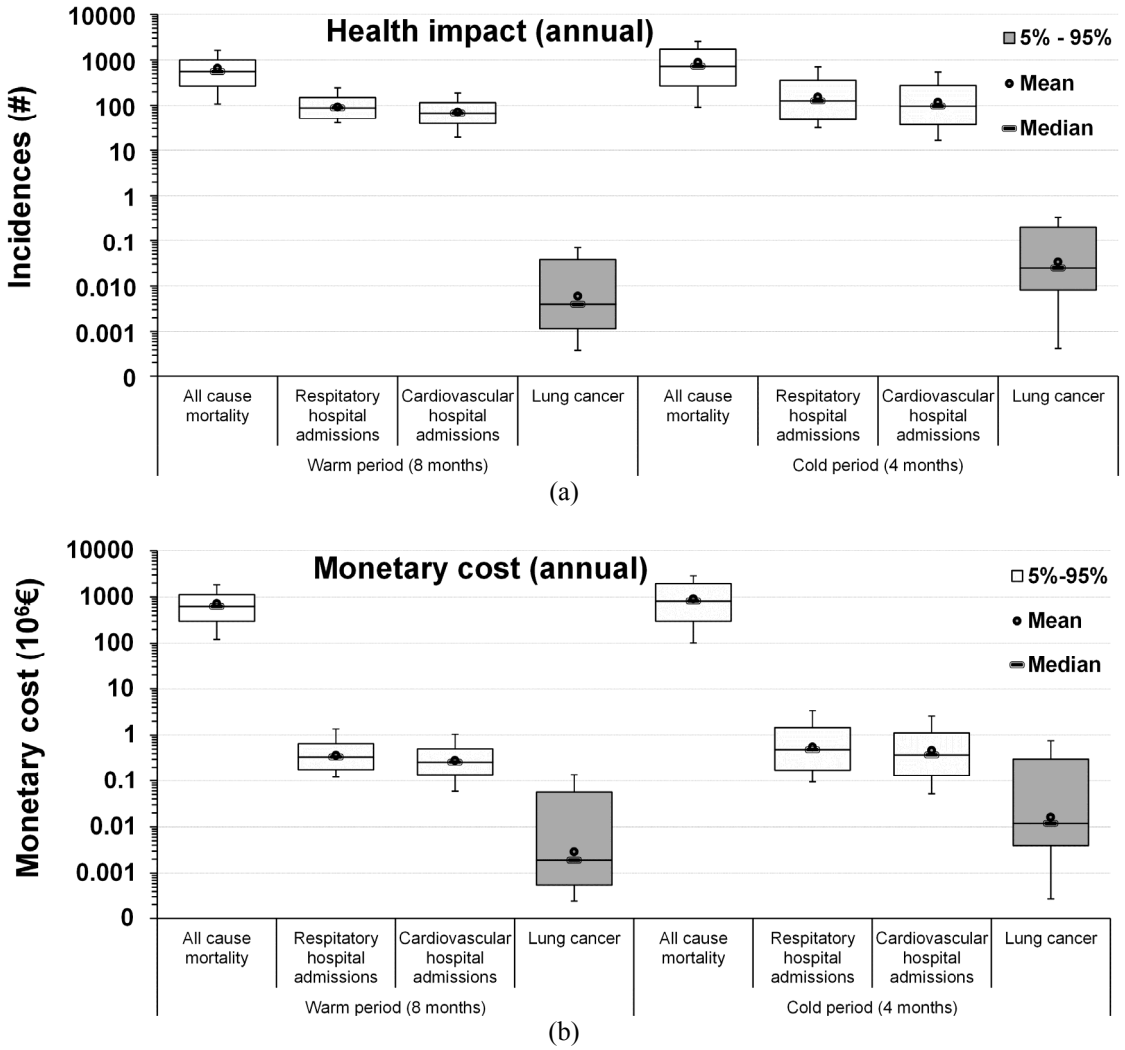


Fig. 3. (a) Health impact assessment of the associated health endpoints (b) Monetary valuation of the associated health endpoints.

(2013); considering that particle translocation is one of the major mechanisms related to air pollution attributed cardiovascular disease (Brook, 2008), the capability to differentiate the fraction of particles that potentially translocate into the systemic circulation would further refine our methodology. However, current health impact assessment methodologies, although based on well-established concentration-response functions (WHO, 2013), fail to capture the potential risks associated to particles with significantly different characteristics (in terms of size distribution and toxicity). This is the result of the fact that the majority of the CRFs rely on older time series of PM_{10}, mainly originated from long range transport, industrial, traffic and space heating sources. As a result, these CRFs rely on particles with different characteristics to the ones produced from intense biomass burning.

Considering the above, the development of a more refined methodology for addressing the differences in lung cancer taking stock of the differences in size distribution and chemical specificity creates the impetus for a paradigm shift. In contrast to other health endpoints, exposure is linked to hazard using a toxicologically derived slope factor. On the contrary, CRFs have been obtained from observational studies, reflecting exposure to particles with specific characteristics. In terms of lung cancer risk, it is worth mentioning that although PAH-TEQ levels are increased by 4 times during the cold season, the respective risk increases by almost 6 times. This is the result of the different particle size distribution of biomass emitted PM compared to the traffic-dominated particulate matter; $PM_{2.5}/PM_{10}$ fraction increases significantly and the particles of smaller size tend to penetrate deeper in human respiratory tract, which in turn have higher TEQ levels per PM mass, thus increasing the overall amount of PAHs deposited across human HRT and the respective lung cancer risk.

Finally, this study provides additional insights related to the toxicity of biomass burning emitted PM. There is a long controversy about biomass-emitted toxicity. Chemical analysis (levoglucosan and PAHs) of a large number of samples (two sites, 2 different PM fractions for a consecutive seven months period) carried out in this study provided important information so as to draw conclusions regarding

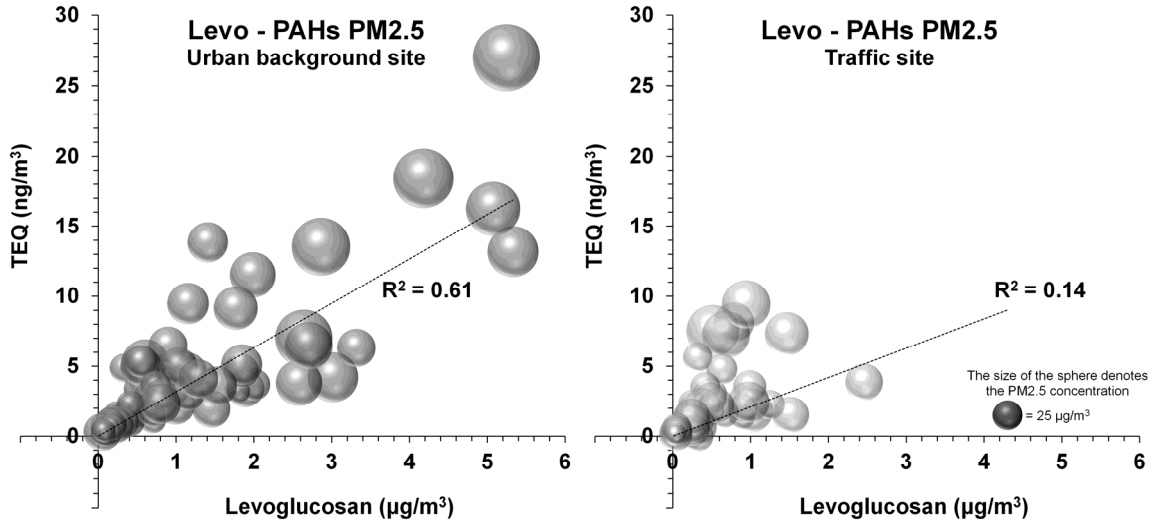


Fig. 4. Correlation between levoglucosan and PAHs levels of $PM_{2.5}$ for the urban background and the traffic site respectively.

the toxicity of the particle-adsorbed PAHs in terms of PAH-TEQ. Biomass combustion generates a large number of fine and ultrafine particles. It is known that biomass burning emitted PM are of very low aerodynamic diameter: most of them (about 80 to 90%) have aerodynamic diameter below 1 μm (Rissler *et al.*, 2006). In addition, the fraction of incomplete combustion emissions is very significant, especially under burning conditions such as the ones found in open fireplaces, resulting in significant formation of PAHs (Lamberg *et al.*, 2011). It is also well established, that the most carcinogenic PAHs compounds (5- and 6-ring), including BaP, are mostly adsorbed to finer particles (Lin *et al.*, 2008). Under these conditions, it would be reasonable to suggest that biomass burning emitted particles and traffic emitted particles are quite similar in PAHs toxicity. Our work overrides this assumption, as it has shown that biomass burning emitted PM with higher PAHs-TEQ content per mass of particle, based on the results of the two measurements sites (0.083 and 0.066 ng μg^{-1} PM for $PM_{2.5}$ and PM_{10} respectively in the urban background site and 0.44 and 0.032 ng μg^{-1} PM for $PM_{2.5}$ and PM_{10} respectively in the traffic site). As depicted in Fig. 4, levoglucosan levels are very well correlated to the TEQ levels in the urban background station, but this is not the case for the traffic station.

CONCLUSIONS

The current study deals with the effects of particulate and gaseous emissions from biomass burning in terms of human exposure, public health burden and the associated monetary cost. Towards this aim a comprehensive assessment framework was developed, combining field measurements, chemical analysis and modelling of IAQ (validated by measurements) and exposure refinement employing HRT deposition for detailed intake fraction estimation. At the same time, we avoided the overestimation of actual daily exposure that would result from just using ambient air levels for health and monetary impact estimation. Results indicated an increase in overall PM exposure of about 40% compared to the normal urban background levels; this value becomes higher for larger particles. Generally, the transition to the cold period (mid-November to mid-March) is accompanied by increased mortality accounting for ca. 200 (expressed on an annual basis) for a population of almost 900.000, reflecting a marginal cost of almost 250 million euro. A very important conclusion of the current study is that biomass emitted particles are i) more toxic in terms of PAH content compared to the ones related to other sources and ii) of lower aerodynamic diameter; as a result, more refined exposure and risk characterization methods are needed so as to properly account for their potential health effects. A take on a comprehensive exposure and health impact assessment of biomass burning in cities is given in this study.

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