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Trace metals in size-fractionated particulate matter in Portuguese hospital: exposure risks assessment and comparisons with other countries

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Abstract

Hospitals are considered as a special and important type of indoor public place where air quality has significant impacts on the potentially health outcomes. Information on indoor air quality of these environments is though limited, namely concerning to exposures to particulate matter (PM) and the related toxicity. This work aims to evaluate risks associated with inhalation exposure to ten toxic metals and chlorine (As, Ni, Cr, Cd, Pb, Mn, Se, Ba, Al, Si, Cl) in coarse ($PM_{2.5-10}$) and fine ($PM_{2.5}$) particles in a Portuguese hospital in comparison with studies representative of other countries. Samples were collected during one month in one urban hospital; elemental PM characterization was determined by proton induced x–ray emission. The non–carcinogenic and carcinogenic risks were assessed according to the methodology provided by USEPA (Region III Risk–based Concentration Table) for three different age categories of hospital personnel (adults > 20 and < 65 years) and for patients (considering nine different age groups, i.e. children with 1–3 years to seniors with > 65 years). The estimated non-carcinogenic risks due to occupational inhalation exposure to $PM_{2.5}$-bound metals ranged from 5.88×10^{-6} for Se (adults 55–64years) to 9.35×10^{-1} for As (adults 20–24 years) with total non-carcinogenic risks (sum of all metals) above the safe level for all three age categories. As and Cl (the latter due to its high abundances) were the most important contributors (approximately 90%) to non-carcinogenic risks. For $PM_{2.5-10}$ non-carcinogenic risks of all metals were acceptable to all age groups. Concerning the carcinogenic risks, for Ni and Pb they were negligible ($<1\times10^{-6}$) in both PM fractions for all age groups of hospital personnel, but potential risks were observed for As and Cr that with values in $PM_{2.5}$ exceeding (up to 62 and 5 times, respectively) USEPA guideline across all age groups; for $PM_{2.5-10}$ increased excess risks of As and Cr were observed particularly for the long-term exposures (adults with 55-64 year). Total carcinogenic risks highly (up to 67 times) exceeded the recommended level for all age

groups, thus clearly showing that occupational exposure to metals in fine particles poses significant risks. If the extensive working hours of hospital medical staff were considered, the respective non- and carcinogenic risks were increased, the latter for $PM_{2.5}$ exceeding the USEPA cumulative guideline of 10^{-4}. For adult patients the estimated non-carcinogenic and carcinogenic risks were approximately three times higher than for personnel, with particular concerns observed for children and adolescents.

Keywords: indoor air; PM; risks; metals; hospital; carcinogens

1. Introduction

There is growing public awareness regarding the health risks associated with poor indoor air quality (Huboyo et al. 2011; Hulin et al. 2012; WHO 2010). Because people spend majority of their time indoors (Klepeis et al. 2001), they are at greater risks of adverse health effects from chronic exposures to indoor air pollutants (Bernstein et al. 2008). Among those concerns is exposure to inhalable particulate matter (PM). In recent years, the scientific attention has focused mostly on fine fraction of particles (<2.5 μm in diameter; i.e. $PM_{2.5}$; Brunekreef et al. 2009; Hoek et al. 2013; Li et al. 2013; Polichetti et al. 2009) that has been linked to both acute and chronic respiratory and cardiopulmonary health effects including lung cancer (Mitchel et al. 2007; Valavanidis et al. 2008). Additional focus has been placed on determining associations with PM components (elemental carbon, sulfates, nitrates, polycyclic aromatic hydrocarbons, biological components; Atkinson et al. 2010; Brunekreef et al. 2009; Gent et al. 2009; Nemmar et al. 2013; Maynard et al. 2007; Sarnat et al. 2008). Although the precise mechanisms of PM health effects are not completely understood (Oeder et al. 2012), the evidence has shown significant associations between PM properties (chemical and biological components, particle surface area and reactivity) and its toxicity (Kelly and Fussel 2012; Nemmar et al. 2013). Therefore, PM chemical components may have high potential to contribute to PM-induced health effects (Schwarze et al. 2006) even though they compose only a small fraction of PM mass (Slezakova et al. 2007, 2009). Most studies of PM have focused on ambient (outdoor) exposures. The contribution and significance of indoor PM, which may differ substantially in composition from outdoor particulates, have yet to be fully explored.

PM composition is very complex. Previously, the risks of toxic compounds such PAHs in indoor and outdoor PM have been shown (Castro et al 2011; Slezakova at al.

2011a). Due to their toxic characters, trace metals are an important component of PM (Senlin et al. 2008). Some airborne trace metals may derive from natural crustal source, but the majority results from anthropogenic activities (Okuda et al. 2008) with the main sources including (Fang et al. 2010; Susaya et al. 2010): vehicle emissions (primary source for chromium, lead cadmium, and barium), industrial and construction processes (responsible for manganese, aluminum, silicon), oil (responsible for nickel) and coal combustions (chromium), and metal industry (metal-specific). In indoor environments the abundances of the trace elements result from infiltration of outdoor emissions (Habil et al. 2013; Hassan 2012) and from various indoor sources which include different wall-paints and indoor equipment and utensils (Chattopadhyay et al. 2003; Kebede et al. 2013; Paoletti et al. 2006; Taner et al. 2013). Most trace metals exist in the solid phase and thus occur almost exclusively in the particle phase of the atmosphere, where they are ubiquitous in both fine and coarse fractions (Hu et al. 2012; Singh et al. 2011). For the health risks assessment, the size distributions of atmospheric trace metals and other elements is significant (Kelly and Fussel 2012). Whereas metals from crustal sources tend to accumulate in coarse mode of particles (i.e, those larger than approximately 1-3 μm; (Lü et al. 2012; Slezakova et al. 2008), the more toxic metals from anthropogenic sources are predominantly found in the fine fraction of atmospheric particles (Chen and Lippmann 2009; Song and Gao 2011; Greene and Morris 2006). In small quantities they might be harmless, but many of the trace metals (and metallic compounds) are harmful to the humans (WHO 2007). According to the International Agency for Research on Cancer (IARC) arsenic, cadmium, and hexavalent chromium and nickel compounds are classified as carcinogenic to humans (IARC 2012) whereas inorganic lead compounds are classified probable carcinogens (IARC 2006). Accumulation in fatty tissues and circulatory system, negative effects on central nervous system and functioning of internal organs as well as acting as cofactors in other diseases

and cancer are some of the negative health effects associated with exposure to these metals (Chen and Lippmann 2009; Kurt-Karakus 2012). Therefore, in order to protect public health European Union (Directive 2004/107/EC) settled limits of atmospheric metals considering three carcinogenic metals (arsenic, nickel and cadmium) in ambient air. As these elements represent hazard to human health careful monitoring should be considered. Furthermore, the investigation of the health risks associated with airborne metals may provide useful information regarding environmental risks of indoor environments.

Hospitals are considered as a special and important type of indoor public place (Banse 2013; Barnett and Barnett 2003) where poor air quality can affect not only the health of the employees but also of patients (due to suppressed immune system they are more susceptible to external influences). Assessment of risks to these occupants resulting from exposure to airborne particulates includes measurements of PM concentration levels and their related toxicity in terms of trace metals (or ions). However, the information concerning PM levels in hospitals is limited (and in Portugal non-existent). Additionally, there is a lack of knowledge on PM trace metals in these environments (Brown et al. 2012; Wang et al. 2006a) and the associated health impacts. Considering the importance of hospital to public health, further studies are necessary in order to fully assess the risks of particulate exposures and the related toxicity in the respective environments. The aim of this study was to estimate the risks associated with exposure to particulate-bound trace metals in hospital environment. Hospital staff and patients were the considered exposed groups. The concentrations of trace elements, namely aluminum (Al), silicon (Si), chlorine (Cl), manganese (Mn), selenium (Se), barium (Ba), arsenic (As), lead (Pb), cadmium (Cd), chromium (Cr), and nickel (Ni) collected in Portuguese hospital were determined in indoor coarse ($PM_{2.5-10}$) and fine ($PM_{2.5}$) particles. The specific objectives of this work were: i) to

evaluate non-carcinogenic risks associated with inhalation exposure to eleven potentially toxic trace elements in $PM_{2.5-10}$ and $PM_{2.5}$; ii) to evaluate carcinogenic risks from inhalation exposure to fine particles and to $PM_{2.5-10}$ and $PM_{2.5}$-bound metals; and iii) to assess and compare PM indoor air quality in a Portuguese urban hospital with studies representative of other countries and existing guidelines.

2. Materials and methods

2.1 Sample collection

Particulates were collected for a period of four weeks in the hospital of Vila Nova de Gaia which belongs to the Metropolitan Area of Oporto (NW region of Portugal), and corresponds to the second largest municipality in Portugal. The hospital complex is surrounded by national roads and is located in the direct vicinity of the busiest highway in Portugal that connects north and south of the country (Fig. 1); the highway is also the main road connection to the north of Spain. Consequently, emissions from road traffic are the main source of atmospheric pollutants in the respective area. Samples were collected daily for a period of 24 hours (7:30 a.m. to 7:30 a.m. next day) by constant flow samplers (Bravo H2, TCR TECORA, Italy) that were combined with PM EN LVS sampling heads (in compliance with norm EN12341 for PM_{10} and EN14907 for $PM2_{.5}$); an air flow rate of 2.3 $m3\ h^{-1}$ was used. The sampling apparatuses were positioned inside a main corridor of the radiology ward that was designated to both children (older than 1 year) and adult patients. Inlets were placed 1.6 m above the floor (in order to simulate human breathing zone) and minimally 1 m from the walls, without obstructing the normal usage of the rooms. PM masses were collected on polytetrafluoroethylene (PTFE) membrane filters with polymethylpentene support ring (2 μm pore size, Ø47 mm, SKC Ltd, UK). During the

monitoring period a detailed record was kept on the activities in the area surrounding the sample collection. Concerning PM indoor sources, no significant differences were observed between the activities performed by the personnel; smoking was prohibited in all areas of the hospital.

2.2 PM mass determination

PM_{10} and $PM_{2.5}$ masses were determined gravimetrically as described previously in detail in Slezakova et al. (2009). Briefly, the initial mean mass of the blank filter was subtracted from the final mean mass of the exposed filter; the difference was then divided by the total volume of air that passed through filter (at 25 °C and 101.3 kPa). $PM_{2.5–10}$ fraction (i.e. coarse fraction with particles of aerodynamic diameter between 2.5 and 10 μm) was determined as difference (by subtraction) between PM_{10} and $PM_{2.5}$.

2.3 Elemental characterization

Elemental characterization of PM_{10} and $PM_{2.5}$ was performed by Proton Induced X–ray Emission (PIXE), which provided analysis for elements from magnesium trough uranium. For the elemental analysis PTFE filters were cut in half. One half of the filter was analyzed whereas the other part was kept for possible replicates and other analysis. PIXE analyses were carried out at a Van de Graaff accelerator, in vacuum. For each of the samples two X–ray spectrum were taken; one with a 1.2 MeV proton beam and no absorber in front of the Si(Li) detector for low energy X–ray elements, and another with a 2.25 MeV proton beam and a 250 mm Mylar® filter to detect elements with atomic number higher than 20. The beam area at the target was 20 mm^2. Spectra deconvolution was performed with the AXIL computer code V3.0 and quantitative analysis was carried out with the DATTPIXE package (Almeida et al. 2003; Freitas et al. 2003).

2.4 Health risk analysis

The non–carcinogenic and carcinogenic risks were assessed according to the methodology provided by USEPA Region III Risk–based Concentration Table (USEPA 2013a). The non–carcinogenic risks of each individual metal were assessed by the non–cancer hazard quotient (THQ) (USEPA 1989): ‘‘the ratio of a single substance exposure level over a specified time period (e.g, subchronic) to a reference dose (RfD) for that substance derived from a similar exposure period’’. THQ assumes that there is a level of exposure (i.e, RfD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the exposure level (E) exceeds this threshold (i.e, if E/RfD exceeds unity), there may be concern for potential non–cancer effects (USEPA 1989); higher values of THQ (above unity) indicate the greater levels of concern.

The carcinogenic risks were assessed as the incremental probability of an individual to develop cancer, over a lifetime, as a result of exposure to that potential carcinogen (i.e. incremental or excess individual lifetime cancer risk; USEPA 1989). Acceptable risk levels for carcinogens range from 10^{-4} (risk of developing cancer over a human lifetime is 1 in 10 000) to 10^{-6} (risk of developing cancer over a human lifetime is 1 in 1 000 000).

The following equations were used to calculate non–carcinogenic and carcinogenic risks associated with inhalation exposure to trace elements in indoor environment:

$$THQ = (EFr \times ED \times ET \times C \times IR) / (RfD \times BW \times AT) \quad (1)$$

$$TR = (EFr \times ED \times ET \times C \times IUR) / AT \quad (2)$$

where THQ and TR (target carcinogenic risk) are dimensionless; EFr is the exposure frequency (250 days per year; USEPA 2013a); ED is the exposure duration (years); ET is exposure time (8 h per day for hospital personnel and 24 h per day for patients; USEPA 2013a); C is the concentration of metal in air ($\mu g\ m^{-3}$); IR is the inhalation rate ($m^3\ day^{-1}$);

BW is body weight (kg); AT is the number of days over which the exposure is averaged (365 days per year × ED for non–carcinogenic effects and 25,500 days, i.e. 70 years × 365 days per year for carcinogenic effects; USEPA 2013a); RfD is the inhalation reference dose (mg $kg^{-1}day^{-1}$); and IUR is the chronic inhalation unit risk $(\mu g\ m^{-3})^{-1}$; USEPA 2013a). Since RfD values are only available for oral exposure (USEPA 2013a), the RfD values were converted from existent USEPA reference concentrations for inhalation exposure according to USEPA (2013b):

$$RfD = (RfC \times IR_A \times AR) / (BW_A \times 100) \quad (3)$$

where RfC is reference concentration (mg m^{-3}); IR_A and BW_A are inhalation rate and body weight of an adult (20 $m^3\ day^{-1}$ and 70 kg; USEPA 2013b); and AR is absorption rate (100%; USEPA 2013b). The converted RfD values are presented in Table 1. Non–carcinogenic risks were estimated for nine trace elements for which RfC values (in brackets) are available (USEPA 2013a): aluminium (5×10^{-3} mg m^{-3}), silicon (3×10^{-3} mg m^{-3}), chlorine (1.5×10^{-4} mg m^{-3}), manganese (5×10^{-5} mg m^{-3}), selenium (2×10^{-2} mg m^{-3}), barium (5×10^{-4} mg m^{-3}), hexavalent chromium – Cr (VI) (1×10^{-4} mg m^{-3}), nickel – refinery dust (5×10^{-5} mg m^{-3}), and arsenic – inorganic (1.5×10^{-5} mg m^{-3}). Similarly when available, the IUR values were retrieved for four carcinogenic elements (possible probable) as the following (USEPA 2013a): nickel (refinery dust; 4.8×10^{-4} $(\mu g\ m^{-3})^{-1}$), arsenic (inorganic; 4.3×10^{-3} $(\mu g\ m^{-3})^{-1}$), lead (acetate; 1.2×10^{-5} $(\mu g\ m^{-3})^{-1}$), and hexavalent chromium (8.4×10^{-2} $(\mu g\ m^{-3})^{-1}$). In this work hospital staff and patients were the considered exposed populations. Hospital staff was represented only by adults (i.e. older than 20 years and < 65 years). Three different age–categories of adults were considered, namely with 20–24 years, 25–54 years and 55–64 years (USEPA 2011). Nine different age–categories ranging from children of 1 year to seniors >65 years of patients were used for the estimation of the target risks (Vieira et al. 2011; USEPA 2011) with the following ED values (in brackets): children

1–3 years (1 year), children 4–6 years (4 years), children 7–10 years (7 years), adolescents 11–14 years (11 years), adolescents 15–19 years (15 years), adults 20–24 years (20 years), adults 25–54 years (25 years), adults 55–64 years (55 years), and seniors >65 years (65 years) (USEPA 2011). Body weights and inhalation rates for the respective age categories were adapted from USEPA (2011) as the following: children 1–3 years (14 kg; 8.5 m^3 day^{-1}), children 4–6 years (21 kg; 10.1 m^3 day^{-1}), children 7–10 years (32 kg; 12.0 m^3 day^{-1}), adolescents 11–14 years (51 kg; 15.2 m^3 day^{-1}), adolescents 15–19 years (67 kg; 16.3 m^3 day^{-1}), adults 20–24 years (72 kg; 15.7 m^3 day^{-1}), adults 25–54 years (77 kg; 15.9 m^3 day^{-1}), adults 55–64 years (77 kg; 14.9 m^3 day^{-1}), and seniors >65 years (72 kg; 13.4 m^3 day^{-1}).

2.5 Statistical analysis

For the data treatment, the Student's t–test was applied to determine the statistical significance (p<0.05, two tailed) of the differences between the determined means.

3. Results and discussion

3.1 PM concentrations

In the studied hospital, 24–h PM_{10} concentrations ranged between 13 to 59 µg m^{-3} with median value of 38 µg m^{-3}. On average 77% of indoor PM_{10} was composed by $PM_{2.5}$ (range of 11 to 42 µg m^{-3}; median of 30 µg m^{-3}); coarse (i.e. $PM_{2.5-10}$) particles ranged between 2.5 and 22 µg m^{-3} (median of 6 µg m^{-3}) and they accounted for 23% of indoor PM. Furthermore, the statistical analysis of the results indicated that $PM_{2.5-10}$ mean (7.4 ± 4.1 µg m^{-3}) was significantly lower (p<0.05) than $PM_{2.5}$ (23 ± 10 µg m^{-3}). Overall obtained PM_{10} and $PM_{2.5}$ were in similar ranges as in non-smoking residences (Minguillón et al. 2012; Slezakova et al. 2009, 2011b) but lower (approximately 3-10 times) than in public places (restaurants, supermarkets, and commercials offices) or schools (Dong et al. 2013; Habil et

al. 2013; Taner at al. 2013). All existent studies dedicated to PM in hospitals are summarized in Table 2. Concerning Europe, available information on PM in hospitals exists only for fine fraction (Fernandéz et al. 2009; Nardini et al. 2004; Sureda et al. 2010). $PM_{2.5}$ levels obtained in the Portuguese hospital were significantly higher (p<0.05) than those found in other European countries. All European studies referred in Table 2 were performed in order to assess environmental tobacco smoke (ETS); $PM_{2.5}$ was used as its marker. Therefore, different organization of these studies, very different sampling protocols with limited period of sample collections may account for some of the observed differences in PM levels. More information on both PM_{10} and $PM_{2.5}$ comes from Asian countries (Table 2). Two studies performed in Taiwan reported similar concentration ranges of PM_{10} (Wan et al. 2011) and $PM_{2.5}$ (Hsu et al. 2012) to those in Portugal. In India and China, observed $PM_{2.5}$ and PM_{10} in hospital environments were much higher than in Portugal (3 to 4 times; Verma and Taneja 2011; Wang et al. 2006a, 2006b). These findings are though not so surprising considering the typically much higher levels of ambient air pollution in Asian countries. Despite the higher levels, Wang et al. (2006a, 2006b) who investigated PM levels in four different Chinese urban hospitals, reported mean $PM_{2.5}/PM_{10}$ ratio of 0.78; a similar mean of 0.77 was observed in this study. Fine particles thus constituted a major fraction of PM_{10} in the studied hospital. These findings are health-relevant because especially $PM_{2.5}$ represents a serious risk to human health; when inhaled these particles may reach the peripheral regions of the bronchioles, and interfere with gas exchange inside the lungs (WHO 2000). Nevertheless, current Portuguese legislation for indoor air quality (Decreto Lei 79/2006) provides limits only for PM_{10} fraction (defined as maximal indoor concentration of 150 $\mu g\ m^{-3}$). In order to protect public health, regulatory aspects of air in relation to indoor $PM_{2.5}$ need to be addressed. Some experts recommend indoor levels be maintained at 50% or less than air quality standards established by US EPA for outdoor air

(Bernstein et al. 2008). However, PM does not have any threshold below which no health damage is observed. In order to minimize the health effects WHO thus recommended guidelines (in ambient air) that represent an acceptable and achievable levels of air pollution (WHO 2006). Concerning $PM_{2.5}$, WHO advises that mean concentration should not exceed 25 μg m^{-3} and 10 μg m^{-3} within period of 24 h and calendar year, respectively. As these guidelines are set for ambient air, they cannot be applied to indoor environments directly; on average people spend 75 to 90% of their time indoors whereas it is only 10–25% outdoors. In the studied hospital more than 50% of $PM_{2.5}$ measured concentrations surpassed the 24-h guideline for ambient air which indicates the potential health risks of the exposed individuals. In addition, the indoor particles can act as carrier for infectious microbes and microbial metabolites that may accumulate in the hospital environments (Hsu et al. 2012), thus representing additional health risks (i.e. transmissions of airborne infectious diseases; Eames et al. 2009; Tang et al. 2011; Hsu et al. 2012).

3.2 Elemental composition

Twenty one elements were determined by PIXE in indoor PM (Slezakova et al. 2012). Out of these, eleven elements were considered for health risk assessment: Al, Si, Cl, Mn, Se, Ba, Cr, Ni, As, Cd and Pb. Table 3 provides summary (means, ranges) of these 11 elements in $PM_{2.5}$ and $PM_{2.5-10}$. The total concentration of eleven elements (i.e. ΣE_{11}) in air ranged between 271 and 1030 ng m^{-3} for $PM_{2.5}$ (mean of 759 ng m^{-3}) and between 134 and 793 ng m^{-3} for $PM_{2.5-10}$ (mean of 349 ng m^{-3}); ΣE_{11} comprised 26 and 25% of the elemental content in $PM_{2.5}$ and $PM_{2.5-10}$, respectively. Indoor elemental concentrations were compared with those from outdoor air from previous studies of the same team (Slezakova et al. 2012, 2007) in the selected area. Overall, the outdoor mean ΣE_{11} ranged between 1875 and 2350 ng m^{-3}

for $PM_{2.5}$ and from 2570 to 2620 for $PM_{2.5-10}$. The respective levels observed in the hospital were approximately 2-3 times lower for $PM_{2.5}$ and 7-8 times for $PM_{2.5-10}$.

Only few studies on PM elemental composition in hospital environments exist. From the available studies that are summarized in Table 2, only two of them (Brown et al. 2012; Wang et al. 2006a) presented results concerning PM composition. In Atlanta (USA) Brown et al. (2012) analyzed elemental compositions of $PM_{2.5}$ in various microenvironments including one hospital. However, only limited results are presented for the hospital and with all data presented in plots. Thus the available information comes mainly from the study in Guangzhou, China (Wang et al. (2006a) where elemental concentrations ranged from 3400 to 5500 ng m^{-3} in $PM_{2.5}$ and from 6280 to 10 280 ng m^{-3} in PM_{10}. These levels were approximately twice higher than in the present study (Table 3), which is expected considering the higher pollution levels in Asian countries in general.

The compositional profiles of these elements were similar for both PM fractions. Out of eleven considered elements, Cl, Si, and Al were the most dominant ones in both PM. These three elements accounted, respectively, for 85 and 90% of ΣE_{11} in $PM_{2.5}$ and $PM_{2.5-10}$. Specifically, Cl was the most abundant in $PM_{2.5}$ (53 % of ΣE_{11}) being followed by Si (19% of ΣE_{11}) and Al (13%) whereas in coarse fraction Si accounted for the majority of ΣE_{11} (50%), and Cl and Al contributed 30 and 13% of ΣE_{11}. The presence of Cl in indoor environments might result from cleaning works and use of cleaning products and disinfectants (Sulaiman et al. 2005), which are abundantly utilized in hospital environments. Dust released from building material can be also potential source of indoor Cl (Abdel Hameed et al. 2004). Considering that studied hospital is situated in a coastal area, indoor chloride may result from penetration of outdoor sea salt sprays particles (Slezakova et al. 2011b). Silicon and Al often result from crustal sources. The subsoil of this region consists of granite that is rich in Al and Si (Begonha 2001); granite is a common affordable material

frequently also used indoors. Thus presence of these elements in indoor environments might be due to the erosion of building materials or from penetration of outdoor particles to indoor ambiences (by air ventilation, low–quality building isolation, and etc).

The abundances of the other elements were for both PM much lower: Ba (2 and 5% of ΣE_{11} in $PM_{2.5}$ and $PM_{2.5-10}$, respectively), Mn (0.4 and 0.2% of ΣE_{11} in $PM_{2.5}$ and $PM_{2.5-10}$, respectively), and Se (0.1% in $PM_{2.5}$ and 0.2% in $PM_{2.5-10}$). Concerning the carcinogenic elements, this study included three known carcinogens (USEPA group A) namely As, Cr and Ni, and Pb that is considered as probable carcinogen based on animal studies (USEPA Group B2). Total mean concentration of carcinogens (ΣE_{carc}) was 96.8 and 4.6 ng m^{-3} in $PM_{2.5}$ and $PM_{2.5-10}$, respectively, thus representing 13 and 1.3% of ΣE_{11}. Specifically, the abundances of Pb, Ni and Cr were low in both PM: Pb (1% and 0.2% of ΣE_{11} in $PM_{2.5}$ and $PM_{2.5-10}$, respectively), Ni and Cr (less than 1 and less than 0.2%, respectively, in $PM_{2.5}$ and $PM_{2.5-10}$); the concentrations of these three carcinogens were at Portuguese hospital much lower than in the study of Wang et al. (2006a): 15-30 times for Cr, 7-13 times for Ni, and 20-30 times for Pb. On the contrary As comprised most of the carcinogenic content in both PM (83% and 60% of ΣE_{carc} in $PM_{2.5}$ and $PM_{2.5-10}$, respectively, i.e. 11 and 0.8% of ΣE_{11}) and its levels ($PM_{2.5}$ mean of 80.3 ng m^{-3}) were approximately twice higher than in the Chinese study (Wang et al. 2006a). No specific indoor source of As was identified in the hospital. In general, As is not typical indoor pollutant but it can be found in indoor places with smoking (Slezakova et al. 2009); environmental tobacco smoke is its major indoor emission source. However, smoking was prohibited in all areas of the studied hospital. Considering also the predominant abundance of As in fine particles (97%), contribution of anthropogenic outdoor emissions could account for indoor As. At this moment there are no guidelines for concentrations of indoor PM-bound metals. The carcinogenic elements are though considered in the European directive (Directive 2004/107/EC) which settles targets

for As, Cd, and Ni in ambient air. The targets are expressed as annual means in PM_{10}, with values of 6, 5 and 20 ng m^{-3} for As, Cd, and Ni, respectively. Mean concentration of As in PM_{10} in hospital (83.0 ng m^{-3}, i.e. sum of $PM_{2.5}$ and $PM_{2.5\text{-}10}$) was 14 times higher than the target value for ambient air. Finally, Cd (also considered as class A carcinogen) was absent in both PM in the studied hospital; this element was the least abundant in study by Wang et al (2006a) with concentration ranges between 6 and 13 ng m^{-3}.

3.3 PM health risks

The non-carcinogenic risks associated with inhalation exposure to particulate trace elements were calculated for three different age groups of hospital staff according to USEPA methodology. The means and the ranges of THQ calculated for individual elements in different PM fractions and for various age groups of hospital staff are presented in Table 4. The estimated mean THQ for $PM_{2.5\text{-}10}$–bound trace elements ranged from 1.07×10^{-6} for Se (adults 55–64 years) to 1.21×10^{-1} for Cl (adults 20–24 years). These results show that mean THQ of all nine elements in $PM_{2.5\text{-}10}$, as well as, total THQ (i.e. sum of individual THQ) were below the unity (THQ < 1; Table 4) for all age categories of hospital staff. Therefore, non-carcinogenic risks from exposure to trace elements in coarse fraction were acceptable to all age groups of hospital staff. For $PM_{2.5}$, significantly higher (p<0.05) non-carcinogenic risks were observed with corresponding values ranging from 5.88×10^{-6} for Se (adults 55–64 years) to 9.35×10^{-1} for As (adults 20–24 years). As and Cl (the latter due to its high abundance) were the most important contributors (approximately 90%) to non-carcinogenic risks. The contributions to THQ of other elements were significantly lower: Ni > Mn > Si >Al > Ba > Cr > Se. For all these elements, individual THQ were below the unity (THQ < 1) across all age groups. The total THQ in fine particles (Table 4) though exceeded safe

level for all three age groups of hospital staff (with the greatest values, i.e. concerns, observed for younger populations), particularly due to the high contributions of As and Cl.

The carcinogenic risks (means, ranges) of hospital staff associated with the exposure to PM–bound four carcinogenic elements are presented in Table 5. The obtained results demonstrate that: i) higher risks were found for metals in $PM_{2.5}$ than $PM_{2.5-10}$; ii) for all carcinogens the highest carcinogenic risks were observed for the age group of adults with 55–64 years); and iii) for all age–groups the highest risks were found for arsenic. Considering the above mentioned, the highest cancer risks were thus observed for arsenic in $PM_{2.5}$ which reached for adults with 55–64 years a value of 6.19×10^{-5}. For the carcinogenic risks, USEPA considers that setting a 10^{-6} risk level for individual chemicals and pathways will generally lead to negligible cancer risks. However, caution is recommended to ensure that the cumulative cancer risk for all potential carcinogenic contaminants does not have a residual cancer risk exceeding (10^{-4}) (USEPA 2013a). As previously mentioned, the highest carcinogenic risks were observed for As (Table 5). In $PM_{2.5}$, TR of As exceeded the USEPA guideline of 10^{-6} for all age categories of hospital staff (Table 5). As was the most threatening carcinogenic metal, primarily due to its high PM content. The minimum As TR value (23 times higher than 10^{-6}) corresponded to adults (20–24 years) and maximum (62 times higher) to adults with 55–64 years, mainly due to their lifetime exposure length. Concerning the coarse particles, the As cancer risks were significantly lower. Excess risks were observed for adults 55-64 years with As TR approximately twice higher (than 10^{-6}). Cr was the second leading contributor to carcinogenic risks of hospital staff mostly due to its high value of inhalation unit risk. The inhalation unit risk of Cr (VI) is based on an assumed 1:6 ratio of Cr (III):Cr (VI) (USEPA 2013b). The concentration of Cr determined in this study was total Cr. Therefore, one seventh of the total Cr (i.e. determined) concentration was used for the health risk assessment. In $PM_{2.5}$ TR of Cr surpassed (2-5

times) the USEPA guideline for all three age categories of hospital staff. Although Cr risks in coarse fraction were mostly negligible, one age category, namely adults with 55-64 years still exhibited TR values slightly higher than recommended. Regarding Ni and Pb, the respective risks were inferior to those of As and Cr. Evaluating all age categories of hospital staff, in $PM_{2.5}$ TR for Ni and Pb were 4-11 times and 41-113 times lower than threshold of 10^{-6}, respectively, whereas it was 23-63 times and 662-1820 times for Ni and Pb in $PM_{2.5\text{-}10}$. Therefore, the carcinogenic risks resulting from occupational exposure to these two elements were negligible for all age categories. The total carcinogenic risks from occupational exposure to metals (i.e. sum of the individual TR) were also assessed for both PM fractions (Table 5). The results shows that total cancer risks of both PM fractions were higher than the USEPA recommended level of 10^{-6} for all age groups of hospital staff. Specifically, TR values of $PM_{2.5}$ were high (24 to 67 times than acceptable). In addition, Table 5 also presents the carcinogenic risks calculated for inhalation exposure to indoor $PM_{2.5}$ concentrations. For all three age groups the carcinogenic risks from exposure to $PM_{2.5}$ exceeded USEPA cumulative threshold risk of 10^{-4} (risk of developing cancer over a human lifetime is 1 in 10 000), indicating adverse health outcomes across all age groups. The respective TR ranged from 1.22×10^{-2} for adults (20–24 years), being the highest for adults with 55-64 years (3.36×10^{-2}). These results imply that 336 employees (55-64 years old) in 10 000 may have lung cancer due to the exposure to $PM_{2.5}$ alone. The estimated risks though might be even higher dues to the synergistic effects between particulate matter and trace elements (Oeder et al. 2012).

The health risks analysis of this work was based on USEPA recommendation for workers (USEPA 2013a**),** with exposure frequency of 250 days per year (corresponds to 5 days per 50 weeks) and exposure time of 8 h per day (i.e. 40 h per week). Medical professionals though often experience increased workloads (Cole et al. 2009) and long

working hours, in some specializations (general surgery, anesthesiology) up to 60 h per week (Dorsey et al. 2003). Thus, the respective inhalation risks might be higher than those here estimated. Specifically for personnel with radiology specializations, the authors reported average of 58 h per week (corresponds to 11.6 h per day; Dorsey et al. 2003). When ET of 11.6 h is considered, the re-estimated total THQ are approximately 1.5 times higher (1.85-2.09 and 0.22-0.25 for $PM_{2.5}$ and $PM_{2.5-10}$, respectively; Fig. 2a). Similarly higher values for carcinogenic risks were obtained with re-estimated total $PM_{2.5}$ TR between 3.52×10^{-5} and 9.69×10^{-5} for adults with 20–24 and 55-64 years; respectively; the corresponding TR in $PM_{2.5-10}$ ranges from 1.84×10^{-6} to 5.06×10^{-6} (Fig. 2b). In essence the major conclusions of the re-evaluated health risk analysis were the same, showing excess risks, both non- and carcinogenic ones, for $PM_{2.5}$. It is also noteworthy that prolonged working hours caused excess cancer risks (TR 2-5 times higher than 10^{-6}) of coarse fraction across all age categories of hospital staff. Typically, the scientific attention is focused on fine particles. These results though demonstrate that impacts of $PM_{2.5-10}$ should not be omitted especially when prolonged exposures might occur. Although coarse particles deposit in upper parts of respiratory system, they can cause additional risks. In that regard it is necessary to consider that on a daily basis, hospital staff is exposed to metals of both PM fractions. The cancer risks resulting from PM combined exposure (i.e. sum of TR of both PM) exceeded the cumulative threshold of 10^{-4} for adults with 55-64 years. However, the respective risks could be eventually even higher if combined with alternative factors (lifestyle, smoking, diet or additional outdoor exposure). When longer ET of 11.6 h was considered, As THQ ranged from 1.20 to 1.36 in $PM_{2.5}$ and between 4.07×10^{-2} and 4.60×10^{-2} in $PM_{2.5-10}$; carcinogenic risks were 3.26×10^{-5}–8.98×10^{-5} in $PM_{2.5}$ and 1.10×10^{-6}–3.05×10^{-6} in $PM_{2.5-10}$. In $PM_{2.5}$ As THQ and TR were of particular concern as they exceeded both unity and USEPA threshold, respectively, across all age categories.

Finally, the non-carcinogenic and carcinogenic risks were also estimated for nine different age–categories of patients (ET of 24 h). The results are shown in Table 4 and 5, respectively. Overall THQ and TR values of adult patients were approximately three times higher than for hospital staff, mostly due to the longer exposure. Particular concerns were observed for children (ΣTHQ 7.40–12.0 in $PM_{2.5}$; 1.15–1.45 in $PM_{2.5–10}$). These findings are relevant because young children have lower tolerance to toxins (Acosta et al. 2009). In addition, due to their behavior (hand-to-mouth activities, touching and mouthing of various dust-contaminated objects; Beamer et al. 2009) children exposure to metals might be even higher (indirectly by indigestion) which could result in increased risks than here estimated. Total carcinogenic risks from inhalation exposure to metals (i.e. ΣTR) exceeded in $PM_{2.5}$ USEPA guideline of 10^{-6} across all age categories of patients, with TR values ranging from 4 (children 1-3 years) to 240 (seniors) times higher than acceptable (Table 5). These results indicate that inhalation exposure to metals in fine particles at the levels observed in hospital might eventually lead to adverse health outcomes (i.e. lung cancer morbidity and mortality) for all age groups (Valavanidis et al. 2008). Finally, as demonstrated in the Figures 3a-b, the additive non- and carcinogenic risks of metals in both PM fractions (i.e. $PM_{2.5}$ + $PM_{2.5–10}$) exceeded for all age categories of patients the USEPA safe levels (THQ>1; TR>10^{-6}). Additive carcinogenic risks from long-term exposures (adults 55-64 years and seniors) were of particular concern as they resulted in TR values that exceeded USEPA cumulative threshold of 10^{-4}. In some cases, due to suppressed immune system, patients may be more susceptible to external influences, so the respective risks for the patients can be higher than here estimated. Studies have shown that hospital patients can acquire microbial contaminants (bacteria, fungi and viruses) from personnel and from indoor environment (Napoli et al. 2012). Although the extent to which the latter contributes towards hospital-

acquired-infection is largely unknown (Talon 1999), the environmental matrices such as air and surfaces can act as reservoirs of microbial contaminants. The risks due to the biological component have not been considered in this study. However, suspended particles are particularly important in that regard because they can carry and transport microorganisms, secondary allergens or proinflammatory compounds (Balaras et al. 2007). There is increasing evidence that PM biological components play central role in biological effects. When PM is inhaled, the biological components are responsible for stimulating alveolar macrophages and respiratory epithelial tissue to release proinflammatory cytokines and chemokines (Nemmar et al 2013). Even when hospital environment is well within the recommended limits of microbiological air quality, the number of particles may be high (and consequently foster the growth of microorganisms; Dascalaki et al. 2008). In order to reduce bacteria, viruses and particle concentrations in hospital, proper air ventilation and its maintenance are mandatory so safe and healthy air environment can be obtained. Particular attention needs to be given to cleaning and prevention of microbial growth indoors (Dancer 2004).

In addition when assessing human risks, metal speciation is of major importance. This might be especially relevant for As and Cr that were the major contributors to TR risks of the exposed populations in the hospital. Arsenic has a complex chemical structure and can be found in inorganic (trivalent; and pentavalent) or organic forms. Whereas the inorganic As is considered by USEPA as class 1 carcinogen, its alkylated forms can be less toxic (Morais et al. 2012). In this study the content of the particulate-bound elements were determined considering the total concentration (only). Concerning Cr its toxicity also depends on the chemical form and subsequent bioavailability (Michalski 2009). Cr (III) are compounds essential to human whereas Cr (VI) is toxic and carcinogenic. In this case study, risk assessment was performed using one seventh of the determined total Cr concentration

based on the assumption 1:6 ratio of Cr (III):Cr (VI) (USEPA 2013b). Nevertheless deeper insight to the chemical speciation of PM-bound metals is particularly important for future health risks assessment studies of indoor air pollution.

Finally, it should be mentioned that there are no similar studies in the literature with which the present results of exposure risks might be compared.

4. Conclusions

In this work, the risks associated with inhalation exposure to particulate-bound trace metals in hospital environment were estimated. Hospital staff and patients were the considered exposed groups.

Non-carcinogenic risks associated with inhalation exposure to $PM_{2.5\text{-}10}$ bound metals were acceptable to all age groups of hospital personnel whereas for fine fraction, total non-carcinogenic risks were above the safe level for all three age categories of hospital staff. Total carcinogenic risks in $PM_{2.5}$ highly (up to 67 times) exceeded the recommended level for the three age groups of hospital personnel, thus clearly showing that occupational exposure to metals in fine particles poses significant risks. If the extensive working hours of hospital medical staff were considered, the non- and carcinogenic risks were increased, the latter exceeding the USEPA cumulative guideline of 10^{-4}.

The non-carcinogenic and carcinogenic risks of adult patients were approximately three times higher than for personnel. Particular concerns (THQ>1, TR>10^{-6}) were observed for children and adolescents.

Hospitals are important public places where indoor air quality has a significant role on the potential health outcomes (both patients and employees). Even if the levels of respective indoor pollutants are low, the potential risks cannot be ignored considering the long-term exposures in these environments. Therefore, when assessing the health risks in hospital, the

specificity of exposure times should be considered. The non-carcinogenic and carcinogenic risks estimated in this work were via inhalation route. However, exposure to metals occurs also via ingestion and dermal contact and if these routes are considered the estimated risks might be higher. Moreover and if possible, metals speciation should be characterized in the several PM fractions.

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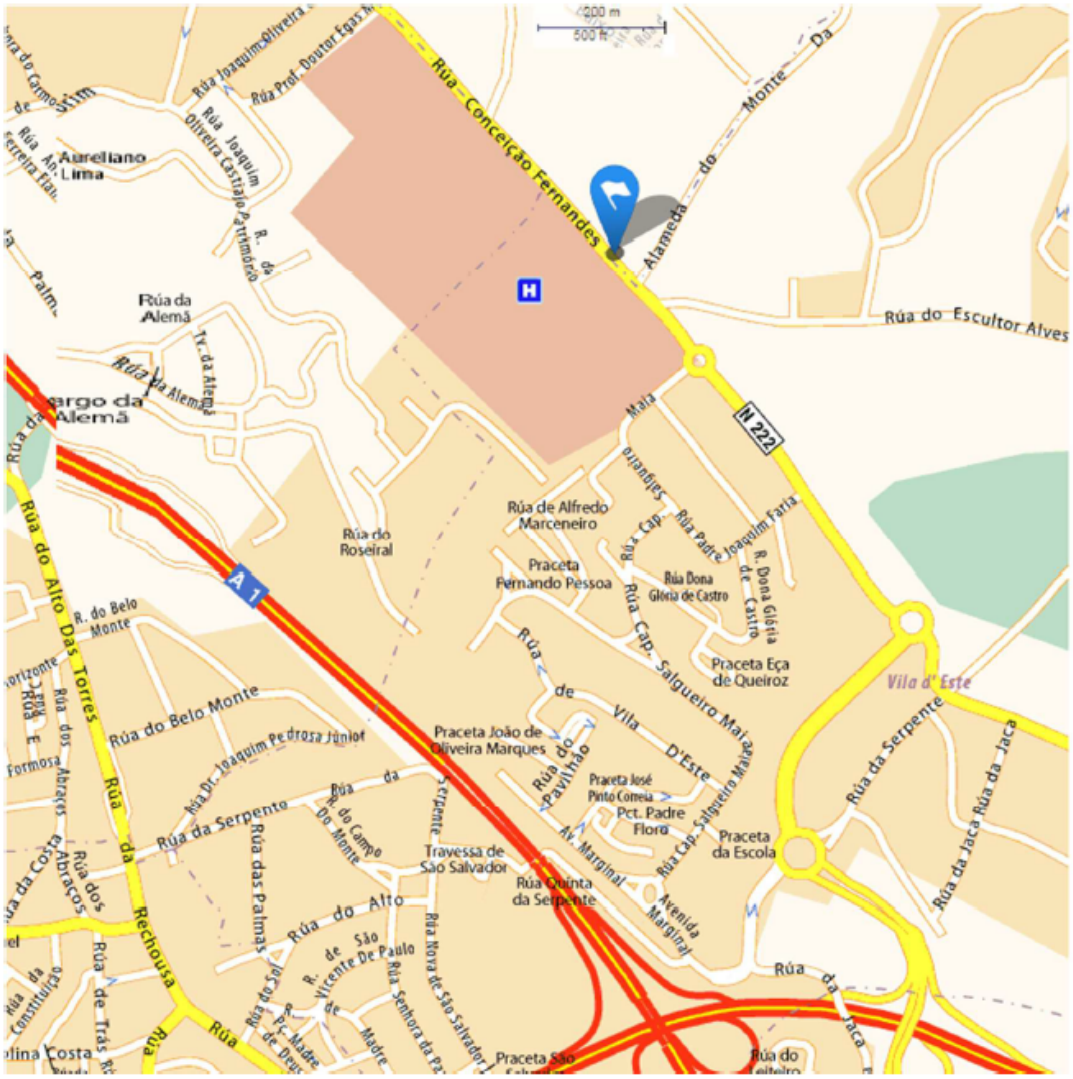

Fig. 1 Location of the studied hospital.

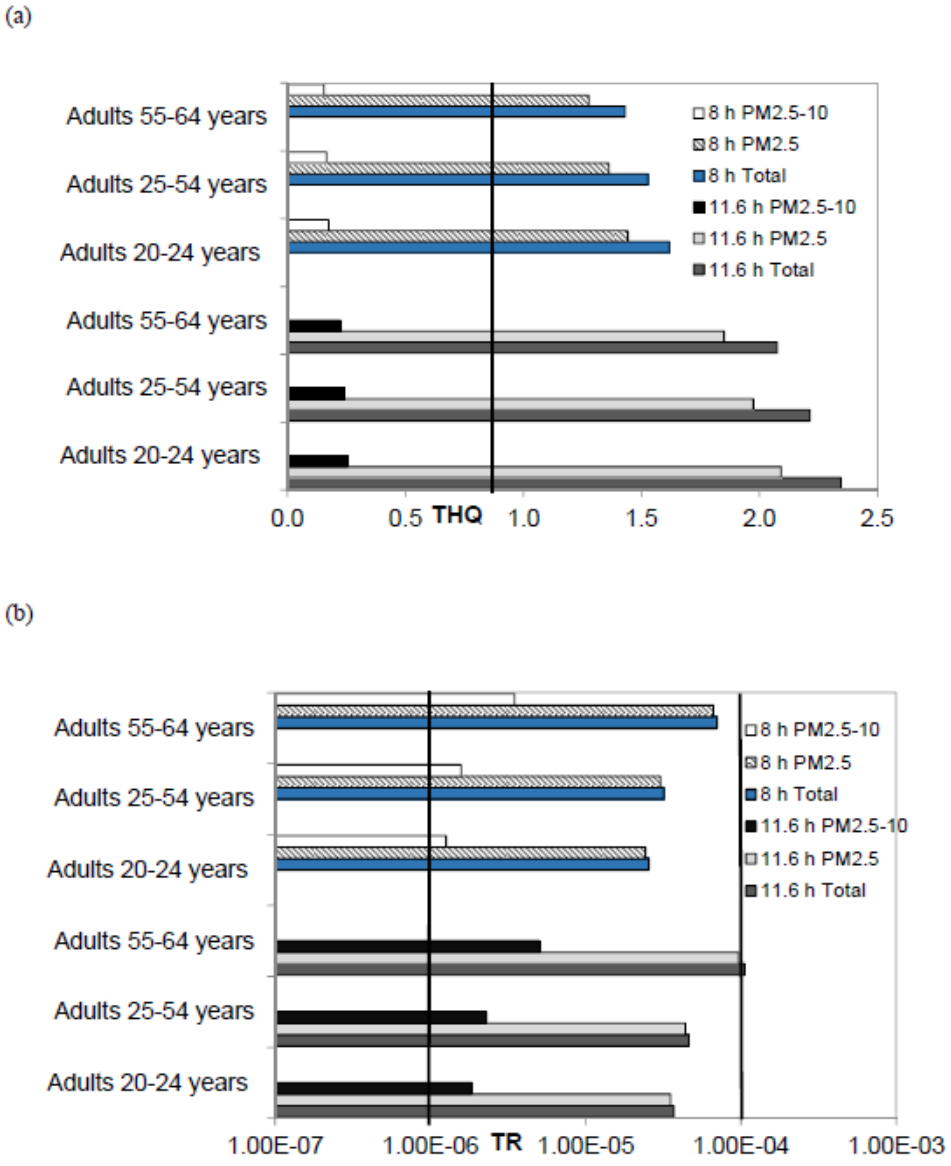

Fig. 2 Risks from inhalation exposure to particulate-bound metals for three age groups of hospital personnel (exposure time (ET) of 8 and 11.6 h per day): a) non carcinogenic; and b) carcinogenic. THQ and TR values are estimated as sum of individual non- and carcinogenic risk values of ten and four elements, respectively, in $PM_{2.5}$, $PM_{2.5\text{-}10}$ and in total PM (i.e. $PM_{2.5}$ + $PM_{2.5\text{-}10}$). Horizontal black lines indicate USEPA health–based guideline levels (THQ =1; and TR of 10^{-6} and 10^{-4}).

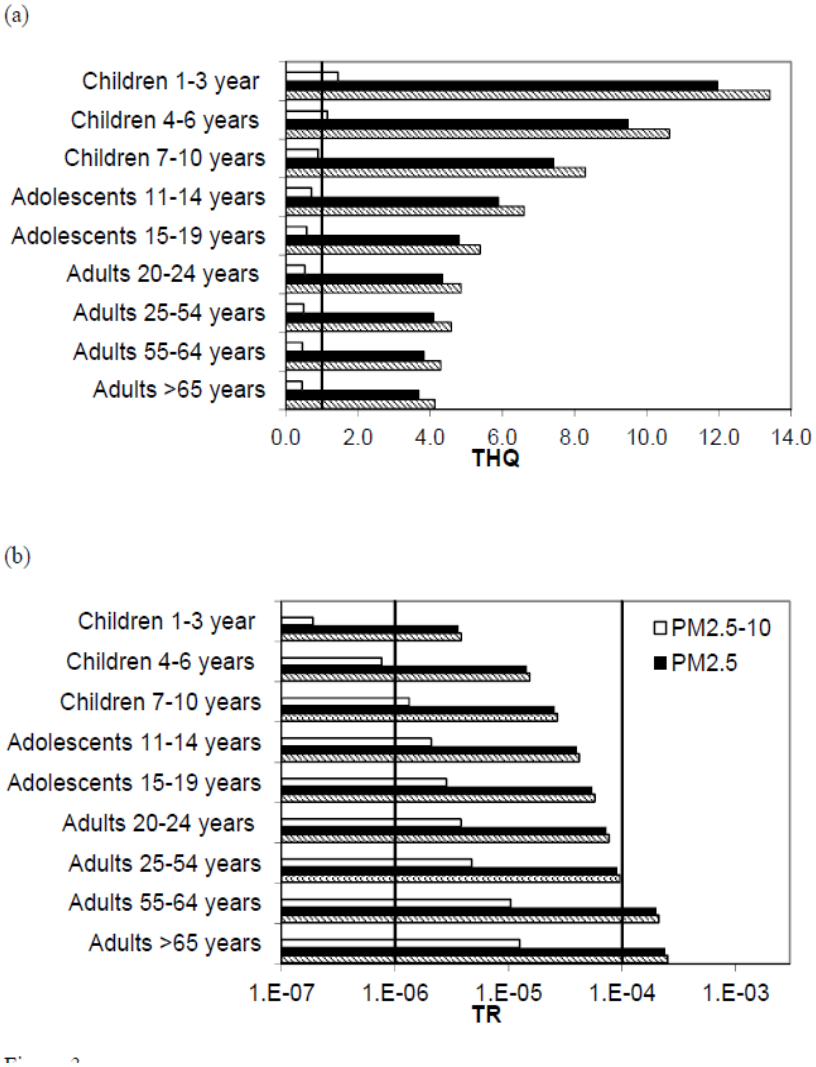

Fig. 3 Risks from inhalation exposure to particulate bound metals for nine age categories of patients (ET of 24 h per day): a) non-carcinogenic; and b) carcinogenic. The TR and THQ values represent, respectively, sum of individual risks of ten and four elements in $PM_{2.5}$, $PM_{2.5-10}$, and in total PM (i.e. $PM_{2.5}$ + $PM_{2.5-10}$). Horizontal black lines represent USEPA health–based guideline levels (THQ=1, and TR of 10^{-6} and 10^{-4}).

Table 1

RfD values of ten elements

Metal	RfC (mg m^{-3})	RfD (mg $kg^{-1}day^{-1}$)
Al	5.00×10^{-3}	1.43×10^{-3}
Si	3.00×10^{-3}	8.57×10^{-4}
Cl	1.50×10^{-4}	4.29×10^{-5}
Mn	5.00×10^{-5}	1.43×10^{-5}
Se	2.00×10^{-2}	5.71×10^{-3}
Ba	5.00×10^{-4}	1.43×10^{-4}
Cr	1.00×10^{-4}	2.86×10^{-5}
Ni	5.00×10^{-5}	1.43×10^{-5}
As	1.50×10^{-5}	4.29×10^{-6}
Cd	2.00×10^{-5}	5.71×10^{-6}

Table 2

Comparison of $PM_{2.5}$ and PM_{10} in hospitals: summary of existing studies

Country	Fraction	Mean (Min – Max) ($\mu g\ m^{-3}$)	Study organization	Sampling protocol	Reference
Portugal	PM_{10} $PM_{2.5}$ $PM_{2.5-10}$	31 (13 – 59) 23 (11 – 42) 7.4 (2.5 – 22)	1 hospital	24-h PM mass samples; collected during 28 days; constant flow (38.6 L min^{-1});	(this study)
Taiwan	PM_{10} $PM_{2.5}$	n.r. (22 – 90) n.r. (5 – 35)	8 hospitals; IAQ study of 39 public places;	2-min (phase 1) and 24-h (phase 2) PM collection; β-ray decay method;	Hsu et al. 2012
Taiwan	PM_{10} PM_{2}	n.r. (0.8–55.6) transplantation room: 10.7 (1.3 – 37.8) trauma room: 5.6 (3.2 – 55.6) cardiovascular surgery room: 3.0 (0.8 – 7.8) colon surgery room: 10.0 (1.6 – 49.1) orthopaedic surgery room: 12.6 (3.3 – 31.2) n.r. (0.1 – 8.4) transplantation room: 0.9 (0.2 – 3.1) trauma room: 1.1 (0.5 – 8.4) cardiovascular room: 0.3 (0.1 – 0.7) colon surgery room: 0.8 (0.3 – 2.6) orthopaedic room: 0.9 (0.4 – 7.5)	1 hospital; various operating rooms;	PM mass concentrations during 60 minutes; weakly sampling for 8 consecutive months; light-scattering aerosol analyzer; constant flow (1.2 L min^{-1});	Wan et al. 2011
Taiwan	PM_{10} $PM_{2.5}$	n.r. (n.r.) n.r. (n.r.)	6 hospitals; IAQ study of 21 public places	walk-through 2-min samples and during 24-h; β-ray decay method;	Wang et al. 2011
China	PM_{10} $PM_{2.5}$	128.13 (61.67– 250.00) 99.06 (40.94 – 214.91)	4 hospitals	24-h PM mass samples collected during total of 32 days; low flow samples (5 L min^{-1});	Wang et al. 2006a, 2006b
India	PM_{10} $PM_{2.5}$	136.36 – 316.11 (73.38 – 441.79) 67.28 – 95.70 (39.55 – 146.25)	5 hospitals	1-2 h continuous PM concentration measurements; light-scattering aerosol analyzer; constant flow (1.2 L min^{-1});	Verma and Taneja 2011
Turkey	$PM_{2.5}$	geriatrics: 18.1±4.5 (8.9 – 23.1) nephrology: 23.4±3.3 (16.4 – 31.4) cardiology: 37.9±13.3 (18.3 58.5)	assessment of 1 medical faculty including its hospital	8-h continuous PM concentration measurements during total of 26 workdays; light scattering sensing	Yurtseven et al. 2012

			and some clinics;	monitor; logging interval 15 s; constant flow rate;	
USA		n.r. (~ 2 – 8)[c]	residential and non-residential indoor micro-environments; 1 hospital;	7 consecutive days in 2 seasons; 24-h PM mass samples and continuous PM concentrations measurements;	Brown et al. 2012
Italy	$PM_{2.5}$	operating room: 1.6 ± 0.9 (n.r.) waiting room: 12.9±1.1 (n.r.) medical office: 14.8 ± 2.2 (n.r.)	2 hospitals; $PM_{2.5}$ assessed as marker for ETS[b]	10-h continuous concentration measurements in various hospital areas; laser-operated aerosol mass analyzer; logging interval 2 min;	Nardini et al. 2012
Spain	$PM_{2.5}$	17.94 (n.r.) dressing rooms: 8.92 (n.r.) fire escapes: 34.43 (n.r.) emergency department room: 16.11 (n.r.) hall: 18.90 (n.r.) general medicine : 12.46 (n.r.) cafeteria: 17.59 (n.r.) main entrance: 19.26 (n.r.) smoking area: 27.32 (n.r.)	53 hospitals; $PM_{2.5}$ assessed as marker for ETS; sample collection for 15 minutes;	15 min PM2.5 concentration samples at each location; light scattering aerosol monitor; constant flow (1.7 L min^{-1});	Sureda et al. 2010
Austria	$PM_{2.5}$	3.00[a] (n.r.)	30 hospitals in 7 European countries; $PM_{2.5}$ assessed as marker for ETS; 8 observed sub-locations: hall/main entrance, emergency department waiting room, internal medicine hospitalization unit, cafeteria, fire escape, general surgery, smoking areas (when existent), and other places	2-min PM mass concentrations at each sub-location; all hospitals sampled within 1-2 weeks; light scattering sensing monitor;	Fernandéz et al. 2009
Belgium	$PM_{2.5}$	3.0 [a] (n.r.)			
France	$PM_{2.5}$	3.5 [a] (n.r.)			
Germany	$PM_{2.5}$	1.5 [a] (n.r.)			
Greece	$PM_{2.5}$	4.0 [a] (n.r.)			
Romania	$PM_{2.5}$	10.0 [a] (n.r.)			
Spain	$PM_{2.5}$	5.0 [a] (n.r.)			

n.r. – not reported

IAQ- Indoor air quality
[a] – median (means not reported)
[b] – environmental tobacco smoke (ETS)
[c] concentration range retrieved from plot once precise figures are not given

Table 3

Mean concentrations of eleven studied elements in $PM_{2.5}$ and $PM_{2.5-10}$ at hospital (ng m^{-3})

	$PM_{2.5}$		$PM_{2.5-10}$	
	mean	range	mean	range
Al	98.8	46.1 – 144	48.7	5.22 – 244
Si	145	62.2 – 204	175	65.1 – 526
Cl	406	177 – 591	104	28.0 –279
Mn	2.73	0.49 – 5.78	0.777	0.06 –3.53
Se	0.762	0.58 – 0.89	0.139	0.12 – 0.42
Ba	9.00	4.26 – 18.4	16.1	2.61 –55.4
Cr	2.14	0.85 – 4.81	0.625	0.10 –2.07
Ni	3.02	0.77 – 7.74	0.506	0.07 –1.96
Cd	n.d.[b]	–	n.d.	–
As	80.3	39.8 – 140	2.72	0.27 –99.5
Pb	11.3	3.65 – 20.3	0.703	0.39 – 8.75
ΣE_{11}	759	271 – 1030	349	134 – 793
ΣE_{total}[a]	2890	1050 – 4510	1390	463 – 4070

[a]Total elemental concentration (i.e. represents sum of 21 elements); Slezakova et al. 2012

[b]n.d. – not detected

Table 4

Risk assessment by target hazard quotients (THQ)

	Target hazard quotient for hospital staff (ET 8 h)									
	$PM_{2.5}$									
	Al	Si	Cl	Mn	Se	Ba	Cr	Ni	As	Σ_{THQ}
Adults	3.45×10^{-3}	8.45×10^{-3}	4.73×10^{-1}	9.52×10^{-3}	6.65×10^{-6}	3.15×10^{-3}	5.34×10^{-4}	1.05×10^{-2}	9.35×10^{-1}	1.44
20–24 years	($1.61–5.03\times10^{-3}$)	($0.36–1.19\times10^{-2}$)	($2.06–6.88\times10^{-1}$)	($1.71–20.2\times10^{-3}$)	($5.07–7.77\times10^{-6}$)	($1.49–6.43\times10^{-3}$)	($2.13–12.0\times10^{-4}$)	($0.27–2.70\times10^{-2}$)	($4.63–16.3\times10^{-1}$)	(0.68–2.39)
Adults	3.26×10^{-3}	7.98×10^{-3}	4.46×10^{-1}	8.99×10^{-3}	6.28×10^{-6}	2.97×10^{-3}	5.04×10^{-4}	9.95×10^{-3}	8.82×10^{-1}	1.36
25–54 years	($1.52–4.75\times10^{-3}$)	($0.34–1.12\times10^{-2}$)	($1.95–6.50\times10^{-1}$)	($1.62–19.1\times10^{-3}$)	($4.87–7.34\times10^{-6}$)	$1.40–6.07\times10^{-3}$)	($2.01–11.3\times10^{-4}$)	($2.54–25.5\times10^{-3}$)	(4.3–15.4×10⁻)	(0.64–2.26)
Adults	3.05×10^{-3}	7.48×10^{-3}	4.18×10^{-1}	8.42×10^{-3}	5.88×10^{-6}	2.78×10^{-3}	4.73×10^{-4}	9.33×10^{-3}	8.27×10^{-1}	1.28
55–64 year	($1.42–4.45\times10^{-3}$)	($0.32–1.05\times10^{-2}$)	($1.82–6.09\times10^{-1}$)	(01.51–17.9×10⁻³)	($4.48–6.88\times10^{-6}$)	($1.32–5.69\times10^{-3}$)	($1.88–10.6\times10^{-4}$)	($2.38–23.9\times10^{-3}$)	($4.10–14.4\times10^{-1}$)	(0.60–2.11)
	$PM_{2.5-10}$									
	Al	Si	Cl	Mn	Se	Ba	Cr	Ni	As	Σ_{THQ}
Adults	1.70×10^{-3}	1.02×10^{-2}	1.21×10^{-1}	2.72×10^{-3}	1.21×10^{-6}	5.62×10^{-3}	1.56×10^{-4}	1.77×10^{-3}	3.17×10^{-2}	0.175
20–24 years	($0.18–8.52\times10^{-3}$)	($0.38–3.06\times10^{-2}$)	($0.33–3.25\times10^{-1}$)	($0.21–12.3\times10^{-3}$)	($1.05–3.67\times10^{-6}$)	($0.91–19.4\times10^{-3}$)	($0.25–5.17\times10^{-4}$)	($0.25–6.85\times10^{-3}$)	($0.31–116\times10^{-2}$)	(4.11×10^{-2}–1.56)
Adults	1.60×10^{-3}	9.61×10^{-3}	1.14×10^{-1}	2.56×10^{-3}	1.15×10^{-6}	5.30×10^{-3}	1.47×10^{-4}	1.67×10^{-3}	2.99×10^{-2}	0.165
25–54 years	($0.17–8.05\times10^{-3}$)	($3.58–28.9\times10^{-3}$)	($0.31–3.07\times10^{-1}$)	($0.20–11.6\times10^{-3}$)	($0.99–3.46\times10^{-6}$)	($0.86–18.3\times10^{-3}$)	($0.24–4.88\times10^{-4}$)	($0.23–6.46\times10^{-3}$)	($0.30–109\times10^{-2}$)	(3.88×10^{-2}–1.47)
Adults	1.50×10^{-3}	9.01×10^{-3}	1.07×10^{-1}	2.40×10^{-3}	1.07×10^{-6}	4.97×10^{-3}	1.38×10^{-4}	1.56×10^{-3}	2.80×10^{-2}	0.155
55–64 year	($0.16–7.54\times10^{-3}$)	($3.35–27.1\times10^{-3}$)	($0.29–2.87\times10^{-1}$)	($0.19–10.9\times10^{-3}$)	($0.93–3.24\times10^{-6}$)	($0.81–17.1\times10^{-3}$)	($0.21–4.57\times10^{-4}$)	($0.22–6.06\times10^{-3}$)	($0.28–103\times10^{-2}$)	(3.64×10^{-2}–1.38)
	Target hazard quotient for patients (ET 24 h)									
	$PM_{2.5}$									
	Al	Si	Cl	Mn	Se	Ba	Cr	Ni	As	Σ_{THQ}
Children	2.86×10^{-2}	7.01×10^{-2}	3.92	7.89×10^{-2}	5.51×10^{-5}	2.61×10^{-2}	4.43×10^{-3}	8.74×10^{-2}	7.75	12.0
1–3 years	($1.34–4.17\times10^{-2}$)	($3.00–9.85\times10^{-2}$)	(1.71–5.71)	(01.42–16.7×10⁻²)	($4.20–6.44\times10^{-5}$)	($1.23–5.33\times10^{-2}$)	($1.77–9.95\times10^{-3}$)	($2.23–22.4\times10^{-2}$)	(3.48–13.5)	(5.64–19.8)
Children	2.27×10^{-2}	5.55×10^{-2}	3.10	6.25×10^{-2}	4.37×10^{-5}	2.07×10^{-2}	3.51×10^{-3}	6.92×10^{-2}	6.14	9.48
4–6 years	($1.06–3.30\times10^{-2}$)	($2.38–7.80\times10^{-2}$)	(1.35–4.52)	($1.12–11.3\times10^{-2}$)	($3.3–5.11\times10^{-5}$)	($0.98–4.22\times10^{-2}$)	($1.40–7.88\times10^{-3}$)	($1.77–17.8\times10^{-2}$)	(3.04–10.7)	(14.47–15.7)
Children	1.77×10^{-2}	4.34×10^{-2}	2.42	4.88×10^{-2}	3.41×10^{-5}	1.61×10^{-2}	2.74×10^{-3}	5.41×10^{-2}	4.79	7.40
7–10 years	($0.83–2.58\times10^{-2}$)	($1.86–6.09\times10^{-2}$)	(1.06–3.53)	($0.88–10.4\times10^{-2}$)	($2.60–3.99\times10^{-5}$)	($0.77–3.30\times10^{-2}$)	($1.09–6.16\times10^{-3}$)	($1.38–13.9\times10^{-2}$)	(2.38–8.36)	(3.49–12.3)
Adolescents	1.41×10^{-2}	3.45×10^{-2}	1.93	3.89×10^{-2}	2.72×10^{-5}	1.28×10^{-2}	2.18×10^{-3}	4.30×10^{-2}	3.82	5.89
11–14 years	($0.66–2.05\times10^{-2}$)	($1.48–4.85\times10^{-2}$)	(0.84–2.81	($0.70–8.24\times10^{-2}$)	($2.07–3.17\times10^{-5}$)	($0.61–2.62\times10^{-2}$)	($0.89–4.90\times10^{-3}$)	($1.10–11.0\times10^{-2}$)	(1.89–6.66)	(2.78–9.76)
Adolescents	1.15×10^{-2}	2.81×10^{-2}	1.57	3.17×10^{-2}	2.21×10^{-5}	1.05×10^{-2}	1.78×10^{-3}	3.51×10^{-2}	3.11	4.80

15–19 years	(0.54–1.67×10^{-2})	(1.21–3.95×10^{-2})	(0.69–2.29)	(0.57–6.72×10^{-2})	(1.69–2.59×10^{-5})	(4.95–2.14×10^{-2})	(0.71–4.00×10^{-3})	(0.90–9.00×10^{-2})	(1.54–5.43)	(2.27–7.96)
Adults	1.04×10^{-2}	2.54×10^{-2}	1.42	2.86×10^{-2}	2.00×10^{-5}	9.44×10^{-3}	1.60×10^{-3}	3.16×10^{-2}	2.80	4.33
20–24 years	(0.48–1.51×10^{-2})	(1.09–3.56×10^{-2})	(0.62–2.06)	(0.51–6.06×10^{-2})	(1.52–2.33×10^{-5})	(4.47–19.3×10^{-3})	(0.64–3.60×10^{-3})	(0.81–8.11×10^{-2})	(1.39–4.89)	(2.04–7.17)
Adults	9.77×10^{-3}	2.39×10^{-2}	1.34	2.70×10^{-2}	1.88×10^{-5}	8.91×10^{-3}	1.51×10^{-3}	2.99×10^{-2}	2.65	4.09
25–54 years	(4.56–14.2×10^{-3})	(1.03–3.36×10^{-2})	(0.58–1.95)	(0.49–5.72×10^{-2})	(1.43–2.20×10^{-5})	(4.21–18.2×10^{-3})	(0.60–3.40×10^{-3})	(0.76–7.66×10^{-2})	(1.31–4.62)	(1.93–6.77)
Adults	9.16×10^{-3}	2.24×10^{-2}	1.25	2.53×10^{-2}	1.77×10^{-5}	8.35×10^{-3}	1.42×10^{-3}	2.80×10^{-2}	2.48	3.83
55–64 year	(4.27–13.4×10^{-3})	(0.96–3.15×10^{-2})	(0.55–1.83)	(0.45–5.36×10^{-2})	(1.34–2.06×10^{-5})	(3.95–17.1×10^{-3})	(5.65–3.19×10^{-3})	(0.71–0.72×10^{-2})	(1.23–4.33)	(1.81–6.34)
Seniors	8.80×10^{-3}	2.16×10^{-2}	1.21	2.43×10^{-2}	1.70×10^{-5}	8.02×10^{-3}	1.36×10^{-3}	2.69×10^{-2}	2.38	3.68
>65 years	(4.11–12.8×10^{-3})	(0.92–3.03×10^{-2})	(0.53–1.76)	(0.44–5.15×10^{-2})	(1.29–1.98×10^{-5})	(3.83–16.4×10^{-3})	(5.43–3.06×10^{-3})	(0.69–6.91×10^{-2})	(1.18–4.16)	(1.74–6.10)

	$PM_{2.5-10}$ Al	Si	Cl	Mn	Se	Ba	Cr	Ni	As	Σ_{THQ}
Children	1.41×10^{-2}	8.44×10^{-2}	10.1	2.25×10^{-2}	1.01×10^{-5}	4.66×10^{-2}	1.29×10^{-3}	1.47×10^{-2}	2.63×10^{-1}	1.45
1–3 years	(0.15–7.07×10^{-2})	(3.14–25.4×10^{-2})	(0.27–26.9)	(0.17–10.2×10^{-2})	(0.87–3.04×10^{-5})	(0.76–16.0×10^{-2})	(0.21–4.28×10^{-3})	(0.20–5.68×10^{-2})	(0.26–96.1×10^{-1})	(2.70×10^{-1}–12.9)
Children	1.12×10^{-2}	6.69×10^{-2}	7.95×10^{-1}	1.78×10^{-2}	7.97×10^{-6}	3.69×10^{-2}	1.02×10^{-3}	1.16×10^{-2}	2.08×10^{-1}	1.15
4–6 years	(0.12–5.60×10^{-2})	(2.49–20.1×10^{-2})	(2.14–21.3×10^{-1})	(0.14–8.10×10^{-2})	(6.88–24.1×10^{-6})	(0.60–12.7×10^{-2})	(0.16–3.39×10^{-3})	(0.16–4.50×10^{-2})	(0.21–76.1×10^{-1})	(2.70×10^{-1}–10.3)
Children	8.72×10^{-3}	5.22×10^{-2}	6.21×10^{-1}	1.39×10^{-2}	6.23×10^{-6}	2.88×10^{-2}	8.00×10^{-4}	9.07×10^{-3}	1.63×10^{-1}	8.97×10^{-1}
7–10 years	(0.94–4.37×10^{-3})	(1.94–15.7×10^{-2})	(1.67–16.7×10^{-1})	(0.11–6.33×10^{-2})	(5.38–18.8×10^{-6})	(0.47–9.93×10^{-2})	(1.28–26.5×10^{-4})	(0.13–35.1×10^{-3})	(0.16–59.4×10^{-1})	(2.11–80.1×10^{-1})
Adolescents	6.94×10^{-3}	4.16×10^{-2}	4.94×10^{-1}	11.1×10^{-3}	4.95×10^{-6}	2.29×10^{-2}	6.37×10^{-4}	7.22×10^{-3}	1.29×10^{-1}	7.14×10^{-1}
11–14 years	(0.74–34.8×10^{-3})	(1.55–12.5×10^{-2})	(1.33–13.4×10^{-1})	(0.87–50.3×10^{-3})	(4.28–15.0×10^{-6})	(0.37–7.90×10^{-2})	(1.02–21.1×10^{-4})	(1.02–28.0×10^{-3})	(0.13–47.3×10^{-1})	(1.68–63.8×10^{-1})
Adolescents	5.66×10^{-3}	3.39×10^{-2}	4.03×10^{-1}	9.04×10^{-3}	4.04×10^{-6}	1.87×10^{-2}	5.19×10^{-4}	5.89×10^{-3}	1.06×10^{-1}	5.82×10^{-1}
15–19 years	(0.61–28.4×10^{-3})	(1.26–10.2×10^{-2})	(1.09–10.8×10^{-1})	(0.70–41.0×10^{-3})	(3.49–12.2×10^{-6})	(0.30–6.44×10^{-2})	(0.83–17.2×10^{-4})	(0.81–22.8×10^{-3})	(0.11–38.6×10^{-1})	(1.37–52.0×10^{-1})
Adults	5.10×10^{-3}	3.06×10^{-2}	3.63×10^{-1}	8.15×10^{-3}	3.64×10^{-6}	1.69×10^{-2}	4.68×10^{-4}	5.31×10^{-3}	9.51×10^{-2}	5.25×10^{-1}
20–24 years	(0.55–25.6×10^{-3})	(1.14–9.19×10^{-2})	(0.98–9.75×10^{-1})	(0.63–37.0×10^{-3})	(3.14–11.0×10^{-6})	(0.27–5.81×10^{-2})	(0.75–15.5×10^{-4})	(0.73–20.5×10^{-3})	(0.94–348×10^{-2})	(1.23–×46.910^{-1})
Adults	4.81×10^{-3}	2.88×10^{-2}	3.43×10^{-1}	7.69×10^{-3}	3.44×10^{-6}	1.59×10^{-2}	4.42×10^{-4}	5.01×10^{-3}	8.98×10^{-2}	4.95×10^{-1}
25–54 years	(0.52–24.1×10^{-3})	(1.07–8.67×10^{-2})	(0.92–9.23×10^{-1})	(0.59–34.9×10^{-3})	(2.97–10.4×10^{-6})	(0.26–5.48×10^{-2})	(0.71–14.6×10^{-4})	(0.69–19.4×10^{-3})	(0.89–328×10^{-2})	(1.16–44.2×10^{-1})
Adults	4.51×10^{-3}	2.70×10^{-2}	3.21×10^{-1}	7.21×10^{-3}	3.22×10^{-6}	1.49×10^{-2}	4.14×10^{-4}	4.49×10^{-3}	8.41×10^{-2}	4.64×10^{-1}
55–64 year	(0.48–22.6×10^{-3})	(1.01–8.13×10^{-2})	(0.87–8.62×10^{-1})	(0.56–32.7×10^{-3})	(2.78–9.73×10^{-6})	(0.24–5.14×10^{-2})	(0.66–13.7×10^{-4})	(0.65–18.2×10^{-3})	(0.83–308×10^{-2})	(1.09–41.4×10^{-1})
Seniors	4.34×10^{-3}	2.60×10^{-2}	3.09×10^{-1}	6.96×10^{-3}	3.10×10^{-6}	1.43×10^{-2}	3.98×10^{-4}	4.51×10^{-3}	8.09×10^{-2}	4.46×10^{-1}
>65 years	(0.47–21.7×10^{-3})	(0.97–7.81×10^{-2})	(0.83–8.29×10^{-1})	(0.54–31.5×10^{-3})	(0.89–9.36×10^{-6})	(0.23–4.94×10^{-2})	(0.64–13.2×10^{-4})	(0.62–17.2×10^{-3})	(0.80–296×10^{-2})	1.05–39.8×10^{-1})

Table 5

Estimated target carcinogenic risks (TR) of PM–bound carcinogenic elements and $PM_{2.5}$

Age group	**Target carcinogenic risk for hospital staff (ET = 8 h)**					
	$PM_{2.5}$–bound carcinogenic elements					$PM_{2.5}$
	Cr	Ni	As	Pb	Σ_{TR}	
Adults 20–24 years	1.68×10^{-6} ($0.67–3.76\times10^{-6}$)	9.45×10^{-8} ($2.41–24.2\times10^{-8}$)	2.25×10^{-5} ($1.12–3.92\times10^{-5}$)	8.87×10^{-9} ($2.86–15.9\times10^{-9}$)	2.43×10^{-5} ($1.19–4.33\times10^{-5}$)	1.22×10^{-2} ($0.58–2.19\times10^{-2}$)
Adults 25–54 years	2.09×10^{-6} ($0.84–4.70\times10^{-6}$)	1.18×10^{-7} ($0.30–3.03\times10^{-7}$)	2.81×10^{-5} ($1.40–4.90\times10^{-5}$)	1.11×10^{-8} ($0.36–1.99\times10^{-8}$)	3.04×10^{-5} ($1.48–5.41\times10^{-5}$)	1.53×10^{-2} ($0.72–2.74\times10^{-2}$)
Adults 55–64 year	4.61×10^{-6} ($1.84–10.3\times10^{-6}$)	2.60×10^{-7} ($0.66–6.66\times10^{-7}$)	6.19×10^{-5} ($3.07–10.8\times10^{-5}$)	2.44×10^{-8} ($0.79–4.37\times10^{-8}$)	6.68×10^{-5} ($3.26–11.9\times10^{-5}$)	3.36×10^{-2} ($1.58–6.03\times10^{-2}$)
	$PM_{2.5–10}$–bound carcinogenic elements					
	Cr	Ni	As	Pb	Σ_{TR}	
Adults 20–24 years	4.89×10^{-7} ($0.78–16.2\times10^{-7}$)	1.58×10^{-8} ($0.22–6.14\times10^{-8}$)	7.64×10^{-7} ($0.76–279\times10^{-7}$)	5.50×10^{-10} ($3.05–68.5\times10^{-10}$)	1.27×10^{-6} ($0.16–29.6\times10^{-6}$)	
Adults 25–54 years	6.11×10^{-7} ($0.98–20.3\times10^{-7}$)	1.98×10^{-8} ($0.27–7.67\times10^{-8}$)	9.55×10^{-7} ($0.95–349\times10^{-7}$)	6.88×10^{-10} ($3.82–85.6\times10^{-10}$)	1.59×10^{-6} ($0.2–37.0\times10^{-6}$)	
Adults 55–64 year	1.35×10^{-6} ($0.22–4.46\times10^{-6}$)	4.36×10^{-8} ($0.60–16.9\times10^{-8}$)	2.10×10^{-6} ($0.21–76.8\times10^{-6}$)	1.51×10^{-9} ($0.84–18.8\times10^{-9}$)\	3.49×10^{-6} ($0.43–81.4\times10^{-6}$)	
Age group	**Target carcinogenic risk for patients (ET = 24 h)**					
	$PM_{2.5}$–bound carcinogenic elements					$PM_{2.5}$
	Cr	Ni	As	Pb	Σ_{TR}	
Children 1–3 years	2.51×10^{-8} ($1.01–5.64\times10^{-7}$)	1.42×10^{-8} ($0.36–3.63\times10^{-8}$)	3.38×10^{-6} ($1.67–5.88\times10^{-6}$)	1.33×10^{-9} ($0.43–2.39\times10^{-10}$)	3.64×10^{-6} ($1.78–6.49\times10^{-6}$)	1.83×10^{-3} ($0.86–3.29\times10^{-3}$)
Children 4–6 years	1.01×10^{-6} ($0.40–2.26\times10^{-6}$)	5.67×10^{-8} ($1.45–14.5\times10^{-8}$)	1.35×10^{-5} ($0.67–2.35\times10^{-5}$)	5.32×10^{-9} ($1.71–9.54\times10^{-9}$)	1.46×10^{-5} ($0.71–2.60\times10^{-5}$)	7.32×10^{-3} ($3.44–13.2\times10^{-3}$)

Children 7–10 years	1.76×10^{-6} ($0.71–3.95\times10^{-6}$)	9.92×10^{-8} ($0.25–25.4\times10^{-8}$)	2.36×10^{-5} ($1.17–4.12\times10^{-5}$)	9.31×10^{-9} ($3.00–16.7\times10^{-9}$)	2.55×10^{-5} ($1.25–4.54\times10^{-5}$)	1.28×10^{-2} ($0.60–2.30\times10^{-2}$)
Adolescents 11–14 years	2.76×10^{-6} ($1.10–6.21\times10^{-6}$)	1.57×10^{-7} ($0.39–4.01\times10^{-7}$)	3.71×10^{-5} ($1.84–6.47\times10^{-5}$)	1.46×10^{-8} ($0.47–2.62\times10^{-8}$)	4.01×10^{-5} ($1.96–7.14\times10^{-5}$)	2.01×10^{-2} ($0.95–3.62\times10^{-2}$)
Adolescents 15–19 years	3.77×10^{-6} ($0.15–8.47\times10^{-6}$)	2.13×10^{-7} ($0.54–5.45\times10^{-7}$)	5.07×10^{-5} ($2.51–8.83\times10^{-5}$)	1.99×10^{-8} ($0.64–3.58\times10^{-9}$)	5.47×10^{-5} ($2.67–9.73\times10^{-5}$)	2.75×10^{-2} ($1.29–4.93\times10^{-2}$)
Adults 20–24 years	5.03×10^{-6} ($2.00–11.3\times10^{-6}$)	2.83×10^{-7} ($0.72–7.27\times10^{-7}$)	6.75×10^{-5} ($3.35–11.8\times10^{-5}$)	2.66×10^{-8} ($0.86–4.77\times10^{-9}$)	7.29×10^{-5} ($3.56–13.0\times10^{-5}$)	3.66×10^{-2} ($1.72–6.58\times10^{-2}$)
Adults 25–54 years	6.28×10^{-6} ($2.51–14.1\times10^{-6}$)	3.54×10^{-7} ($0.90–9.08\times10^{-7}$)	8.44×10^{-5} ($4.19–14.7\times10^{-5}$)	3.32×10^{-8} ($1.07–5.97\times10^{-8}$)	9.11×10^{-5} ($4.45–16.2\times10^{-5}$)	4.58×10^{-2} ($2.15–8.22\times10^{-2}$)
Adults 55–64 year	1.38×10^{-5} ($0.55–3.10\times10^{-6}$)	7.79×10^{-7} ($1.99–20.0\times10^{-7}$)	1.86×10^{-4} ($0.92–3.24\times10^{-4}$)	7.31×10^{-8} ($2.36–13.1\times10^{-8}$)	2.00×10^{-4} ($0.98–3.57\times10^{-4}$)	1.01×10^{-1} ($0.47–1.81\times10^{-1}$)
Seniors >65 years	1.66×10^{-5} ($0.66–3.73\times10^{-5}$)	9.35×10^{-7} ($2.39–24.0\times10^{-7}$)	2.23×10^{-4} ($0.11–3.88\times10^{-5}$)	8.78×10^{-8} ($2.83–15.7\times10^{-8}$)	2.40×10^{-4} ($1.17– 4.28\times10^{-4}$)	1.21×10^{-1} ($0.57–2.17\times10^{-1}$)

	$PM_{2.5–10}$–bound carcinogenic elements				
	Cr	Ni	As	Pb	Σ_{TR}
Children 1–3 years	7.34×10^{-8} ($1.17–24.3\times10^{-8}$)	2.38×10^{-9} ($0.33–9.21\times10^{-9}$)	1.15×10^{-7} ($0.11–41.9\times10^{-7}$)	8.25×10^{-11} ($4.58–10.3\times10^{-11}$)	1.90×10^{-7} ($0.24–44.5\times10^{-7}$)
Children 4–6 years	2.93×10^{-7} ($0.47–9.72\times10^{-7}$)	9.51×10^{-9} ($1.32–36.8\times10^{-9}$)	4.58×10^{-7} ($0.45–167\times10^{-7}$)	3.30×10^{-10} ($1.83–41.1\times10^{-10}$)	7.62×10^{-7} ($0.94–17.8\times10^{-7}$)
Children 7–10 years	5.14×10^{-7} ($0.82–17.0\times10^{-7}$)	1.66×10^{-8} ($0.23–6.44\times10^{-8}$)	8.02×10^{-7} ($0.80–293\times10^{-7}$)	5.78×10^{-10} ($3.21–71.9\times10^{-10}$)	1.33×10^{-6} ($0.16–31.1\times10^{-6}$)
Adolescents 11–14 years	8.07×10^{-7} ($1.29–26.7\times10^{-7}$)	2.62×10^{-8} ($0.36–10.1\times10^{-8}$)	1.26×10^{-6} ($0.13–46.1\times10^{-6}$)	9.08×10^{-10} ($5.04–113\times10^{-10}$)	2.09×10^{-6} ($0.26–48.8\times10^{-6}$)
Adolescents 15–19 years	1.10×10^{-6} ($0.18–3.65\times10^{-6}$)	3.57×10^{-8} ($0.49–13.8\times10^{-8}$)	1.72×10^{-6} ($0.17–62.8\times10^{-6}$)	1.24×10^{-9} ($0.69–15.4\times10^{-9}$)	2.86×10^{-6} ($0.35–66.7\times10^{-6}$)
Adults 20–24 years	1.47×10^{-6} ($0.24–4.86\times10^{-6}$)	4.75×10^{-8} ($0.66–18.4\times10^{-8}$)	2.29×10^{-6} ($0.23–83.7\times10^{-6}$)	2.65×10^{-9} ($0.92–20.5\times10^{-9}$)	3.81×10^{-6} ($0.47–88.8\times10^{-6}$)
Adults 25–54 years	1.83×10^{-6} ($0.29–6.08\times10^{-7}$)	5.94×10^{-8} ($0.82–23.0\times10^{-8}$)	2.86×10^{-6} ($0.28–105\times10^{-6}$)	2.06×10^{-9} ($1.14–25.7\times10^{-9}$)	4.76×10^{-6} ($0.59–111\times10^{-6}$)
Adults 55–64 year	4.04×10^{-6}	1.31×10^{-7}	6.30×10^{-6}	4.54×10^{-9}	1.05×10^{-5}

Seniors >65 years	(0.65–13.4×10^{-6}) 4.84 × 10^{-6} (0.78–16.0×10^{-6})	(0.18–5.06×10^{-7}) 1.57 × 10^{-7} (0.22–6.08×10^{-7})	(0.63–203×10^{-6}) 7.56 × 10^{-6} (00.75–276×10^{-6})	(2.52–56.5×10^{-9})\ 5.45 × 10^{-9} (3.02–67.8×10^{-9})	(0.13–24.4×10^{-5}) 1.26 × 10^{-5} (0.16–29.3×10^{-5})