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Health impact and monetary cost of exposure to particulate matter emitted from biomass burning in large cities

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HIGHLIGHTS

- Health and monetary impact assessment of exposure to PM from biomass burning.
- 200 excess deaths annually (for a 900,000 population) are expected in the cold season.
- The respective monetary cost ranges from 200m€ to 1.2b€.
- Monetary cost of health burden compounds the fiscal burden of austerity measures.

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ABSTRACT

The study deals with the assessment of health impact and the respective economic cost attributed to particulate matter (PM) emitted into the atmosphere from biomass burning for space heating, focusing on the differences between the warm and cold seasons in 2011–2012 and 2012–2013 in Thessaloniki (Greece). Health impact was assessed based on estimated exposure levels and the use of established WHO concentration–response functions (CRFs) for all-cause mortality, infant mortality, new chronic bronchitis cases, respiratory and cardiac hospital admissions. Monetary cost was based on the valuation of the willingness-to-pay/accept (WTP/WTA), to avoid or compensate for the loss of welfare associated with illness.

Results showed that long term mortality during the 2012–2013 winter increased by 200 excess deaths in a city of almost 900,000 inhabitants or 3540 years of life lost, corresponding to an economic cost of almost 200–250m€. New chronic bronchitis cases dominate morbidity estimates (490 additional new cases corresponding to a monetary cost of 30m€). Estimated health and monetary impacts are more severe during the cold season, despite its smaller duration (4 months). Considering that the increased ambient air concentrations (and the integral of outdoor/indoor exposure) are explained by shifting from oil to biomass for domestic heating purposes, several alternative scenarios were evaluated. Policy scenario analysis revealed that significant public health and monetary benefits (up to 2b€ in avoided mortality and 130m€ in avoided illness) might be obtained by limiting the biomass share in the domestic heat energy mix. Fiscal policy affecting fuels/technologies used for domestic heating needs to be reconsidered urgently, since the net tax loss from avoided oil taxation due to reduced consumption was further compounded by the public health cost of increased mid-term morbidity and mortality.

1. Introduction

Indoor biomass burning for residential heating is a major source of indoor and outdoor air quality degradation. One of the major components of biomass burning is emitted PM. The association of biomass burning PM with potential health effects, as well as their toxicity potency in comparison to other PM combustion sources have been investigated through a controlled study of human exposure to wood smoke, epidemiological studies (observational or interventional), as well as with toxicological tests — a very comprehensive review on both perspectives was carried out by Naeher et al. (2007).

To date, solely a single controlled study of human exposure to wood smoke has been published (Barregard et al., 2006). Inflammatory mediators and coagulation factor levels of the exposed subjects were altered and free radical-mediated lipid peroxidation increased after wood smoke exposure. Although this is the only controlled study of wood smoke exposure published to date including a small number of subjects (13), it is suggestive of wood smoke-associated systemic inflammatory effects.

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The majority of information regarding direct human health effects associated with wood smoke exposure is derived from a relatively large number of epidemiologic studies that have documented respiratory effects of residential wood burning, especially in children (Naeher et al., 2007). One of the earliest studies was conducted in Michigan by Honicky et al. (1985) who compared respiratory symptoms in 31 children who lived in homes with wood stoves with an equal number of children who lived in homes without wood stoves. Exposure to smoke from wood stoves resulted in exacerbation of severe symptoms of respiratory diseases. Related health effects research in Seattle shows associations between PM2.5 exposure and lung function decrements in children (Koenig et al., 1993), visits to the emergency departments for asthma (Norris et al., 1999), hospitalizations for asthma (Sheppard et al., 1999), and increases in asthma symptoms in children (Yu et al., 2000), as well as increases in exhaled nitric oxide (Koenig et al., 2005). Considering that wood burning is one of the major sources of exposure to fine PM in the Seattle area, the related studies resulted in significant associations between wood burning PM and adverse health outcomes. Another study examined the relationship of woodstoves to otitis media and asthma in a case–control study of home environmental air pollutants in Springville, NY (Daigler et al., 1991). Use of biomass burning sources resulted in increased incidence rate of several adverse health effects such as otitis media and severe shortness of breath. Thus, indoor biomass burning was identified as the second most important source of exposure to fine PM after environmental tobacco smoke (Ostro et al., 1991).

In the study of Happo et al. (2013) health-related toxicological properties of PM1 emissions from five modern and two old technology appliances were examined. Mice were intratracheally exposed to a wide range of particulate samples and then bronchoalveolar lavage fluid (BALF) was assayed for indicators of inflammation, cytotoxicity and genotoxicity. The results indicated that although older technologies emit higher amounts of PM1 per energy unit produced, PM1 emitted from modern appliances induced higher inflammatory potential, probably due to ash-related compounds.

A recent meta-analysis by Diette et al. (2012) concluded that inhalation of wood smoke at a relatively low level, had the potential to suppress the immunity of the respiratory system, resulting in increased susceptibility to infections as well as to several types of lung disease.

To date, only a limited number of studies have evaluated the results of shifting between biomass and conventional fuel for domestic heating (intervention studies). The most comprehensive study up to now is an intervention study in Australia carried out by Johnston et al. (2013) who evaluated the effect of community education campaigns, enforcement of environmental regulations, and a wood heater replacement program on daily mortality. This bundle of actions resulted in a 17 μg/m^3 reduction of ambient air PM10 in the wintertime, which, was reflected, in turn, in reduced annual cardiovascular and respiratory mortality, especially for males.

In their study in Austria Haluza et al. (2012) estimated that replacement of light oil by biomass as well as fossil gas would result in increased mortality and morbidity due to the related hike in PM ambient air levels. The higher the biomass energy share, the higher the PM attributed mortality and morbidity. Although interesting from the perspective of scenario analysis, the use of PM10 as input in concentration–response functions, does not seem to fully capture the variability on PM concentrations related to biomass burning. Thus inter-scenario differences might well be underestimated.

Despite the uncertainties related to monetization of air pollution related impacts, significant efforts have been made based on the Value-per-Statistical-Life (VSL) concept. Alberini et al. (2006) concluded that VSL is not significantly lower for older persons, but income was positively associated with willingness to pay (WTP). Roman et al. (2012) elucidated the uncertainties related to VSL for a reduction in mortality risks, while Ortiz et al. (2011) identified altruism towards children, and a strong income effect on WTP to avoid the loss of welfare associated with illness. In the recent years, scientific interest has shifted towards the assessment of climate change impact health costs. The respective cost is in the range of 100 billion euros annually in the EU-27 region (Watkiss and Hunt, 2012). Finally, the study carried out by Desaigues et al. (2011), aimed at the economic valuation of air pollution mortality. Based on a 1493 person survey carried out in 9 EU countries, the monetary value of a life year (VOLY) was estimated to be equal to 25,000 euro.

Over the last couple of years, the use of biomass as heating source was allowed in Greece as a CO_2-neutral means of space heating in the large metropolitan areas of Athens and Thessaloniki affecting more than half of the country's population. At the same time the use of light heating diesel was heavily taxed apparently to combat illegal trafficking of heating diesel. In the same period Greece faced a financial crisis with significant repercussions on the average household income. That combination of parameters resulted in increased use of biomass for residential heating in year 2012, followed by a significant increase of ambient air, indoor air and exposure to PM10 and PM2.5. In this study, we aim to quantify the health and socioeconomic effects related to that shift from light heating diesel to biomass burning, as well as to evaluate alternative scenarios of residential heating energy share.

2. Methodology

2.1. Exposure assessment

To better reflect the effect of biomass burning on public health, actual population exposure was estimated. Thus, indoor concentrations taking into account outdoor penetration as well as indoor sources (biomass burning among them) were estimated. The estimation was based on a mass balance model, that takes into account the major processes governing particle concentration i.e., emissions, deposition, indoor/outdoor exchange rate, and outdoor infiltration. A detailed description of the indoor air model and the related parameterization has been given by Sarigiannis et al. (2014) on the model and the related parameterization. In summary, the mass balance is described by the following formula:

$$V \cdot \frac{dC_{ind}}{dt} = Q \cdot (inf \cdot C_{out} - C_{ind}) + E - k_{dep} \cdot C_{ind} \cdot V$$

where:

E	*the strength of the emission sources* (*mass* / *time*)
V	*the volumes of the indoor location*
C_{out}	*the outdoor concentration*
C_{ind}	*the indoor concentration of the indoor location*
Q	*the indoor–outdoor air exchange rate*
inf	*the infiltration fraction*
k_{dep}	*the deposition rate*

Particle penetration into buildings from the ambient air depends on pollutant species, geometry, surface materials and pressure drop across the leakage path. It is usually expressed by means of a dimensionless penetration factor; typical values of the latter for domestic environments were taken from the PTEAM study (Özkaynak et al., 1996). These values are normally distributed with a mean value of 1 and a standard deviation of 0.06. Background emission rates for PM10 and PM2.5 were taken equal to 5600 and 1400 μg/h respectively (not including indoor smoking or long-term burning activities). These values are considered to be representative of Greece (Hänninen et al., 2004). Biomass burning emissions into the indoor environment from open fireplaces and other biomass combustion sources (e.g., stoves) were estimated experimentally in our study, found to be equal to 2300 μg/h. Deposition of atmospheric aerosol particles on indoor surfaces (the floor, walls, ceiling and furniture) takes place via electrostatic and thermostatic processes, and it is significantly affected by the type of air mixing (turbulent vs.

laminar flow). The distribution for this variable was taken from the PTEAM study (Özkaynak et al., 1996) and the respective values for PM10 and PM2.5 were 0.67 and 0.39 h^{-1} respectively. Finally, a typical residence volume equal to 245 m^3 was considered as representative (UNECE, 2006).

2.1.1. Personal exposure

Personal exposure is the average concentration of PMx to which an individual is exposed to over a given period of time. If over time T the individual goes through n locations, spending a fraction f_n of time in location n where the concentration of the pollutant under consideration is C_n, then the personal exposure for this period T, represented by the concentration E_T, is given by (Ott, 1982):

$$E_T = \sum_n f_n \cdot C_n.$$

Micro-environments were differentiated in terms of time spent within them. Time-weighted factors were used, based on the time–activity data from the EXPOLIS study enriched with data from the MTUS[1] database. Exposure factors used were cross-checked against the European Commission's EXPOFACTS database.[2] Thus, to estimate exposure, we used information on actual detailed time activity patterns, linking several types of activities and their respective duration to specific indoor micro-environments. By using detailed activity patterns and linking them to specific micro-environments an additional factor influencing the actual human intake of PMx, namely inhalation rate, was accounted for. Different types of activities demand different levels of effort that correspond to different inhalation rates. A detailed description of activity-dependent inhalation rates is given in Sarigiannis et al. (2012). For this purpose, based on the time weighed contribution of individual activities, their intensity and the corresponding inhalation rate, we derived adjustment factors for each location and activity type. As a result, PMx intake was given by the following formula:

$$E = \sum_n f_{loc} \cdot C_{loc} \cdot \mathrm{inh}_{\mathrm{act}}$$

where $\mathrm{inh}_{\mathrm{act}}$ is the inhalation adjustment factor for each type of micro-environment and activity encountered in the calculations.

2.2. Health impact assessment

To estimate the health effects of PM exposure, we used well established epidemiological concentration–response functions for outdoor PM (WHO, 2005). Differences in toxicity depending on particle composition (e.g., concentration and types of PAHs adsorbed) were not taken into account, since the current level of epidemiological knowledge does not allow the use of different concentration–response functions based on the differences in composition, an opinion which is also supported by WHO (2007). Different concentration–response functions might be used only for particles of different aerodynamic diameters (PM10 and PM2.5). Other authors have come to similar conclusions. According to Boman et al. (2006), biomass burning related health outcomes were similar to the ones observed from acute effects of exposure to PM from different sources (Boman et al., 2003), concluding that there is no reason to consider biomass burning PMs less toxic. Moreover, a limited number of studies provided evidence on the impact of biomass smoke exposure on lung cancer (Behera and Balamugesh, 2005; Hernández-Garduño et al., 2004). Similarly, there is relative paucity of established cohort results on outcomes such as asthma and cardiovascular disease related to PM that originated from indoor biomass use. This is in contrast to other PM sources that have been extensively evaluated with regard to their effects on human health, such as sidestream smoke.

Based on an extensive review of studies investigating the toxicity of PM, Naeher et al. (2007) concluded that the scientific evidence from the studies existing so far is sufficient enough to justify that PM emitted from biomass combustion is no less toxic than PM emitted from other sources. A recent study from our group (Sarigiannis et al., 2015) determined age-differentiated lung cancer risk from PAHs adsorbed onto particulate matter emitted from biomass burning. When differences in respiratory tract physiology in different age groups and lung deposition dynamics of different PMx size fractions are taken into account a significant variation in exposure to PAHs is observed, resulting in similarly varying lung cancer risks. Based on the difference of the size fraction between particles emitted from biomass burning and the ones emitted from traffic and industrial sources, the estimated lung cancer risk from biomass-derived particles may be higher than the risk from other types of atmospheric particulate matter.

Among the currently regulated pollutants in wood smoke, fine particles (PM2.5) serve as the best exposure metric in most circumstances (Sarigiannis et al., 2014). In addition, PM2.5 tends to be among the most elevated in relation to existing air quality standards. The concentration–response functions used in this study are given in Table 1. For endpoints where original PM2.5 functions are not available, the respective PM10 CRF estimates were converted to estimates of PM2.5 by multiplying the PM10 CRFs by a conversion factor obtained from the con-current measurements of PM10 and PM2.5 in the Apheis-3 study (APHEIS, 2004). Apheis-3 measured PM10 and PM2.5 in 26 cities — 11 of the cities had simultaneous PM10 and PM2.5 measurements. On average, the ratio of mass concentration of PM2.5 to PM10 was about 0.65 and ranged from 0.5 in Gothenburg, Sweden to 0.71 in Rouen, France. The PM2.5/PM10 ratio was used as the conversion factor between PM2.5 and PM10.

As the relationship between PM measurements and health effects is log-linear, the conversion from PM10 CRFs to PM2.5 CRFs followed the mathematical formula below:

$$\mathrm{PM}_{2.5}\mathrm{CRF} = \exp(\ln(\mathrm{PM}_{2.5}\mathrm{CRF})) = \exp\left(\frac{\ln(\mathrm{PM}_{10}\mathrm{CRF})}{0.65}\right).$$

The detailed analysis for the extrapolation of PM10 concentration–response functions is given in the final deliverable of the HEIMTSA project case studies report (IOM, 2011), based on the initial concept described by WHO (WHO, 2004d). Briefly, the epidemiological studies from where the CRFs for the endpoints of interest were retrieved are given below:

Infant mortality: Quantification of PM10 effects on infant mortality followed the CAFE study (Hurley et al., 2005), which was based on a cohort of four million infants (Woodruff et al., 1997). Post-neonatal infant mortality is considered as death between the ages of one month and one year. The associations reported between infant mortality (all-cause) and PM10 produced a CRF (relative risk) of: 4% (95% CI: 2%, 7%) for increase in infant mortality per 10 μg/m^3 PM10.

Chronic bronchitis (adults): Schindler et al. (2009) reported on the relationship between chronic bronchitis in the SAPALDIA cohort and PM10. Their study comprised 7019 participants who had completed the follow-up questionnaire, lived at the same address for at least one year before baseline and had sufficiently complete core data. Chronic bronchitis here is defined as chronic cough and/or chronic phlegm, which persisted for at least 3 months per year for the last 2 years. By processing the data in the CAFE study (Hurley et al., 2005), an impact function for new cases of chronic bronchitis was derived, estimating a 22% (95% CI: 2%, 38%) increase per 10 μg/m^3 PM10. Under the coordination of WHO existing data

[1] MTUS: Multinational Time Use Study (http://www.timeuse.org/mtus/).
[2] EXPOFACTS: Exposure Factors database maintained by the European Commission's Joint Research Centre (http://expofacts.jrc.ec.europa.eu/).

Table 1
Concentration–response functions (CRFs) for the several endpoints addressed in the study.

Health endpoint	CRF	Reference	Background rate	Age group
Mortality (all causes)	6.2% (95% CI: 4%, 8.3%) change per 10 μg/m^3 $PM_{2.5}$	WHO (2013)	8417 annual deaths/881,288 population (EUROSTAT, 2011; WHO, 2008), life table data (WHO, 2014a)	Adults aged 30 years and older
Infant mortality	4% (95% CI: 2%, 7%) change per 10 μg/m^3 PM_{10}	Hurley et al. (2005) and IOM (2011)	145 post-neonatal deaths per 100,000 live births (9141 annual births) (EUROSTAT, 2011; WHO, 2008)	1 month to 1 year
Chronic bronchitis (adults)	11.7% (95% CI: 4%, 18.9%) change per 10 μg/m^3 PM_{10}	WHO (2013)	390 new cases annually per 100,000 adults at risk (adjusted for remission — remission rate of 56.2%) (Schindler et al., 2009)	Adults aged 18 years and older
Chronic bronchitis (children)	1.08% (95% CI: −2%, 19%) change per 10 μg/m^3 PM_{10}	Hoek et al. (2012) and WHO (2013)	18.6% (Hoek et al., 2012)	Children aged 6–12 years
Cardiac hospital admissions	0.6% (95% CI: 0.3%, 0.9%) change per 10 μg/m^3 PM_{10}	Hurley et al. (2005) and IOM (2011)	723 emergency cardiac admissions per 100,000 population, all ages, per year (Hurley et al., 2005)	All ages
Respiratory hospital admissions	0.9% (95% CI: 0.7%, 1.0%) change per 10 μg/m^3 PM_{10}	Hurley et al. (2005) and IOM (2011)	617 emergency respiratory hospital admissions per 100,000 population, all ages, per year (Hurley et al., 2005)	All ages

were re-analyzed in the frame of the Health Risks of Air Pollution in Europe (HRAPIE) project (WHO, 2013). Based on the recently published systematic review of studies on the relationship of COPD to air pollution (Schikowski et al., 2014), the HRAPIE team produced revised estimates, which brought the CRF to an 11.7% (95% CI: 4%, 18.9%) change per 10 μg/m^3 of PM10.

Chronic bronchitis (children): The WHO (2013) evaluated the results of the PATY study (Hoek et al., 2012), which analyzed data collected by cross-sectional studies previously conducted in 11 countries. A borderline significant association of bronchitis prevalence with long-term average PM10 concentration in cities was reported with a RR of 8% (95% CI −2%, 19%) per 10 μg/m^3 annual mean PM10.

Cardiac hospital admissions: According to a WHO meta-analysis of PM10 and cardiovascular hospital admissions, there is stronger evidence that daily variations in PM are related to cardiac hospital admissions (i.e., those affecting arteries near the heart itself) than to cerebrovascular admissions (stroke, etc.) or to cardiovascular admissions in general (WHO, 2004b,c). Based on this conclusion, the effects of PM10 on cardiac hospital admission (ICD 9 codes 390–4292) have been quantified. The Apheis-3 report includes a CRF linking PM10 and cardiac hospital admissions based on data from the APHEA-2 study of eight European cities (Barcelona, Birmingham, London, Milan, Paris, Rome, Stockholm and the Netherlands, considered as one large urban area) (Le Tertre et al., 2002). The derived CRF for cardiac admissions (relative risk) for all ages was a 0.6% (95% CI: 0.3, 0.9%) change in cardiac hospital admissions per 10 μg/m^3 PM10.

Respiratory hospital admissions: The WHO meta-analysis of hospital admissions and PM10 provides European risk estimates for different age groups (WHO, 2004c). For adults aged 65 and older, studies in eight cities, six of them from the APHEA 2 study, were included in the meta-analysis producing a CRF of 0.7% (95% CI: 0.2%, 1.3%) per 10 μg/m^3 PM10 in adults aged 65 and older. For people aged 0–64, information on daily variations in PM10 and respiratory hospital admissions was only found for three European cities — two in England and one in Rome (WHO, 2004b,c). The three studies reported rates separately for the 0–14 and 15–64 age groups. The results for the two age groups were similar and the overall 0–64 CRF is roughly 1% (95% CI: 0%, 2%) per 10 μg/m^3 PM10 in people aged 0–64. Previously published systematic reviews and meta-analyses from Europe as well as North America were incorporated in the meta-analysis ensuring that the most up-to-date estimates were included in the overall risk estimate. Eleven risk estimates were included producing a CRF (relative risk) for respiratory hospital admissions of 0.9% (95% CI: 0.7, 1%) change in respiratory hospital admissions per 10 μg/m^3 PM10.

Mortality: For quantification in Europe the CAFE cost–benefit analysis (CAFE CBA) used a relative risk (RR) estimate of 1.06 for all-cause mortality hazards (95% CI 1.02–1.11) per 10 pg/m^3 PM2.5. This estimate was taken from analyses of American Cancer Society (ACS) data by Pope et al. (2002), and was based on a recommendation by the Task Force on Health (UNECE/WHO, 2004; WHO, 2006; UNECE/WHO, 2004). It reflects an effect of exposures averaged over the 16+ years of mortality follow-up. The relative risk estimate used in CAFE is different from the corresponding RR estimate of 1.04 (95% CI 1.01–1.08), in all-cause mortality hazards, used by the WHO Global Burden of Disease (GBD) project (WHO, 2004a) for quantification of mortality effects worldwide. That lower estimate was also taken from Pope et al. (2002); it expresses the effect of exposure as measured close to the start of the follow-up period. The experts team participating in the HRAPIE study under the coordination of WHO (2013) re-analyzed all cohort studies published before January 2013 (Hoek et al., 2013). Thirteen different studies conducted in the adult populations of North America and Europe contributed to revising the respective estimates, which bring the CRF to 1.062 (95% CI 1.040, 1.083) per 10 μg/m^3.

Relative risk was calculated for the average concentration X, using the following for $PM_{2.5}$:

$$RR = CR\,F^{\left(\frac{X}{10}\right)}.$$

The exponential is used because the original analyses used a proportional hazard, i.e., log-linear, regression model. From this relative risk was derived the attributable fraction *AF* which was derived as follows:

$$AF = \left(\frac{RR-1}{RR}\right)$$

and this was multiplied by the background rate of disease *BR* to derive the estimated health impact *HI* as the number of cases expected to represent the respective adverse health outcome in the population of interest *P*. Thus, the health impact *HI* is given as:

$$HI = AF \cdot BR \cdot F.$$

The actual burden of mortality (i.e., the attributable death due to $PM_{2.5}$ exposure) was expressed in years of life lost (YLL). To estimate YLL, life tables (WHO, 2014b) were employed, where national population and the corresponding mortality data per age band are used. These data are used to derive the population-weighed annual average

Table 2
Monetary evaluation (€) of health endpoints considered in this study.

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)
Mortality (all cause) based on VOLY	25,000	40,000	100,000	Desaigues et al. (2011)
Infant mortality	1,120,000	2,475,000	11,200,000	Holland et al. (2004)
Chronic bronchitis (children and adults)	43,000	60,000	100,000	Krupnick and Cropper (1992)
Cardiovascular hospital admissions	2990	2990	8074	Holland et al. (2004) and Navrud (2001)
Respiratory hospital admissions	2990	2990	8.074	Holland et al. (2004) and Navrud (2001)

concentration for a pollutant *C*. The attributable fraction *AF* is multiplied with the background mortality rate *D* for the given age band in order to compute the number of attributable deaths (*AD*) per age band.

$$AD = AF \cdot D.$$

The number of attributable deaths *AD* per age band was then multiplied with the expected years of life (Y_{LE}) for the given age band

$$YLL = AD \cdot Y_{LE}.$$

Demographic data regarding the population and mortality/morbidity rates distribution for the several age groups were obtained from the EUROSTAT databases (EUROSTAT, 2011). Additional data for background rate of disease and mortality were obtained by WHO (2008). The CRFs used for the selected health endpoints are given in detail in Table 1.

2.3. Monetary cost

The starting point for the valuation of health end-points is the identification of the components that comprise changes in welfare. These components should be added to reckon the total welfare change, assuming no overlap between impact categories to avoid double-counting:

- Resource costs i.e., medical costs paid by the health service or covered by insurance, and any other personal out-of-pocket expenses made by the individual (or family).
- Opportunity costs i.e., the cost in terms of lost productivity (work time loss or performing at less than full capacity) and the opportunity cost of leisure (leisure time loss) including unpaid work.
- Dis-utility i.e., other social and economic costs including any restrictions on or reduced enjoyment of desired leisure activities, discomfort or inconvenience (pain or suffering), anxiety about the future, and concern and inconvenience to family members and others.

The welfare changes represented by components (i) and (ii) can be approximated using existing market prices for these. This measure needs to be added to a measure of the affected individual's loss of utility, as the latter is reflected in a valuation of the willingness-to-pay/accept (WTP/WTA), to avoid/compensate for the loss of welfare associated with the illness. For the endpoints considered in this paper, Table 2 presents a summary of the unit values derived in the course of the HEIMTSA project (UBath, 2011). These values are the result of both an evaluation of the evidence available in the existing literature and ad hoc population surveys in several European countries.

For each health endpoint unit values are identified on the basis of an informal meta-analysis of the evidence, which accounted for the distribution of available values and an assessment of the quality and geographical focus of each study, incorporating thus considerable uncertainty in health valuation. Uncertainty derives from a combination of (a) paucity of the evidence base, (b) difficulty that people have with identifying their preferences for (avoidance of) health conditions, and (c) lack of maturity in the study methods themselves. Nonetheless, in the view of the wealth of data upon which they are based, the unit values given in Table 2 are robust estimates of both the central estimate and the upper and lower bounds of monetary valuation for each of the health endpoints considered in our study.

Mortality costs of air pollution were also estimated based on change of life expectancy (LE), using the value of a life year (VOLY) as monetary valuation metric. According to the VOLY methodology air pollution premature mortality results in shorter LE loss per death compared to other causes of mortality such as fatal accidents (resulting in 30–40 years of LE loss), upon which VSL estimates are based (Desaigues et al., 2011).

2.4. Scenario analysis

The results of the 2012–2013 measurements were compared to the ones made in 2011–2012 to understand better the effect that different policy measures regulating the market price of heating fuel in tandem with the incumbent economic crisis in Greece and other countries in the European South may have on non-occupational exposure of the urban population to particulate matter and the associated health and monetary impact. Own-price elasticity of light heating oil was taken as $el_{oil} = -0.39$ (IOBE, 2013). A field survey encompassing ca. 300 households across the greater area of Thessaloniki implemented using the online SurveyMonkey tool provided consumer behavior information that was used to generate the cross-fuel elasticity table below (Table 3).

The scenarios are based on reasonable assumptions and existing trends related to the energy market share of different fuels and heating systems (e.g., the market share of natural gas use for space heating had been increasing over the last years but it is currently stabilized to around 40%). However the interplay of multiple factors such as financial pressures or incentives might result in unexpected figures (as occurred with the increased biomass use), favoring one technological solution for space heating over another. Through analysis of specific scenarios we highlighted the attributable differences in public health burden, should specific space heating practices be adopted. The different policy scenarios examined (and the corresponding average urban background concentrations) are presented in Table 4.

Differences in the urban background concentration, which in turn were reflected in population exposure to PM for the intervention scenarios examined herein, were estimated based on a data assimilation algorithm coupling: (a) a regulatory atmospheric dispersion model (CALPUFF) (Scire et al., 2000a, 2000b) following a Lagrangian Gaussian formulation applied on area sources that was supported by a refined and up-to-date emissions inventory at high spatial resolution; (b) the OSPM model (Berkowicz et al., 2008) for capturing traffic-related emissions taking into account street canyons; and (c) spatially distributed

Table 3
Cross-price elasticities of alternative space heating energy carriers.

	Light heating oil	Natural gas	Biomass	Electricity
Light heating oil	–	n/a	−0.97	−0.24
Natural gas	n/a	–	n/a	n/a
Biomass	−1.03	n/a	–	0.25
Electricity	−4.1	n/a	3.98	–

n/a: sufficient data not available to support the estimation of elasticity.

Table 4
Fuel/technology use distribution and corresponding urban background concentrations.

Scenario	Oil	Natural gas	Biomass burning	Electricity	PM2.5 (μg/m^3)
2011–2012	44.0%	40.0%	5.6%	10.4%	41.2 (measured)
2012–2013	22.3%	40.0%	26.7%	15.7%	62.6 (measured)
Scenario 1	38.5%	41.5%	10.0%	10.0%	36.3
Scenario 2	43.5%	41.5%	5.0%	10.0%	28.4
Scenario 3	23.5%	62.5%	4.0%	10.0%	26.5
Scenario 4	20.0%	70.0%	0.0%	10.0%	20.0

measurements of PMx concentrations using ground monitors across the Thessaloniki metropolitan area. The computed annual average concentrations at the receptor points for the OSPM and the CALPUFF models together with the observations from air pollution monitoring stations across the greater area of Thessaloniki are used as input to a spatial interpolation model employing ordinary kriging. The relative weight of the pollution estimates from the atmospheric models and ground monitors are optimized to reduce the residual error based on an advanced Kalman filter formulation.

The following figure shows the resulting PM2.5 map of the metropolitan area of Thessaloniki in 2011 (Fig. 1).

In order to simplify the estimation of the effect that different space heating fuels and technological options have on the atmospheric burden with particulate matter in Thessaloniki an empirical model was built, correlating the 2011–2013 ambient air concentration data, the distribution of fuel/technology used for these years and the respective PM emission factors. The latter are equal to 0.36, 2.16 and 160 mg PM2.5/MJ for natural gas (boilers), oil (boilers) and biomass burning (open and closed fireplaces, wood stoves, pellet and wood log boilers) as derived from both an extensive literature review and experimental work regarding boiler emission efficiencies for different types of fuel (Nussbaumer et al., 2008). The emission factor for the biomass burning systems is a weighted average of the emission factors of each of the biomass combustion technologies considered weighed by the respective relative market shares (see Table 5). The latter were derived through the field survey described above.

The following regression model for the interpolation of projected ambient air PM concentrations during the cold period of a given year was derived based on the emission factors and the comparison of the concentration data between 2011/2012 and 2012/2013 cold and warm period data:

$$C_{cold} = C_{warm} + \mathrm{a} \cdot f_{oil} \cdot E_{oil} + \mathrm{b} \cdot f_{ng} \cdot E_{ng} + \mathrm{c} \cdot f_{bio} \cdot E_{bio}$$

where C_{warm} is the urban background concentration in the warm period of the same year, f_{oil}, f_{ng} and f_{bio} are the residential heating technological energy share for light heating oil, natural gas and biomass respectively and a, b, c are the regression coefficients (respectively equal to 0.95. 0.97 and 2) that best fit the results for the average winter urban background levels in the Thessaloniki area. Additional parameters that might affect the model were statistically insignificant, since the meteorological conditions between the two years did not differ substantially and long-range particulate pollution transport did not show significant variation for the duration of the study. During the cold period of 2011/2012 daily mean temperature, wind speed and precipitation were 12.1 °C, 3.7 km/h and 0.8 mm, while the respective values for 2012/2013 were 12.8 °C, 4.3 km/h and 1.3 mm. The number of heating degree days was higher during the 2011–2012 winter (1528 deg-days) compared to the 2012–2013 winter (1329 deg-days) (IOBE, 2013).

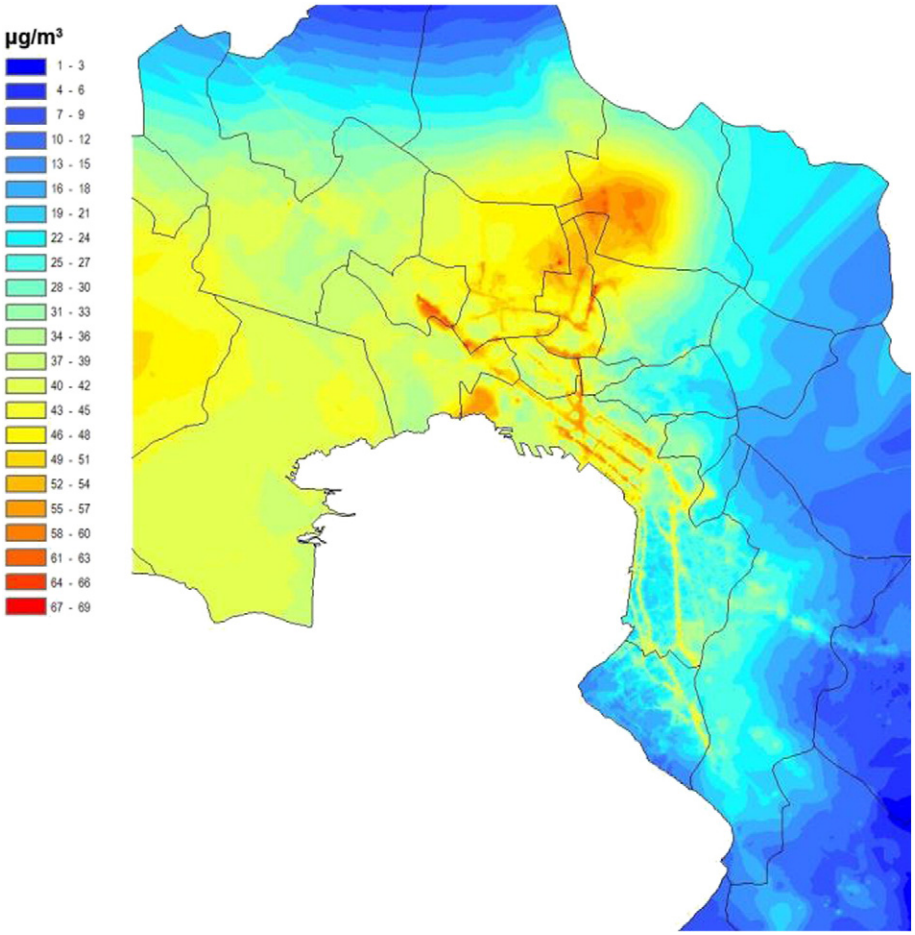

Fig. 1. PM2.5 concentration map in the greater area of Thessaloniki (2011).

Table 5
Emission factors and relative market shares of biomass combustion technologies.

Biomass burning technologies	Relative market share (%)	Average emission factor (mg PM2.5/MJ)
Open fireplace	30	160
Closed fireplace	10	41
Pellet boiler	10	21.6
Wood stove	40	240
Wood log boiler	10	96

2.5. Uncertainty assessment

All health impact and monetary valuation calculations were computed adopting a probabilistic framework that allows the incorporation of uncertainty and variability across the health impact pathway with Monte Carlo sampling. The latter involved a large number of samples (500,000 iterations were executed for each calculation on Crystal Ball; Oracle, 2011) based on the distributions of the input parameters. Literature-derived prior distributions with regard to CRFs and the monetary valuation were used for each health endpoint. 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, a log-normal distribution was fitted to the mean, min and max values (given in Table 2) based on prior knowledge on the type of probability distribution characterizing expression of societal preference with regard to willingness to pay. Exposure distributions were computed through the INTERA platform (Sarigiannis et al., 2012), based on the distributions of ambient air concentrations, air exchange rate, penetration coefficient, PM deposition and emission rates, already described elsewhere (Sarigiannis et al., 2015). Overall, a fully probabilistic heath impact and monetary valuation methodology was used in the study to ensure statistical robustness of the results and rigorous quantification of residual uncertainty.

3. Results

3.1. Health impact and monetary valuation

The estimated health impact and the corresponding monetary valuation for the selected mortality and morbidity indicators are illustrated in Figs. 2 to 8. Overall, the number of excess deaths (Fig. 2) attributed to PM exposure in the metropolitan area of Thessaloniki (with a population of more than 1 m inhabitants) for 2011 are 1369, while the respective number for 2012 is 1287. What is significantly different between these two years is the distribution between the warm and the cold seasons. Despite the fact that the duration of the cold period is practically half that of the warm period, in 2012 the impacts were higher in the cold period (728 out of 1288 annual deaths), in contrast to 526 cases out of 1369 (annually) in 2011. Thus, the air pollution burden associated with particulate matter during the cold season of 2012–2013 resulted in an estimate of more than 200 excess premature deaths attributable to airborne PM exposure than in the corresponding period in 2011–2012. Excess mortality during the cold period of the year corresponds to 3540 years of life lost (Fig. 3) taking into account the population age distribution.

Similar figures are derived for morbidity indices: new cases of chronic bronchitis (Figs. 5 and 6) dominate adverse health outcomes, followed by respiratory hospital admissions (Fig. 7) and cardiovascular hospital admissions (Fig. 8). In the remaining scenarios, impacts are gradually decreasing, reflecting the improvement of ambient and indoor air quality resulting from the reduction of biomass and the increase of natural gas use for space heating.

Monetary costs related to annual mortality are in the range of billion euros no matter whether we apply the value of statistical life (VSL) or the value of a life year (VOLY) methodology. Following the VOLY method, the estimated costs are about 30% lower, mainly reflecting the different costing structures of life expectancy lost according to the age pyramid of the population studied. Morbidity cost related to chronic bronchitis (especially for children) is, generally, one order of magnitude lower (hundreds of millions). Morbidity costs related to cardiovascular and respiratory morbidity (the most directly immediate effects of increased PM pollution) are in the range of million euros.

Scenario analysis showed that large benefits could be expected from potential policy interventions bearing changes in the composition of the fuel mix used for residential heating. These benefits are attributable to the differences in particulate emissions between biomass combustion and the competing fuels and space heating systems. The most effective way to achieving this result would seem to be a relaxation of the high taxation imposed in the last three years on heating oil in Greece.

4. Discussion

In this study the health impact and the associated monetary cost of non-occupational exposure to PM in the ambient and indoor air were assessed, focusing on the effect of the increased use of biomass as domestic heating source. Current methodological schemes for assessing impacts of biomass combustion rely on the association of indoor biomass burning with several adverse health outcomes. These studies are subject to major uncertainties related to the actual additional burden of exposure related to infiltration of outdoor air in the indoor air (Noonan and Balmes, 2010). Our methodology reduces these uncertainties by addressing the differences related to the variability of temporal emissions and the corresponding outdoor and indoor concentrations. In this way, the impact on the urban population is taken into account, distinguishing between households that use

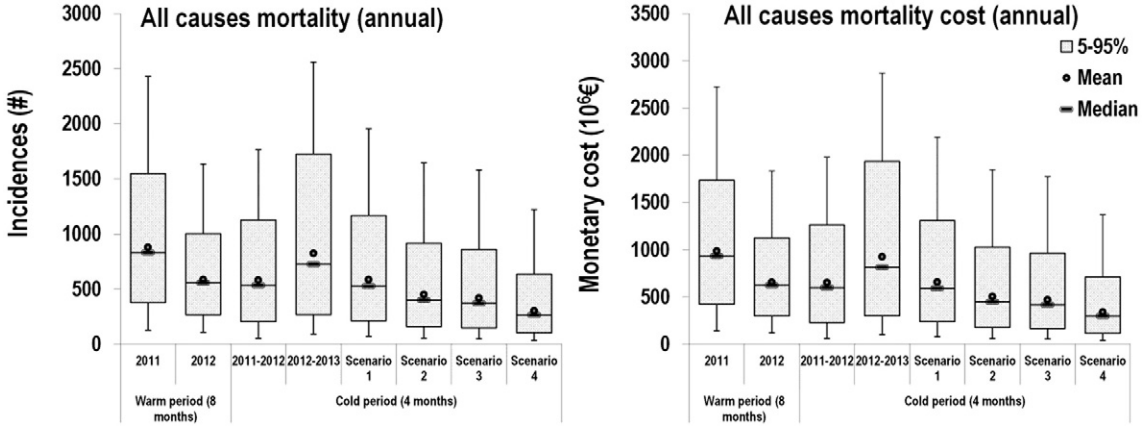

Fig. 2. All cause mortality and related monetary cost.

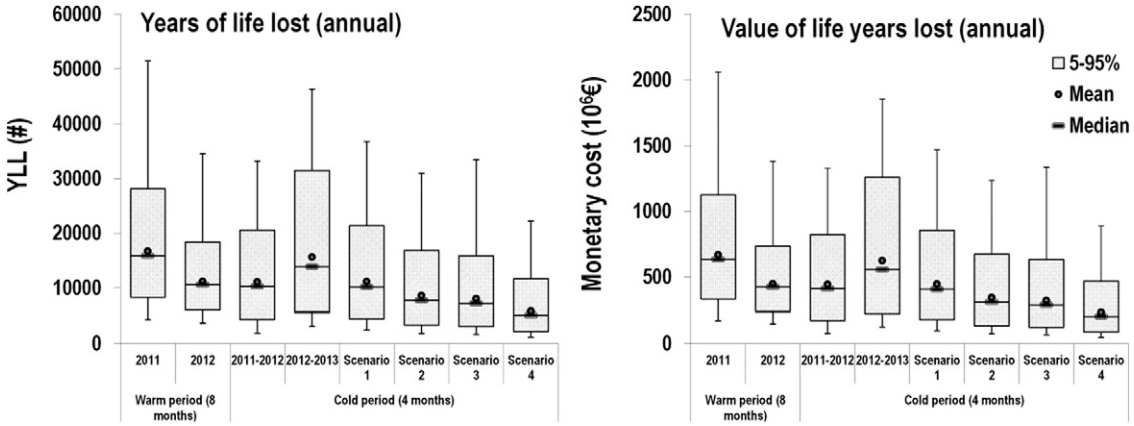

Fig. 3. All cause mortality (expressed in YLL) and related cost of life years lost.

biomass for indoor space heating and the ones that do not do so. The contribution of biomass combustion indoors is very important since uncontrolled biomass burning results in a significant degradation of the overall urban air quality with dire consequences on indoor air quality from ambient air penetration indoors. Moreover, biomass combustion sources emit particles directly into the indoor air burdening it even further. Quantifying the outdoor:indoor air interactions as in the present study is key for correctly estimating the overall health burden to the urban population. According to the WHO (2007), PM that originates from biomass burning is at least as harmful for public health as PM coming from different combustion sources (Naeher et al., 2007). This allows aggregate exposure to PM from different sources (traffic, other combustion and biomass burning) and environmental interactions (such as the outdoor-to-indoor air penetration) to be tackled within a unified modeling framework, thus providing an aggregate input for the estimation of the total health impact on the local population. Our team has shown recently that lung cancer risk from PAHs adsorbed onto PM emitted from biomass combustion is higher than the corresponding risk from PM in car exhaust (Sarigiannis et al., 2015). In this study, however, we shall adopt the WHO assumption and consider similar concentration–response functions to hold for PM emitted from all combustion sources.

Our results on health impacts concord with the ones derived by other recent studies. A direct comparison to a conceptually similar study carried out in Austria (Haluza et al., 2012), indicates that the expected health impact attributed to PM is higher in the city of Thessaloniki. This can be explained by the overall higher levels of PM, the incorporation of actual exposure and inhalation rate based on type and intensity of activity of the exposed population and of the indoor:outdoor interaction in our methodology. Moreover, we have used PM2.5 instead of PM10 as the exposure metric, considering that this is more relevant for explaining the adverse health effects of biomass burning.

Compared to the intervention study of Johnston et al. (2013), we underestimate cardiac effects. This could be due to the relative paucity of mechanistic information characterizing epidemiological studies and the respective CRFs. Wood smoke particles are generally smaller than 1 μm, with a size distribution that peaks between 0.15 and 0.4 μm (Hays et al., 2002). As with other combustion mixtures, such as diesel and tobacco smoke, fresh wood smoke contains a large number of ultrafine particles (UFPs) with aerodynamic diameter smaller than 100 μm. UFPs condense rapidly as they cool and age. Considering that biomass-originated PM is characterized by very low aerodynamic diameter, PM2.5 is rather a proxy than the actual exposure metric. When ultrafine PM is emitted to the atmosphere it undergoes several transformations (hydroscopic growth, condensation, etc.), which result in very fast particle growth and the formation of PM of a larger aerodynamic diameter (Sarigiannis et al., 2002; Sarigiannis et al., 2004). Cardiovascular disease attributed to exposure to particulate matter is related primarily to translocation of ultrafine particles from the respiratory tract through the alveoli via systemic circulation. Thus, cardiovascular problems and health effects not attributed to lung or systemic inflammation seem to be associated primarily with exposure to UFPs rather than larger particles (Brook, 2008). Overall, the quantification of exposure to airborne PM and the assessment of the associated health impact would benefit greatly from the incorporation of mechanistic elements such as human respiratory tract deposition and oxidative stress potential in the determination of the CRFs used for health impact assessment.

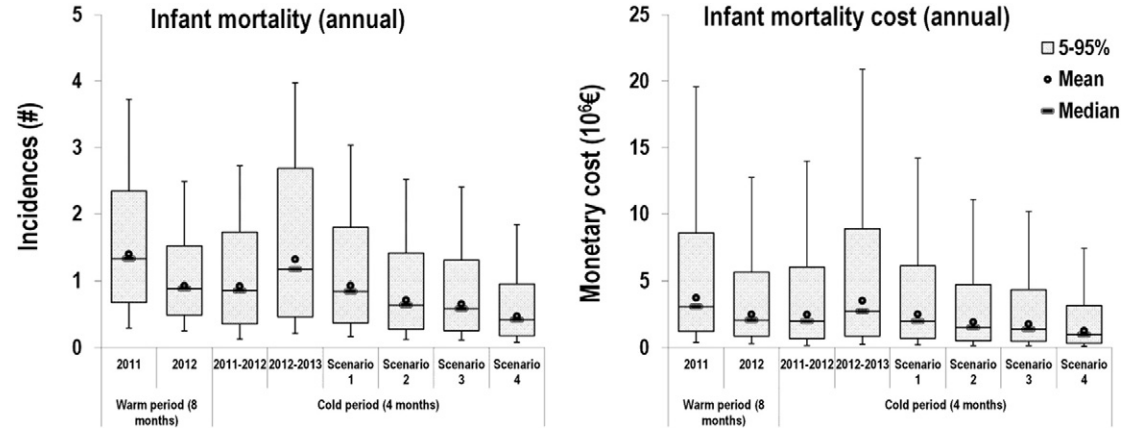

Fig. 4. Infant mortality and related monetary cost.

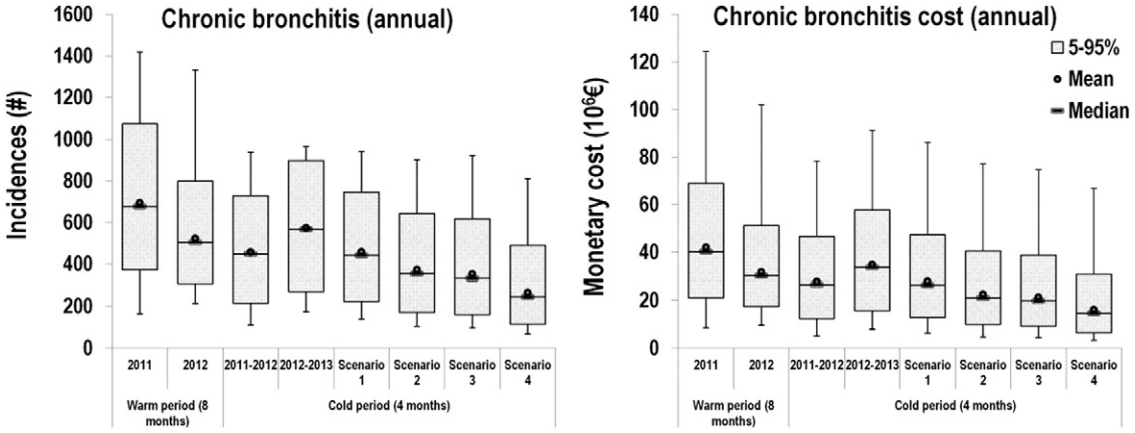

Fig. 5. Chronic bronchitis (adults) new cases and related monetary cost.

Socioeconomic status affects the use of energy resources and the activity patterns related to specific exposure scenarios of variable intensity. Several studies have shown that variations in socioeconomic status may have a significant impact on human exposure to pollutants. For example, children of lower socioeconomic status families are more vulnerable to asthma (Neidell, 2004; Pediatrics, 2000), while increased mortality due to exposure to PM10 concentrations has been associated with lower income (Forastiere et al., 2007). In addition, health status is also affected by behavioral factors, such as nutrition, smoking, alcohol consumption and physical activity (Arcus-Arth Broadwin and Lam, 2001; Adler and Newman, 2002); therefore, socioeconomically disadvantaged populations experience more often chronic diseases (e.g., COPD or hypertension) during their life (Lee et al., 1995). Families of higher socioeconomic status may also invest in their health and they may be more likely to seek preventive health care (Leaderer et al., 1999). Since 2009 significant reductions were observed in GDP and available income coupled to increased unemployment in Greek cities (IMF, 2012). All three are key determinants of population well-being. In the period 2012–2013 the dire economic situation of Greek households was further burdened by a heavy tax on light heating diesel,

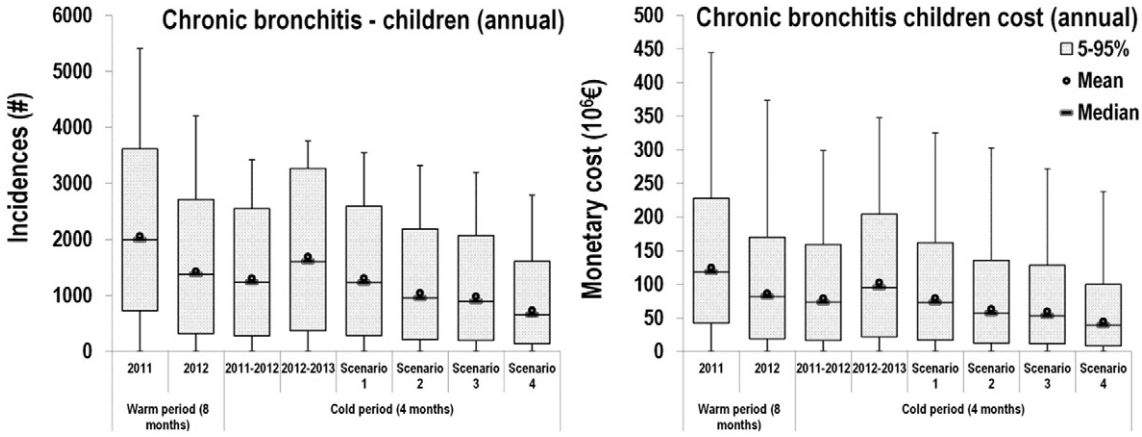

Fig. 6. Chronic bronchitis (children) new cases and related monetary cost.

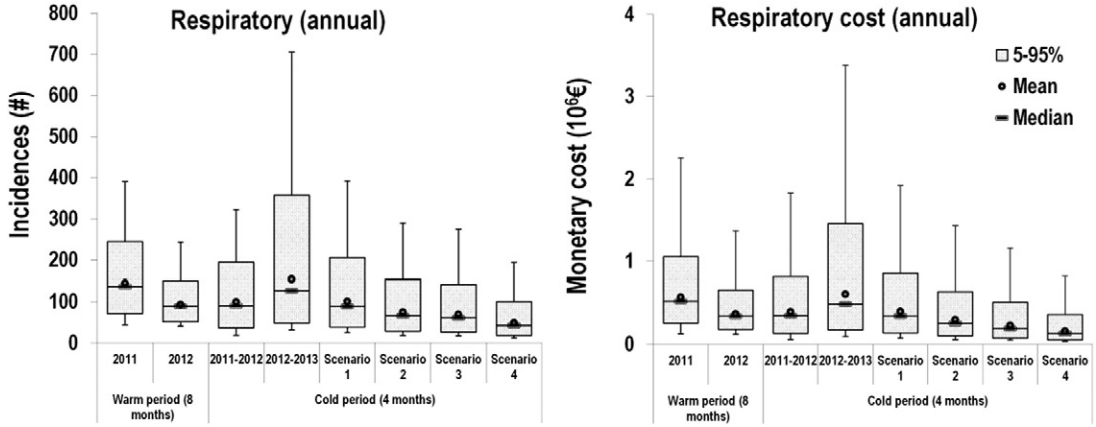

Fig. 7. Respiratory hospital admissions and related monetary cost.

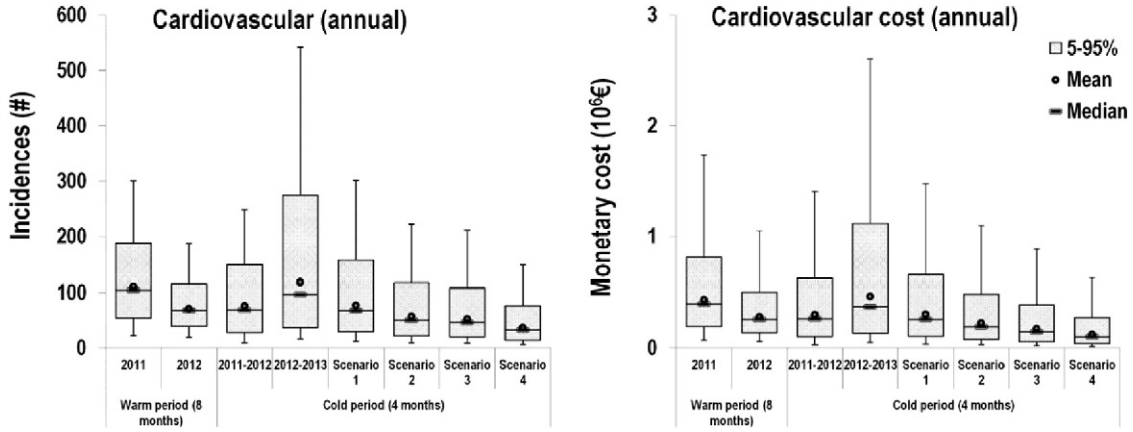

Fig. 8. Cardiovascular hospital admissions and related monetary cost.

which resulted in a market price increase of ca. 50% in 2012–2013 compared to that in 2011–2012. The compounded effect of reduced available income and the dramatically increased retail price of heating oil and – to a lesser extent – of natural gas, led many households to much cheaper fuels such as woody biomass. This, in turn, resulted in the estimated increase of overall mortality attributed to PM in the cold season of 2012–2013 compared to the one in 2011–2012.

According to the WHO, indoor smoke is ranked as the second most important environmental health risk factor, after poor water/sanitation/hygiene. Among all major policy-relevant risk factors, indoor air pollution from solid fuel is tenth globally, and fourth in less developed countries (Naeher et al., 2007). Degradation of outdoor and indoor air quality amplifies the differences between high and low-income countries. In Greece, the main culprits for the health burden attributed to exposure to airborne particles have shifted recently from traffic to biomass use for heating; this observation is consistent and can be explained by the financial figures described in Fig. 9.

The latter indicate the significant repercussions of the incumbent financial crisis on the average household income, which when combined with the simultaneous increase in the price of light heating diesel due to extra-heavy taxation led a large number of Greek households to energy poverty. The associated health effects are reckoned to be disproportionally high compared to the short-term potential financial gains from increased taxation and similar austerity measures. In practice, according to the Foundation for Economic and Industrial Research (IOBE, 2013), there were no actual financial benefits from the increased taxation of light diesel for space heating. On the contrary, the revenues from excise tax and VAT imposed on heating oil from the over-taxation were 187 million euros less than the previous year in Greece. This was due to the lower amount of heating oil taxed (2,629,931 kt in 2011–2012 compared to 1,335,083 kt in 2012–2013). Austerity measures resulted in a dramatic change in consumer behavior, with a consequent consumer shift to alternative fuels for space heating (such as illicit or unregulated biomass burning). This change resulted in both reduced tax revenues to the state and urban air quality deterioration, which would result in an increase of 40% in health burden costs between the winters of 2012–2013 and 2011–2012.

5. Conclusions

This study deals with the health and economic impact of the increased biomass use in Greece taking the example of Thessaloniki, the second largest city in the country and a major economic, industrial and commercial hub in Southeastern Europe. An integrated methodology was developed, using PM2.5 as the exposure metric to associate adverse health outcomes through the use of well established CRFs. Total exposure to PM was considered taking into account outdoor:indoor air interactions, population time activity patterns, and inhalation rate correction factors related to the intensity of the respective human activities. This was used as input to up-to-date CRFs, and health impact assessment was coupled to monetary

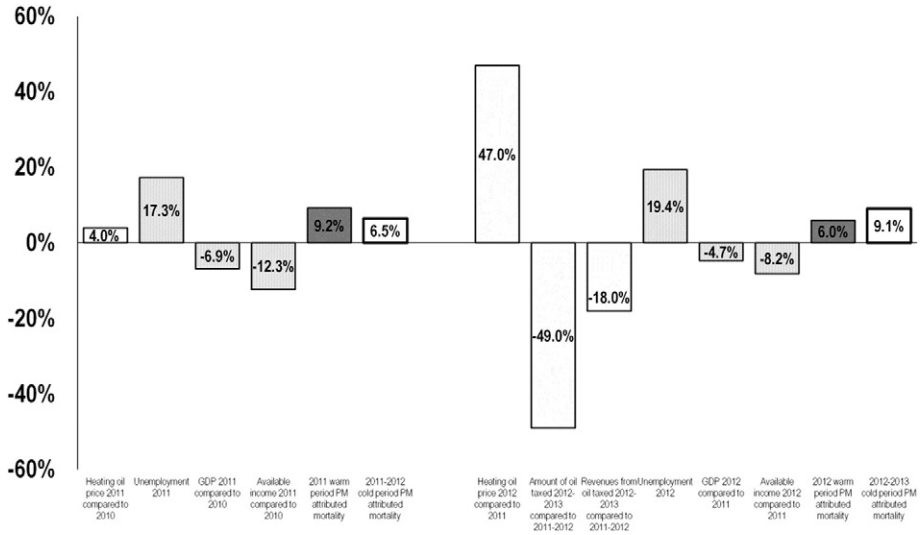

Fig. 9. Financial figures and estimated mortality for the years 2011 and 2012.

valuation functions for the endpoints of interest. Results showed an increase of about 200 excess premature deaths (out of a population of almost 900,000 inhabitants) or 3400 YLL from all causes during the 2012–2013 winter compared to the 2011–2012 winter. The additional health burden corresponds to an average cost ranging between 200–250 million euros (up to 1.2 billion). Morbidity, expressed in new cases of chronic bronchitis, cardiovascular disease and respiratory problems, corresponds to a financial cost on the order of 30 million euros. The most important adverse health impact attributed to PM exposure is chronic bronchitis in children. Considering the high overall cost the heavy taxation of light heating diesel in Greece should be reconsidered. Thus, the effect of biomass displacement was evaluated by an alternative scenario analysis, which clearly showed the benefits of policy interventions that would discourage the use of biomass as fuel source for domestic heating. Fiscal policies at the national level should take into account environmental health externalities and indeed consider how the increased public health burden would influence mid- and long-term financial turnovers.

Based on the results of the study and taking into account the importance of public health and the related socioeconomic cost, specific interventions are recommended, such as:

- Reduction of the price of light heating diesel through relaxation of the related excise tax
- Financial incentives for light heating diesel replacement by natural gas or electricity
- Enhancement of the support given to residential energy conservation schemes and simplification of the relevant regulatory framework to ease the access of larger numbers of households to such programs.

If biomass continues to be used, then the following need to be taken into consideration to reduce population exposure to fine and ultrafine particles:

- Closed fireplaces are more efficient in terms of heating performance as well PM emissions. In that case, indoor PM emissions are almost negligible.
- Emission factors for fine particles are highly dependent on fuel characteristics and burn conditions (smoldering vs. flaming). Similarly, emission factors for specific organic chemicals are influenced by fuel moisture content and burning conditions (Guillén and Ibargoitia, 1999; Khalil and Rasmussen, 2003). Thus, frequent ventilation of indoor spaces, good combustion conditions and high quality, low moisture wood would ensure relatively controlled direct exposure to the urban population.

Acknowledgments

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