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Review

Sources, health effects and control strategies of indoor fine particulate matter ($PM_{2.5}$): A review

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GRAPHICAL ABSTRACT

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ABSTRACT

Indoor air quality is directly influenced by indoor $PM_{2.5}$. Short-term and long-term exposure of $PM_{2.5}$ in the micro environment would severely detriment the health of both humans and animals. The researches both at home and abroad dating from 2000 were analyzed and summarized mainly in the following 3 sections: source apportionment, health effects and control methods. Health effects were illustrated in both epidemiology and toxicology. The epidemiology was explicated in morbidity and mortality, the toxicology was illuminated in inflammatory reaction, oxidative stress, genotoxicity, mutagenicity and carcinogenicity. Control methods were showed in two aspects (sources and means of transmission), of which each was resolved by corresponding control strategy. Abundant investigations indicated that comprehensive control strategies were needed for sources decrement and health burden mitigation of indoor $PM_{2.5}$. Based on the increasingly wide research of indoor $PM_{2.5}$, the concept of indoors was essentially expanded, and on the basis of the summary of all the aspects mentioned above, both the scope and depth of indoor $PM_{2.5}$ research were found insufficiently. Meantime, the potential direction of development in indoor $PM_{2.5}$ research were projected, in hope of contributing to further relevant study of engineers in ambient environment and building environment.

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Contents

1. Introduction

$PM_{2.5}$ refers to PM with an aerodynamic diameter <2.5 μm(micro) (Fig. 1), also called fine particulate matter and was firstly established in 1997 by the U.S. environmental protection agency (USEPA) (Federal-Register, 1997) to protect public health, which was introduced by china in some standards and acts such as Ambient Air Quality Standards (GB 3095—2012) (MEP, 2012). From the 1970s to now, due to the increased health risk and economic burden of unreasonable industry development, ambient $PM_{2.5}$ or outdoor $PM_{2.5}$ have been studied widely, but it will continues. Numerous studies have been conducted about the association between ambient $PM_{2.5}$ and its impact (Pui et al., 2014; Stieb et al., 2012; Tam et al., 2015). However, nowadays people have spent as much as 80% to 90% hours indoors, such homes, offices, and transport vehicles (Klepeis et al., 2001; Leech et al., 2002; Robinson and Nelson, 1995). Meantime indoor air quality, which have a directly significant influence on health as well as efficiency of study and work (Deng et al., 2016; Lu et al., 2016; Norbäck et al., 2016), is becoming increasingly worse. Therefore, indoor $PM_{2.5}$ should be paid more attention, following some indoor gaseous pollutant studies, such as methane, radon and SO_2. Compared with outdoor $PM_{2.5}$ and indoor gaseous pollutants studies, the depth of indoor $PM_{2.5}$ study is far from enough (Schneider et al., 2003; Sundell, 2004), while more and more papers were published

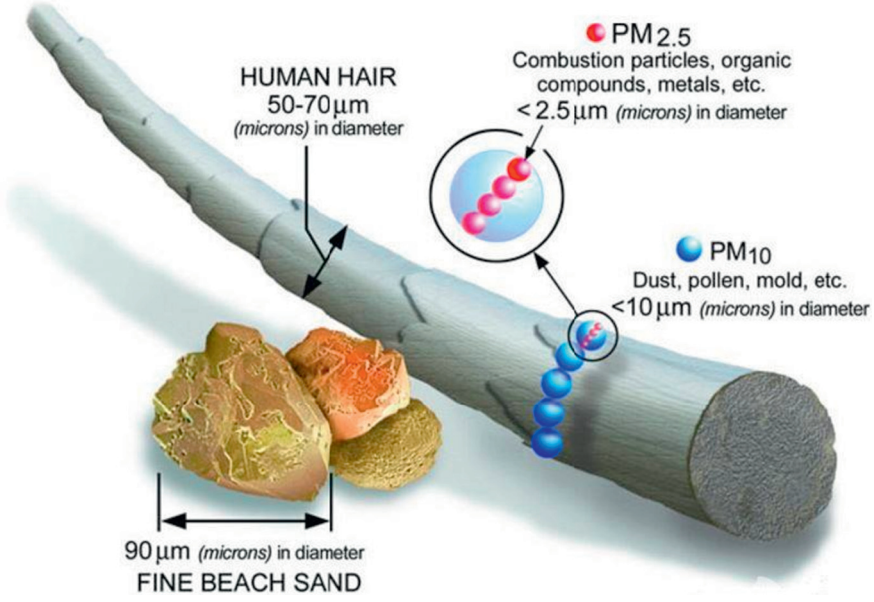

Fig. 1. Comparison of particle diameter.

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about source apportionment, specific mechanism of health impacts, and mitigation of indoor $PM_{2.5}$ (Chang et al., 2011; Meng et al., 2009; Wallace, 1996).

The aim of this paper is to provide a summary of studies on indoor $PM_{2.5}$. To the best of our knowledge, overall reviews about indoor $PM_{2.5}$ is little. For example, while Wang et al. (2016) summarized the pollution situation, control technologies etc. health effects of indoor $PM_{2.5}$ wasn't explained. Although the fine fraction of indoor particles was analyzed by Wallace (1996), only several large research projects of the United States were selected, from which a thorough understanding of the world was difficult to get. This paper only involves the articles published during 2000 and 2016, which were searched with specific search terms and selection criteria to generalize the overall situation of indoor $PM_{2.5}$.

2. Methodology

2.1. Literature search

For the purpose of this review, PubMed, Web of Science, Embase, Google Scholar and CNKI electronic databases were searched for all published studies. Papers' associated level with research contents were also evaluated by using the following terms: “indoor air quality” or “indoor pollution”, “indoor $PM_{2.5}$” or “indoor fine particulate matter” and “sources” or “source apportionment” for indoor $PM_{2.5}$ sources investigation; “indoor $PM_{2.5}$”, “health effects”, “short-term” and “long-term” for health effects summing-up; “air purification” and “standard” for control technologies provision. When searching, date was restricted from 2000 to 2016. Original studies about human health effects published in English were included, with several excellent published in Chinese. Additional studies from the reference lists of these identified articles and previous relevant published reviews were also searched. The selection of original studies was based on the inspection of the titles and/or abstracts of the potentially eligible articles. Then, potentially eligible articles were browsed entirely and quickly, and relevant sections were read carefully to determine whether they were suitable for the inclusion and exclusion criteria in the review.

2.2. Study selection (inclusion and exclusion criteria) and data extraction

As for sources, any field or numerical studies were included as long as they demonstrated the existence of sources, even some sort of source that couldn't be perceived by humans. With regard to health effects, epidemiologic studies would be included on condition that they presented the short- and/or long-term exposure to $PM_{2.5}$ with effect estimate (relative risk (RR)/odds ratio and 95% confidence intervals (95% CI)). Short-term was defined as a few hours to a few days (0–7 days) before the event day, long-term was defined as >1 year. And, if there were no RR, mortality or morbidity of all-cause disease, respiratory diseases or circulatory diseases shown in a paper, it would be extracted. If a study did not present enough quantitative data, an email would be sent to the author, and the study would be excluded without getting an answer. For ease of comparison, same parameters, like mortality with same period of lag time and $PM_{2.5}$ mass concentration in $\mu g/m^3$, were chosen. For instance, in Table 2 only total disease mortality, CVD mortality and respiratory disease mortality with 0–1 lag days were chosen as possible.

According to the inclusion and exclusion criteria in the study selection, two investigators searched and extracted date from all eligible studies independently to ensure the accuracy of information. Then two investigators checked the selected data carefully and repeatedly until no mistakes were found.

3. Background

3.1. History of $PM_{2.5}$ pollution problem

Some researches indicated that $PM_{2.5}$ had already existed before man appeared. Volcanic eruption and forest fire, both of them would emit $PM_{2.5}$ to environment (Vellingiri et al., 2015; Yi and Bao, 2016). Those gaseous or liquid particles suspending in the air, together with other materials, change the earth quietly and slowly (Duggen et al., 2010; Robock, 2000). With the appearance of humans, houses were built for shelter. However, the fresh air was also blocked at the same time, resulting in accumulation of $PM_{2.5}$ indoors. With the technological advancement, more humans' activities were carried out indoors, from early cooking by biofuels and cleaning to studying, working and present industrial activities, all of which contributed to indoor $PM_{2.5}$ increment. At present, cooking by biofuels is still the main source of indoor pollutants. Therefore, the deterioration of indoor air quality (IAQ) by indoor $PM_{2.5}$ pollution worth studying, whether it is the past or the present.

There were few studies about IAQ owing to backwardness in science and technology before the 20th century (Sundell, 2004), not mention to $PM_{2.5}$ that was later known to the public because of severe hazes in some counties, like China (Asian-Development-Bank, 2013; Zhang and Samet, 2015). But it didn't indicate that indoor $PM_{2.5}$ pollution didn't existed. An experimental archeology project was undertaken in two Danish reconstructed Viking Age (from 790 to 1066) houses with indoor open fireplaces. Volunteers inhabited the houses under living conditions similar to those of the Viking Age, including cooking and heating by wood fire. Observation showed that the average personal exposure of CO and $PM_{2.5}$ was 6.9 ppm and 0.41 mg/m^3, respectively, more than reported from modern studies conducted in dwellings using biomass for cooking and heating (Christensen and Ryhl-Svendsen, 2015; Zhang and Samet, 2015). The results demonstrated that as early as the Danish Viking Age, indoor air was polluted by $PM_{2.5}$ emitted from wood combustion and $PM_{2.5}$ in wood smoke had an adverse effect on human health.

PM pollution isn't becoming a hot spot of study until the mid-20th century. The original government report (U. K. Ministry Of Health, 1954) on the health effects of London Smog Disasters, which happened between December 4, 1952 and December 9, 1952, claimed that additional deaths from this time through the end of March was >8000. Then the U.K. Ministry of Health estimated that 5655 people died from influenza in the first 3 months of 1953 (U. K. Ministry Of Health, 1956). The main pollutants were sulphur oxides and dust. Similarly, photochemical smog event happened three times in Los Angeles, in 1943, 1952 and 1955. During the event in 1952, >400 the elderly over 65 died, which also occurred in September 1955. Over the period of 1950–1951, the economic loss in America had reached 1.5 billion dollars, Both London Smog Disasters and Los Angeles Photochemical Smog Event were one of the ten major environmental hazards in the 20th Century (Peng, 2001), in which there were totally five smog events. Thus, it is obvious that in 20th century air was polluted severely. Due to the frequent large-scale air pollution related to PM, countries have issued series of relevant regulations and standards to fight against the PM. For example, City of London (Various Powers) Act 1954, followed by Clean Air Act in 1956, was introduced by British government (Heys, 2012).Clean Air Act of USA was promulgated in 1970, which was amended in 1990 (EPA, 1997; Sueyoshi and Goto, 2009). Afterwards, it was found by researchers that in PM it was $PM_{2.5}$ that had a major impact on health (Hassanvand et al., 2015; Hoek et al., 2013). Accordingly, $PM_{2.5}$ become a hot spot of research interest about air pollution, replacing the TPS.

Although acts and standards were focused on the treatment of $PM_{2.5}$ outdoors, not indoors, the studies on indoor $PM_{2.5}$ have been increasing. For one thing, national studies reported that people spent most of their time indoors (Klepeis et al., 2001); for another, interior space of building may be considered as a safe place when outdoor air was severely

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contaminated. Therefore, the sources, component, effects of indoor $PM_{2.5}$ and the penetration of outdoor particles needed to be fully understood. Indoor $PM_{2.5}$ study started mainly in Euro-American countries in 1970s–1980s, earlier than China. Since the 1970s, numerous articles were published, most of which, however, were mainly for Environmental Tobacco Smoke (ETS) (Carrington, 2000; Kesteloot, 1986; Smoking, 1986; Sterling et al., 1982). The Harvard Six-City study began in 1979 and continued until 1988, which was conducted by the Harvard University School of Public Health and took measurements in at least 1400 homes. The New York State ERDA study started in a total of 433 homes in two New York State counties in 1986, conducted by Research Triangle Institute. The EPA Particle TEAM (PTEAM) Study was conducted in 178 homes in Riverside, California in 1990 by Research Triangle Institute and the Harvard University School of Public Health (Wallace, 1996). However, in developing countries such as China, indoor $PM_{2.5}$ research lags behind the United State as the representative of the Euro-American countries.

3.2. Extending of the concept "indoor"

In general, the concept of indoor refers to the airtight building space, typically including residences, office rooms, classrooms, libraries, supermarkets and cinemas, etc. However, because of the change of human behaviors and lifestyle, human activities have gone beyond the limit bound by the concept above. Correspondingly, indoor $PM_{2.5}$ research subsequently tends to be diverse. Hence, the concept of indoor is extended in this paper, after which it includes not only usually considered airtight architectural spaces, but also various non-building airtight spaces that are closely related to public life, typically including compartment of bus and train (Zhang and Li, 2012), aircraft cabin, isolation ward (Yorifuji et al., 2016a, 2016b), etc.

4. Review of published data

4.1. Sources of indoor $PM_{2.5}$

The sources of indoor $PM_{2.5}$ consist of indoor origins and outdoor infiltration (Fig. 2). outdoor $PM_{2.5}$ sources include crustal dust, vehicle emission (gasoline and diesel), coal combustion and some industry activities, such as steel plants, and the secondary particles produced by chemical reaction of primary particles in atmosphere from the outdoor sources listed above (De Kok et al., 2006; Dutton et al., 2009; Gao et al., 2016; Geng et al., 2013; Ho et al., 2006; Lemos et al., 2012; Pui et al., 2014; Wang et al., 2013a, 2013b). Due to air exchange between indoors and outdoors, chemical component of indoor $PM_{2.5}$ is associated with sources and component of outdoor $PM_{2.5}$. The principal indoor sources of $PM_{2.5}$ is smoking, cooking, fuel combustion for heating, human activities and burning incense (Table 1 and Fig. 2). Fig. 2 is a summary of indoor $PM_{2.5}$ studies published during 2000 and 2016.

Table 1 showed that $PM_{2.5}$ level in smoking facilities (zone) was significantly higher than it in nonsmoking facilities (zones) and outdoor environment. According to Morawska et al. (1997), 10 min after ETS generated, the initial ETS size distribution in an indoor environment peaked mainly between 60 and 90 nm. $PM_{2.5}$ level was closely related to levels of CO and other air pollutants, and smoking had a great influence on adjacent facilities (zones) (Liu et al., 2014). The concentration of indoor $PM_{2.5}$ was linked to season and construction type. The $PM_{2.5}$ concentration in heating season was clearly higher than it in non-heating season (Han et al., 2015; Zhu, 2012), exposure time of high $PM_{2.5}$ level in houses using clean energy was less than it in houses using coal and biomass (Li et al., 2016; Salje et al., 2014). $PM_{2.5}$ exposure level of indoor people caused by cooking and heating was relevant to fuel type, stove type, population group and cooking habits (Gurley et al., 2013; Li et al., 2016; Njenga et al., 2016; Zhang et al., 2014). Cooking was an important source of indoor particles, especially frying and grilling, and it released up to 10 times or more the level of $PM_{2.5}$ observed during non-cooking period (Hea et al., 2004; Wallace et al., 2004; Zhang et al., 2014). Incense was found to be a significant source of polycyclic aromatic hydrocarbons (PAHs), carbon monoxide, benzene, isoprene, $PM_{2.5}$ and PM_{10} (Bootdee et al., 2016; Fan and Zhang, 2001; Li and Ro, 2000). Lee and Wang (2004) observed that $PM_{2.5}$ emission rates of different incense types varied considerably. For the ten incense types in his experiment the $PM_{2.5}$ emission rates ranged from 9.8 to 2160.3 mg/h. Concentration change of indoor pollutants brought about by home decoration and high $PM_{2.5}$ exposure of women and children needed to be studied further. In addition, Table 1 showed that

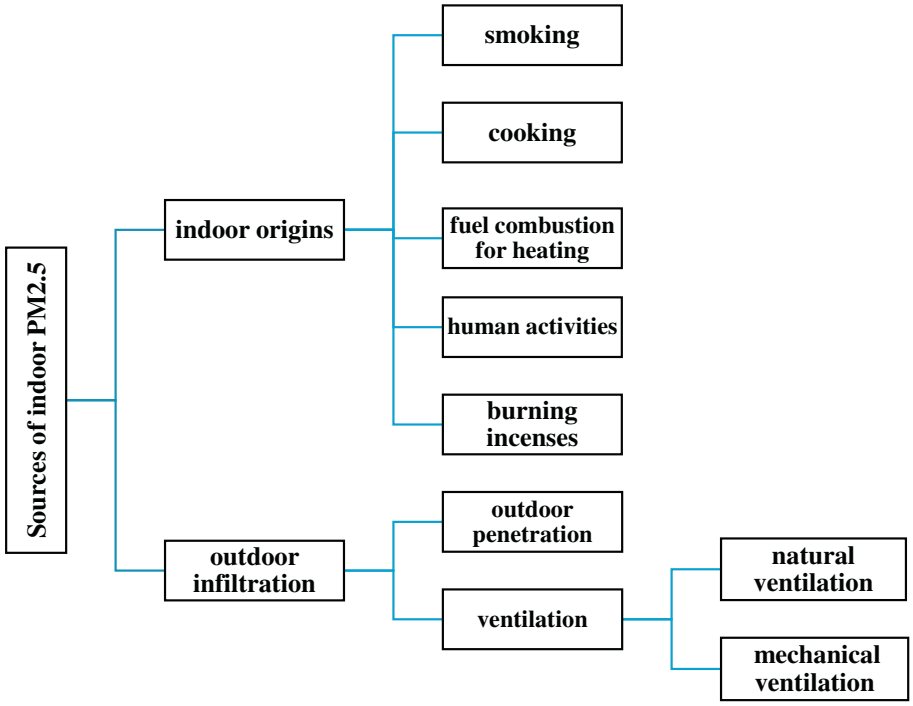

Fig. 2. Tree structure diagram of the indoor $PM_{2.5}$ sources.

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Table 1
A summary of some researches on indoor $PM_{2.5}$ published between 2000 and 2016.

City	Sampling time	Sampling site	Study subject	Partial study results	References
Birmingham	2014	Restaurant/bar	Smoke-permitted vs. smoke-free	$PM_{2.5}$ concentration: smoking (287 μg/m^3) > non-smoking (34 μg/m^3)	(Gurung et al., 2016)
Beijing	2010	Restaurant/bar	Smoke-permitted vs. smoke-free	SHS non-smoking: smoking sections (43 μg/m^3) > smoking facilities (40 μg/m^3) > nominal non-smoking sections (27 μg/m^3) > non-smoking sections (15 μg/m^3)	(Liu et al., 2014)
San Francisco	–	Residence	Smoke-permitted vs. smoke-free	$PM_{2.5}$ concentration: smoking (up to 630 μg/m^3) > non-smoking (up to 160 μg/m^3)	(Acevedo-Bolton et al., 2014)
Boston	2012	Multiunit housing	Smoke-permitted vs. smoke-free	Households within buildings with smoke-free policies showed lower $PM_{2.5}$ concentrations compared to buildings without these policies (median: 4.8 vs. 8.1 μg/m3); secondhand smoke transfer to smoke-free apartments was demonstrable with directly adjacent households.	(Russo et al., 2015)
Cairo	2005–2006	96 venues, including waterpipe etc.	Smoke-permitted vs. smoke-free	indoor $PM_{2.5}$ levels in venues where tobacco smoking was banned, places offering waterpipe to patrons of cafes, Ramadan tents and venues such as public buildings with poor enforcement of smoking restrictions was 72–81, 478, 612, 171–704 μg/m^{3}respectly; smokers contributed significantly to the overall $PM_{2.5}$ level.	(Loffredo et al., 2016)
Dhaka	2009	Residence	Wood	Each hour that $PM_{2.5}$ concentrations exceeded 100 lg/m^3 was associated with a 7% increase in incidence of ALRI[a] among children aged 0–11 months	(Gurley et al., 2013)
Montana	2010–2012	Residence	Wood	$PM_{2.5}$ concentration was 32.3 μg/m^3; $PM_{2.5-10}$ was significantly correlated with $PM_{2.5}$	(McNamara et al., 2013)
Xinzhou	2012–2013	Temple	Incense	$PM_{2.5}$ concentration was between 1.43 and 59.20 μg/m^3, which was 1.75–2.70 times higher than the above standard (25 μg/m^3).	(Wu et al., 2015)
Taiyuan	2005–2007	Residence	Solid fuel etc.	Indoor medium $PM_{2.5}$ concentration was 68 μg/m^3, and PM_{10} was 230 μg/m^3. PM levels in winter are strongly correlated with solid fuel usage for cooking, heating, and ventilators. PM_1 levels in cases are >3 times higher than that in control	(Mu et al., 2013)
Singapore	2009	School	Incense	During the Hungry Ghost Festival, many elements in $PM_{2.5}$ increased between 18% and 60%; the order of percentage increase in elemental Component was Zn > Ca > K > Mg…	(Khezri et al., 2015)
Lanzhou	2013	Residence	Natural gas/electricity vs. coal	Natural gas/electricity reduces $PM_{2.5}$ by 40%–70%; $PM_{2.5}$ concentration: heating season (125 μg/m^3) > non -smoking (80 μg/m^3)	(Li et al., 2016)
Dhaka	2009	Residence	Natural gas/electricity vs. biomass	Exposure time when $PM_{2.5}$ concentration exceeded1000 μg/m^3; biomass (66 min/d) > natural gas/electricity vs. biomass (35 min/d)	(Salje et al., 2014)
Kibugu	2014	Residence	Gasifier vs. traditional stoves and improved stoves	The gasifier domestic cooking system saved 27%–40% of fuel, reduced cooking time by 19%–23% and reduced emissions of $PM_{2.5}$ and CO by 40%–90%.	(Njenga et al., 2016)
Cajamarca Region	2009	Kitchen of in rural houses	Chimney stoves vs. traditional open fire stoves	Not statistically significant, but a post hoc stratification of chimney stoves by level of performance revealed mean $PM_{2.5}$ and CO levels of fully functional were 28% lower (136 μg/m^3) and 45% lower (CO, 3.2 ppm) in the kitchen environment compared with the control stoves ($PM_{2.5}$, 189 μg/m^3, CO, 5.8 ppm)	(Hartinger et al., 2013)
Porto	2013	Nursery	Regular service	$PM_{2.5}$ concentration was 158 μg/m^3, exceeding WHO standard by 80%; I/O ratio was over 1, often exceeding 2	(Branco et al., 2014)
Pennsylvania and Texas	2011 — 2012	Retail store	Regular service	$PM_{2.5}$ concentration was 11 μg/m^3; the contribution to $PM_{2.5}$ from indoor sources was 53%	(Zaatari and Siegel, 2014)
Guangzhou	2004	4 hospitals	Regular service	Indoor $PM_{2.5}$ was 99 μg/m^3,exceeding USEPA standard (65 μg m^{-3}) in 1997; 90% EC and 85% OC were found in $PM_{2.5}$; the correlation coefficient between indoor $PM_{2.5}$ and ambient $PM_{2.5}$ was 0.78	(Wang et al., 2006)
Milan	2011–2013	7 schools	Regular service	Both $PM_{2.5}$ and PM_{10} were more than the 24-h guideline values of WHO, indoor CO_2 often exceeded ASHRAE CO_2 limit, significant sources were determined.	(Rovelli et al., 2014)
Changsha	2014	A shopping mall	Regular service	Outdoor average $PM_{2.5}$ concentration at any time was higher than that of indoor. Indoor average $PM_{2.5}$; concentration on weekend were greater than that of weekday. Among different functional areas, the average $PM_{2.5}$ concentration of cosmetics area was the highest, followed by dining area, public walkway, clothes area and shoes & bags area	(Hu and Li, 2015)
Xian	2007–2008	Residence	Heating vs. no-heating (Summer vs. winter)	Indoor $PM_{2.5}$ concentration in winter and summer was 237.2 μg/m^3 and 96.7 μg/m^3, respectively. Biomass burning in summer was the dominant primary source for $PM_{2.5}$ (31% for indoor and 44% for outdoor), and those for winter were coal combustion (21% for indoor and 29% for outdoor) and biomass burning (24% for indoor and 16% for outdoor).	(Zhu, 2012)
Beijing	2013–2014	A three-bedroom apartment	Heating vs. no-heating	Indoor $PM_{2.5}$ concentrations were significantly correlated with outdoor $PM_{2.5}$ concentrations but with 1–2 h delay; Shorter lag time between indoor and outdoor $PM_{2.5}$ concentrations was found in heating period than non-heating period	(Han et al., 2015)
Shanghai	2013	Residence	Group A (unhealthy child living)vs. Group B (healthy child living)	$PM_{2.5}$ in child's bedroom of A and B was 221 μg/m^3 and 182 μg/m^3, respectively Indoor $PM_{2.5}$ and PM_{10} concentrations in all investigated houses exceeded the Chinese national standards; SVOC in house dust of four living rooms showed very high concentrations with 3 to 4 times the EU limit.	(Zhang et al., 2016)
Los Angeles	–	Experiment chamber	Popcorn vs. water (microwaving)	$PM_{2.5}$ generated by popcorn was 249 μg min^{-1}; UFPs and $PM_{2.5}$ generated by microwaving popcorn were 150–560 and 350–800 times higher than the emissions from microwaving water, respectively	(Zhang et al., 2014)

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Table 1 (*continued*)

City	Sampling time	Sampling site	Study subject	Partial study results	References
Kavala	2016	Hospital	Regular service	Indoor $PM_{2.5}$ 24-h ranged from 10.16 μg/m^3 to 21.78 μg/m^3 in the lab and from 9.86 μg/m^3 to 26.27 μg/m^3 in the triage, exceeding the health limit 25 μg/m^3	(Loupa et al., 2016)

[a] ALRI is short for acute lower respiratory infection; 2) SHS is short for secondhand smoking.

homes (kitchens and bedrooms), classrooms, shisha facilities, restaurants and bars, hospitals, child care facilities and retail stores were exposed to $PM_{2.5}$ pollution. Hence research scope of indoor $PM_{2.5}$ needed to be extended further.

4.2. Component of indoor $PM_{2.5}$

In general, the component was same in various indoor environment, although there may be little variance and it may differs significantly in proportion due to different types of indoor environment (Bootdee et al., 2016; Coombs et al., 2016; McNamara et al., 2013; Rovelli et al., 2014; Russo et al., 2015; Wang et al., 2006). According to several studies (De Kok et al., 2006; Geng et al., 2013; Lemos et al., 2012), the main components of $PM_{2.5}$ were organic compounds, inorganic compounds, biological components and carbon. Organic compounds mainly included saturated and unsaturated hydrocarbon, such as volatile organic compounds (VOCs) and PAHs. Inorganic compounds were mainly water-soluble inorganic salts and inorganic elements; the former were primarily nitrate, ammonium and sulfate, the latter were S, Br, Cl, As, Cs, Cu, Pb, Zn, Al, Si, Ca, P, K, V, Ti, Fe and Mn etc. Biological components primarily consisted of bacteria, fungi, viruses, pollen and plant fibers. Carbon comprised elemental carbon (EC) and organic carbon (OC).

4.3. Effects of indoor $PM_{2.5}$

4.3.1. Influence factors

The influence of $PM_{2.5}$ was associated with concentration, component, size, human age and exposure time length of $PM_{2.5}$ (Gurley et al., 2013; Han et al., 2015; Lemos et al., 2012). According to Samoli et al. (2013), when cumulative lag were 0–1, 2–5, 0–5 days, respiratory mortality increased 0.72% (95% CI: 0.11%–1.55%), 1.63% (95% CI: 0.62%–2.65%) and 1.91% (95% CI: 0.71%–3.12%) respectively with a 10-μg/m^3 increase in $PM_{2.5}$. The longer the cumulative lag, the greater the adverse impact of $PM_{2.5}$ to respiratory mortality. Some $PM_{2.5}$ samples in a triage room of an emergency department were collected and organic compounds, such as Dehydrocholate, hydrocortisone acetate and γ toad poison, were found, which may result in different health risks for members in hospital (Samoli et al., 2013). There is a general consensus that the smaller the size of particles, the greater the health effects. Furthermore, a study indicated that the percentages of carcinogenic PAHs and metals (transition metals) were greater in smaller particulate matters (Dacunto et al., 2014); it was reported that $PM_{0.1}$ was able to reach the alveolar parenchyma (parenchyma) and infiltrate pulmonary Interstitial tissue, making $PM_{0.1}$ more pathogenic (Donaldson and Borm, 2004). Moreover, some studies showed that $PM_{2.5}$ with concentration lower than national limit still damaged health (Yorifuji et al., 2016a, 2016b).

4.3.2. Comparison between $PM_{2.5}$ and PM_{10}

For one thing, compared with PM_{10}, $PM_{2.5}$ smaller in size owned larger specific surface area and bigger adsorption ability, thus toxic heavy metals was more easily bound to $PM_{2.5}$ (Fig. 3) (Widziewicz and Loska, 2016), and so did acid oxides, organic pollutants and pathogenic microorganisms. A research demonstrated that the percentages of trace metal (transition metal) elements and carcinogenic PAHs in $PM_{2.5}$ were almost 2 times the percentage of them in PM_{10} (Dacunto et al., 2014). The results of a study indicated that the survival rate of rat macrophages (MAC) dealt with $PM_{2.5}$ and PM_{10} with concentration 150 μg/ml was 85.4% and 89.9%, respectively, which indirectly demonstrated $PM_{2.5}$ had a greater impact on human health than PM_{10} (Dacunto et al., 2014). Another, in comparison with PM_{10}, $PM_{2.5}$ was lighter, contributing to a higher morbidity and a higher deposition rate in pulmonary (Fig. 4) (Widziewicz and Loska, 2016), thus $PM_{2.5}$ stayed longer in human respiratory tract fora. It was reported that at least 60% of $PM_{2.5-10}$ would deposit in the outer part of the chest. Particles with a size ranging from 1 to 2.5 μm mainly deposited in bronchial and alveolar, part of which stayed in the lung tissue for a long time and formed the focus in pulmonary interstitial tissue. $PM_{0.1}$ could invade alveolar and stay in it, then quickly entered blood circulation system by respiration, finally flowed into human kidney, liver, heart, brain and other organs. In conclusion, $PM_{2.5}$ did more harm to health than PM_{10} (Elsevier Science Ltd., 1994).

4.3.3. Epidemiological study of $PM_{2.5}$

The epidemiological studies date from the end of 1980's, which primarily focus on the relationship between long-term or short-term $PM_{2.5}$ exposure and morbidity or mortality (Respiratory diseases, CVD, etc.). Tables 2 and 3 presented the association between short-term exposure and effects in a form of percentage increase of morbidity and mortality with a 10-μg/m^3 increase of $PM_{2.5}$. It should be paid attention to that only papers published during 2000 and 2016 were selected for the Tables 2 and 3.

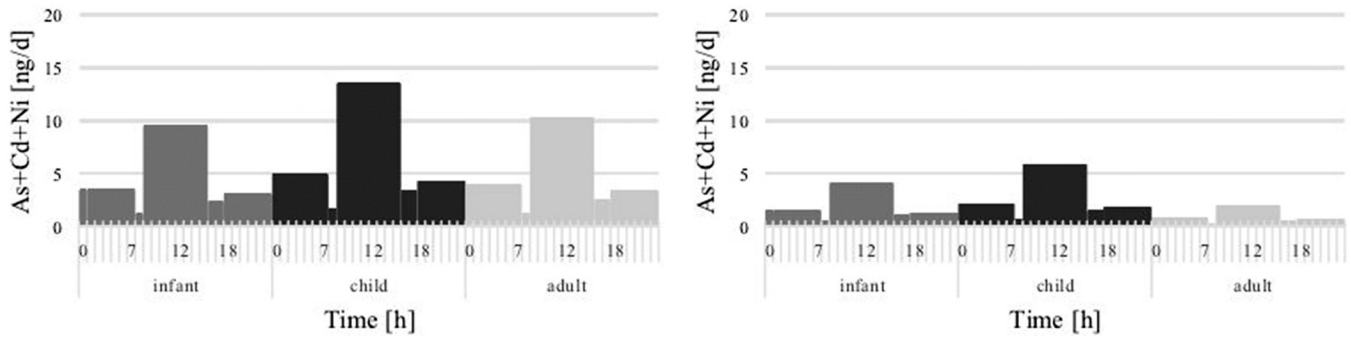

Fig. 3. Daily inhaled mass of metals bound to $PM_{2.5}$ and PM_{10} for Bielsko-Biala (B).

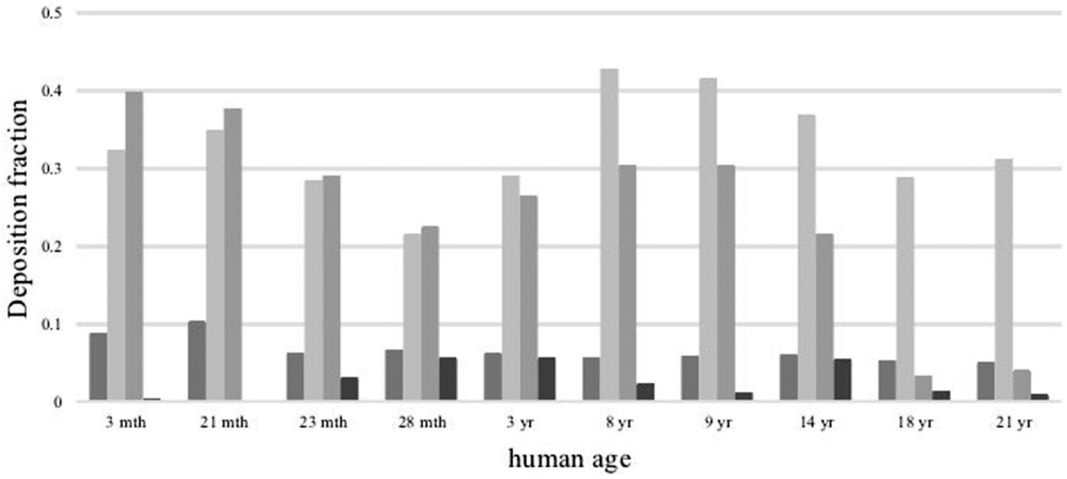

Fig. 4. Averaged regional deposition in tracheobronchial (TB) and pulmonary (P) of $PM_{2.5}$ and PM_{10}.

In Tables 2 and 3, it was easily known that mortality and morbidity of CVD and respiratory diseases rose as the $PM_{2.5}$ concentration increased, and the relationship between short-term exposure and health effects was affected by age. In general, morbidity was more sensitive in reflecting the influence of air pollution on human health, and outpatient emergency treatment rate and admission rate were always served as an index of incidence rate, so outpatient rate was included in Table 3. Furthermore, it was also concluded that mortality generated by short-time $PM_{2.5}$ exposure was influenced by season, region (urban and rural) and co-pollutants (Samoli et al., 2013). Now some researches indicated genetic variation as well as mortality of diabetes was linked with $PM_{2.5}$ exposure (Fromme et al., 2007; Quirós-Alcalá et al., 2016). The loss caused by $PM_{2.5}$ exposure also could be characterized by DALYs (Disabled -adjusted life years) and YLL (Year of lost life).

4.4. Toxicological study of $PM_{2.5}$

There were not toxicology studies in china as many as Euro-American countries. Now there was no consensus in academia about specific pathogenic mechanism of $PM_{2.5}$ to respiratory system and CVD. However, what was widely accepted were inflammatory reaction, oxidative stress, genetic toxicity, mutagenicity and carcinogenicity.

4.4.1. Inflammatory reaction

MAC of alveolus stimulated by $PM_{2.5}$ synthesized and secreted inflammatory mediators, such as tumor necrosis factor (TNF-a) and nuclear factor (NF-κB), breaking the balance between human inflammation and anti-inflammation, which results in inflammation. Some medical experts indicated that increased level of active oxygen (ROS) and inflammation were probably ones of the important mechanisms by which $PM_{2.5}$ destroyed Endothelial Progenitor Cells (EPCs). Endothelial dysfunction or injury were considered as main reasons of CVD (Cui et al., 2016). Several investigation demonstrated that Pb, Al, Cu, and transition metals etc. would cause inflammation and pulmonary fibrosis and other pulmonary diseases (Prieditis and Adamson, 2009; Varshney et al., 2016).

4.4.2. Oxidative stress

Active free radicals in $PM_{2.5}$ and reactive oxygen species (ROS) and active nitrogen from stimulated MAC and epithelial cells activated the target cell oxidation pathway, eventually causing inflammation and other effects (González-Flecha, 2004). Pathogenic mechanism of oxidative stress included both approaches: the one was gene mutation through gene damage; the other was destroying cell membrane and altering the membrane permeability through lipid peroxidation, which contributed to physiological changes, such as airway inflammation etc. (Baulig et al., 2003). In addition, a researcher showed that excessive free radicals would attack the cell biological macromolecules, damaging the cells (Szigeti et al., 2016).

4.4.3. Genetic toxicity, mutagenicity and carcinogenicity

Heavy metals and PAHs damaged the double helix structure of DNA alone or by synergistic action, resulting in gene mutation. MAC stimulated by $PM_{2.5}$ released cytokines, such as growth factor, which changed the cell cycle and made cells keep dividing forever. Consequently, tumor formed. There was an investigation confirming that in the air of Beijing existed particulate PAHs, such as bingo pyrene and all air samples had stronger mutagenicity, in which frameshift mutation was the main (Silva Da Silva et al., 2015); Several research workers pointed out that PM was able to drive normal cells deteriorate into cancer cells by altering cells function and inhibiting apoptosis (Chang et al., 2011); it was found in an academic report that Cr and Pb were carcinogenic (Varshney et al., 2016).

4.5. Indoor $PM_{2.5}$ control

4.5.1. Standards of indoor $PM_{2.5}$

The $PM_{2.5}$ study in china is relatively later. Previous standards such as Indoor Air Quality Standard only provided limit for PM_{10}. The Standard of the Measurement and Evaluation for Efficiency of Building Ventilation (JGJ/T309-2013), issued on July 2nd, 2013, came into effect on February 1st, 2014. This standard is applicable to the test and evaluation of ventilation effect of civil buildings and requires the daily average concentration of indoor $PM_{2.5}$ should be $<75\ \mu g/m^3$. The latest Ventilation for Acceptable Indoor Air Quality (ANSI/ASHRAE62.1-201), issued by the United States, was put into effect on August 21, 2015. This act provides for indoor $PM_{2.5}$ with a concentration $<15\ \mu g/m^3$. Based on long-term monitoring, it was found that whether there existed smoker had a significant effect on indoor $PM_{2.5}$ concentration. Hence, The Residential Indoor Air Quality Guideline issued by Canada in 2012 defined $PM_{2.5}$ as a monitoring object, but it didn't provided specific $PM_{2.5}$ limit. In Indoor Air Quality Guidelines-Implementation Outline of Domestic Fuel, WHO pointed out that the $PM_{2.5}$ limit for burning domestic fuel with ventilation and without ventilation was 230 $\mu g/m^3$ and 800 $\mu g/m^3$, respectively. It also set the target emission rate of $PM_{2.5}$ for domestic fuel and its recommended strength was strong. This guideline showed that controlling $PM_{2.5}$ emission resulting from the combustion of domestic fuel was of great significance.

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Table 2
Estimated relative risk of mortality associated with each 10 μg/m^3 increase in $PM_{2.5}$ in the research published during 2000−2016.

Study	Time	$PM_{2.5}$[a]/μg/m^3	Lag time	Evaluation index	Relative risk (95% CI)	Reference
A population-Based Study in New England	2003–2008	8.21 (5.10)	0–1	Total disease mortality	2.14% (1.38%−2.89%)	(Shi et al., 2016)
Tokyo mortality study	2002–2013	Median 16.7	0–1	Total mortality of infants Mortality of the infant below 1 years old	1.06% (1.01%–1.21%) 1.10% (1.02%–1.19%)	(Yorifuji et al., 2016a, 2016b)
A time series studies in Austrian Cities	1990–2007	22.4	0–1	Total disease mortality CVD mortality	0.50% (−0.7%–1.50%) 0.1% (−1.5%–1.7%)	(Neuberger et al., 2013)
A time series studies in Austrian Cities	1990−2007	16.3	0–1	Total disease mortality CVD mortality	0.5% (−0.6%–1.5%) 0.4% (−1.0%–1.8%)	(Neuberger et al., 2013)
A case-crossover study in Three Southeastern states	2007–2011	11.1 (4.4)	0–1	Total disease mortality CVD mortality	1.56% (1.19%–1.94%) 2.32% (1.57%–3.07%)	(Lee et al., 2016)
A mortality study in Mediterranean Cities	2001−2010	13.6−27.7	0–1	Total disease mortality CVD mortality Respiratory disease mortality	0.55% (0.27%–0.84%) 0.57% (0.07%–1.08%) 0.72% (−0.11%–1.55%)	(Samoli et al., 2013)
A multicity time-series study in East Asia	2000–2009	Median 17.7−70.1	0–1	Total disease mortality CVD mortality Respiratory disease mortality	0.38% (0.21%–0.55%) 0.96% (0.46%–1.46%) 1.00% (0.23%–1.78%)	(Lee et al., 2015)
A time-series study in Shanghai	2007–2008	53.9 (31.4)	0–3	Total disease mortality CVD mortality Respiratory disease mortality	0.57% (0.12%–1.03%) 0.07% (−1.27%–1.41%) 0.79% (0.1%–1.46%)	(Wang et al., 2013a, 2013b)
An ambient air pollution study in Ningbo	2011–2013	60.1	0–3	Total disease mortality	0.57% (0.20%–0.95%)	(He et al., 2016)
A time series studies in Beijing	2004–2009	76 (56)	0–3	Respiratory disease mortality	0.69% (0.54%–0.85%)	(Li et al., 2013)
A time series study in Hong Kong	2001–2010	37.8 (22.5)	0–5	Total disease mortality	1.02% (1.01%–1.03%)	(Tam et al., 2015)
An ecological longitudinal time-series study in Madrid	2003–2005	19.16 (8.64)	0–2 0–6	Total circulatory system mortality	1.022% (1.005%–1.039%) 1.025% (1.007%–1.043%)	(Maté et al., 2010)
An Extended Follow-up of the Harvard Six Cities Study	1979–1998	15 (ranging 10–22)	8 years	All-cause mortality Cardiovascular mortality	16% (7%–26%) 28% (13%–44%)	(Laden et al., 2006)
An Extended Follow-up of the Harvard Six Cities Study	1974–2009	16 (ranging 11–24)	32 years	All-cause mortality Cardiovascular mortality Lung-cancer mortality	14% (7%–22%) 26% (14%–40%) 237% (7%–75%)	(Lepeule et al., 2012)
American Cancer Society (ACS) study	1982–1998	18 (9–34)	–	All-cause mortality	26% (8%–47%)	(Pope et al., 1995)
ACS study	1982–1998	18 (4)	–	All-cause mortality Cardio-pulmonary mortality Long cancer mortality	6% (2%–11%) 9% (3%–16%) 14% (4%–23%)	(Pope et al., 2002)
ACS sub-cohort study	1982–2000	(−9–27)	18 years of follow-up	All-cause mortality Cardio-pulmonary mortality	17% (5%–30%) 26% (1%–60%)	(Jerrett et al., 2005)
Women's Health Initiative Observational Study	1994–1998	14 (3–28)	6 years of follow-up	Cardiovascular mortality	76% (25%–147%)	(Miller et al., 2007)
Netherlands cohort study	1987–1996	28 (23–37)	–	All-cause mortality Cardio-pulmonary mortality	6% (−3%–16%) 4% (−10%–21%)	(Beelen et al., 2008)
Nurses' health study	1992–2002	14 (6–28)	–	All-cause mortality	26% (2%–54%)	(Puett et al., 2008)
Medicare national cohort	2000–2005	13 (4)	–	All-cause mortality	4% (3%–6%)	(Zeger et al., 2008)
California teachers study	2002–2007	18 (7–39)	–	All-cause mortality Cardio-pulmonary mortality	6% (−4%–16%) 19% (5%–36%)	(Ostro et al., 2010)
Health professionals follow-up study	1989–2003	18 (3)	–	All-cause mortality Cardiovascular mortality	−14% (−28%–2%) 3% (−17%–26%)	(Puett et al., 2011)
Vancouver cohort	1999–2002	4 (0−10)	–	Cardiovascular mortality	7% (−14%–32%)	(Beelen et al., 2008)
US trucking industry cohort	1985–2000	14 (4)	–	All-cause mortality Cardiovascular mortality	10% (3%–18%) 5% (−7%–19%)	(Hart et al., 2011)
A Canadian national cohort	1991–2001	9 (2–19)	–	All-cause mortality Cardiovascular mortality	10% (5%–15%) 15% (7%–24%)	(Crouse et al., 2012)
Italian a population-based cohort	2001–2010		9 years of follow-up	Non-accidental mortality	1.04% (1.03%–1.05%)	(Cesaroni et al., 2013)

[a] Mean with minimum - maximum in parentheses (μg/m^3). One number in parentheses is standard deviation.

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Table 3
Estimated relative risk of morbidity associated with each 10 μg/m^3 increase in $PM_{2.5}$.

Study	Time	$PM_{2.5}$ (SD)/μg/m^3	Lag time	Evaluation index	Relative risk (95% CI)	Reference
A Case-Crossover Study in Kaohsiung	2006–2010	31.65	0	CVD morbidity	1.01% (1.00%–1.03%)	(Chen et al., 2015)
A Population-Based Study in Beijing	2013	102.1 (73.6)	0	Total respiratory disease morbidity Upper respiratory tract infection morbidity Lower respiratory tract infection morbidity	0.23% (0.11%–0.34%) 0.19% (0.04%–0.33%) 0.34% (0.14%–0.53%)	(Xu et al., 2016)
A time series studies in Austrian Cities	2000–2007	20.5	0	Total cardiopulmonary disease morbidity CVD morbidity Total respiratory disease morbidity	0.1% (−1.4%–1.7%) 0.3% (−1.8%–2.4%) −1.6% (−5.2%–2.0%)	(Neuberger et al., 2013)
A diseases study in Doña Ana County	2007–2010	10.9	0	CVD morbidity (emergency) Total respiratory disease morbidity (emergency) CVD morbidity (hospitalization) Total respiratory disease morbidity (hospitalization)	4.5% (−3.2%–12.9%) 3.0% (−2.1%–8.3%) −1.4% (−6.2%–3.6%) 1.3% (−5.0%–8.0%)	(Rodopoulou et al., 2014)
A case-crossover study in Mid-Atlantic States	2000–2006	11.92 (5.68)	0–1	CVD morbidity Total respiratory disease morbidity	0.78% (0.5%–1.0%) 2.2% (1.9%–2.6%)	(Kloog et al., 2014)
A case-crossover design in Fukuoka	2005–2010	20.3 (11.2)	0–1	Total morbidity Total respiratory disease morbidity	1.008% (1.00%–1.014%) 1.02% (1.007%–1.05%)	(Michikawa et al., 2014)
A cohort study in Santiago	1995–1996	52.0 (31.6)	0–1	Infant below 1 year old respiratory illnesses (wheezing bronchitis)	5% (0–9%)	(Pino et al., 2004)
An adult's health study in Arkansas	2000–2012	12.4 (5.9)	0−2	CVD morbidity Total respiratory disease morbidity	1.52% (−1.1%–4.2%) 1.45% (−2.64%–5.72%)	(Rodopoulou et al., 2015)
A time-series study in Shanghai	2007–2008	53.9 (31.4)	0–3	Outpatient rate	0.45% (0.16%–0.73%)	(Wang et al., 2013a, 2013b)
A time series studies in Beijing	2004–2009	76 (56)	0–3	Total respiratory disease morbidity	1.32% (1.02%–1.61%)	(Li et al., 2013)
A study about pneumonia in Hong Kong	2011–2012	30.88 (16.79)	0–3	Emergency hospital admissions for pneumonia	1.15% (0.46%–1.84%)	(Qiu et al., 2014)
A study about pneumonia in Hong Kong	2011–2012	30.88 (16.79)	0–4	Emergency hospital admissions for pneumonia	1.47% (0.80%–2.14%)	(Qiu et al., 2014)
A time series study in Hong Kong	2001–2010	37.8(22.5)	0–5	Hospitalization rate	1.02% (1.01%–1.02%)	(Tam et al., 2015)
The nurses' health (NSH)study	1989–2006 2000–2006	8.7 (4.5)a 7.3 (4.1)a	1 year 0f fellow-up	CVD morbidity of women with diabetes	1.02% (0.94%–1.10%), 1.11% (0.96%–1.29%)b	(Hart et al., 2015)

4.5.2. Control strategies

Good indoor air quality is one of prerequisites for green building. In order to ensure the healthy and comfortable indoor environment, reasonable measures should be used to control indoor $PM_{2.5}$ concentration. According to the sources of indoor $PM_{2.5}$, control strategies can be divided into two categories: sources control and control during transmission.

4.5.2.1. Sources control. Control in source should be carried out in two aspects since indoor $PM_{2.5}$ was from not only indoor environment but also outdoor atmosphere. For outdoor $PM_{2.5}$ control, Tables 4–6 presents the control targets of WHO, China, the United States and European Union (UN, 2008).

Tables 4–6 showed that WHO Air Quality Guideline (AQG) (WHO, 2005) was the most stringent among the standards, followed by the European Union. The control targets of America and China were below the Interim target-1 and the second standard of China was equal to the WHO Interim targets-1, which suggested that Chinese $PM_{2.5}$ control lagged behind America and world average.

The strategies to control outdoor $PM_{2.5}$ were as follows:

- Reducing dust emission from land and buildings comes first. One the one hand, government should ensure urban greening to reduce the land uncovered and keeps it up with the urban expansion and infrastructure construction. Urban greening on both sides of road should be combined with road hardening to avoid re-entrainment of fugitive dust. Besides, the selection of green vegetation also ought to be combined with air purification. On the other hand, underground pipe, like drainage facilities, ought to be designed as rationally as possible to avoid silt that results from rain or snow. During infrastructure construction, it is the construction units' duty to set up continuous containment facilities in order to prevent sand or gravel from being uncovered. Dust control is also required for building materials and waste during transportation, the vehicles used also need to be cleaned.
- Then, vehicles emission must be controlled. Firstly, the emission standard of vehicles must be implemented strictly. Secondly, the petroleum refining upgrade should be accelerated to prepare for improvement of gasoline and diesel used by vehicle. Thirdly, the new energy vehicle

Table 4
WHO air quality guidelines and interim targets for $PM_{2.5}$[a].

	Statistical method	$PM_{2.5}$[b] ($\mu g/m^3$)	Basis for the selected level
Interim target-1 (IT-1)	Annual mean concentration	35	These levels are associated with about a 15% higher long-term mortality risk relative to the AQG level
	24-h concentration	75	Based on published risk coefficients from multi-centre studies and meta-analyses (about 5% increase of short-term mortality over the AQG value).
Interim target-2 (IT-2)	Annual mean concentration	25	In addition to other health benefits, these levels lower the risk of premature mortality by approximately 6% (2%–11%) relative to the IT-1 level
	24-h concentration	50	Based on published risk coefficients from multi-centre studies and meta-analyses (about 2.5% increase of short-term mortality over the AQG value).
Interim target-3 (IT-3)b	Annual mean concentration	15	In addition to other health benefits, these levels reduce the mortality risk by approximately 6% (2%–11%) relative to the IT-2 level
	24-h concentration	37.5	Based on published risk coefficients from multi-centre studies and meta-analyses (about 1.2% increase in short-term mortality over the AQG value).
Air Quality Guideline (AQG)	Annual mean concentration	10	These are the lowest levels at which total, cardiopulmonary, and lung cancer mortality have been shown to increase with >95% confidence in response to long-term exposure to $PM_{2.5}$
	24-h concentration	25	Based on relationship between 24-hour and annual PM levels.

As with US, Chinese standard for indoor $PM_{2.5}$ has been gradually strengthened over the years.

[a] 99th percentile (3 days/year).

[b] For management purposes, Based on annual average guideline values; precise number to be determined on basis of local frequency distribution of daily means. The frequency distribution of daily $PM_{2.5}$ or PM_{10} values usually approximates to a log-normal distribution.

technology ought to be promoted continuously, combined with further development of public transportation.

- Lastly, industrial emission must be cut down. Before combustion, coal should be desulfurized coal-fired. Boilers with 30 t/h ought to be eliminated as soon as possible. In north China central heating is supposed to be generalized. At the same time, highly efficient pulverized coal technology and briquettes need to be promoted. Moreover, natural gas should be utilized more widely in industry, as well as renewable energy, such as hydro energy, solar energy, geothermal energy, wind energy and nuclear energy, and so on.

It is to meet the requirements for fresh air that there exists air exchange between indoor environment and outdoor environment. In terms of outdoor infiltration, the indoor $PM_{2.5}$ control strategies are presented below: when outdoor $PM_{2.5}$ concentration is high, close doors and windows. If not, open the doors and windows to dilute indoor $PM_{2.5}$. The key of the method is real-time monitoring to indoor and outdoor $PM_{2.5}$. By improving air distribution, raising ventilation rate, fresh air (filtered) volume and regularly cleaning and disinfecting air-conditioning system, etc. indoor $PM_{2.5}$ can be decreased in buildings that have been already fixed centralized or semi-centralized air-conditioning system with fresh air system. Some experts thought that for public buildings, air-cleaning facilities could be equipped in existing fresh air units, return air inlet and blast pipe, air cleaners also could be used directly there.

Table 5
$PM_{2.5}$ mass concentration limits in GB 3095-2012 of china and in national ambient air quality standards in 2012 of America.

	Primary standards		Second standards	
	Annual mean concentration	24-h mean concentration	Annual mean concentration	24-h mean concentration
China[a]	15	35	35	75
America[b]	12	35	15	35

[a] Primary standards of china is applicable to nature reserves, scenic spots and other areas in need of special protection; Second standards of china is applicable to Residential areas, mixed area of commercial areas, transportation and residential areas, cultural areas, industrial areas and rural areas.

[b] Primary standards of America are aimed at public health protection, including Asthma patients and the sensitive population, such as children and the elderly; Second standards of America are aimed at social material wealth protection, including visibility, animals, crops, vegetation, buildings, etc.

The strategies to control indoor $PM_{2.5}$ were as follows:

- The first is to control smoking. The government and relevant departments should actively promote smoking ban to improve public health awareness. Smoking zones and non-smoking zones need to be set up in public area, such as train stations and restaurants. Function zone is required to be divided reasonably to avoid cross contamination. In a research it was demonstrated that five to seven nonsmokers who worked in the bar were saved each year by smoking ban in Ontario, saving 5–6.8 million Canadian dollar (namely, 4.9–6.6 million American dollars) (Repace et al., 2013).
- The second is to control solid fuels usage. Ventilation of kitchen should be strengthened and it may be better that local meteorological characteristics are taken into consideration when functional division of a house was implemented. Then, traditional solid fuels, like straws, coal, wood and asphalt blocks, must be reduced as much as possible, combined with clean fuels encouragement by government. Clean fuels refer to natural gas, methane and electricity. Stove ought to be optimized to maximize efficiency. Gasifiers as well as ventilated stove should be promoted. Cooking habits (patterns) must be changed and the operation and maintenance of stove need to be improved. Moreover, the significance of indoor air quality ought to be aware by more people. There was an investigation finding that granular biomass could decrease primary $PM_{2.5}$ by 79%–85% emitted by biofuels and $PM_{2.5}$ emission could be reduced be 60%–68% by replacing the original coal briquettes by coal cakes (Shen, 2016). In addition, $PM_{2.5}$ emission of bituminous coal combustion was cut down by 54%–67% by means of replacing traditional stoves by optimized stoves. In comparison with low efficiency stoves, $PM_{2.5}$ emission was reduced by 76% by using optimized stoves, and 95% by using gasifiers. What's more, a research demonstrated that the maintenance and functional level of stove was also worth considering, besides stove type. (Hartinger et al., 2013).
- The third is to arrange indoor activities reasonably. It's a good habit for people to clean house regularly, select decoration reasonably, try not to keep pets, burn incenses less and increase indoor greening etc. All of these methods do help to lower indoor $PM_{2.5}$ concentration. A study indicated that improving ventilation rate combined with timely cleaning after school decreased the PM concentration in a nursery from (79 ± 22) $\mu g/m^3$ to (64 ± 15) $\mu g/m^3$, fully demonstrating that indoor cleaning could control PM greatly (Branco et al., 2014).

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Table 6
Standards and limit value of $PM_{2.5}$ in Directive 2008/50/EU.

	Statistical method	Concentration (μg/m^3)	Requirement
Limit value	Annual mean concentration	25	Be effective on January 1, 2015
	24-h mean concentration	–	
Target value	Annual mean concentration	20	Be effective in 2015
	24-h mean concentration	–	

Exposure limit value		
AEI basic concentration range(μg/m^3)	Target reduction percentage	Target completion will be assessed in 2020
≤8.5	0	
8.5–13	10%	
13–18	15%	
18–22	20%	
≥22	Reduce to 18 μg/m^3 by feasible method	

4.5.2.2. Control during transmission. The employment of air conditioning with excellent filtration performance or air purifiers has been the main control method in $PM_{2.5}$ transmission process. Now air purification equipment chiefly involve air freshener, ultrasonic atomizer, indoor water features and plants, such as *Chlorophytum comosum* and Scindapsus aureus that have great absorption property for $PM_{2.5}$ and some harmful gaseous pollutants. At present, non-woven fabric with primary efficiency has been commonly used in household air conditioning, while medium-high efficiency filter is often used in the air conditioning unit of clean room. Therefore, high efficiency particulate air filter (HEPA) may be useful in residential air conditioning to increase feasibility of indoor $PM_{2.5}$ removal. Since the filtration efficiency of HEPA for particles with a diameter of 0.3 μm is up to 99.9%, indoor $PM_{2.5}$ can be substantially removed (Price et al., 2005). However, in the meantime the equipment resistance was correspondingly increased by HEPA. Heudorf et al. (2009) summarized that the purification technology currently employed in various brands of air filters involved filtration, absorption, catalysis, electrostatic interaction and plasma technology, and so on. The utilization rate of the combination of filtration and absorption was as high as 100%, namely almost all air filters utilized these two purification technologies. He also pointed that the purifiers' removal efficiency for $PM_{2.5}$ was above 90%.

5. Conclusion

High indoor $PM_{2.5}$ concentration had a direct impact on human health. According to sources, components, health effects of indoor $PM_{2.5}$ mentioned above, it could be concluded that indoor environment was heavily polluted with complex sources. Although domestic study on indoor $PM_{2.5}$ was launched later, follow-up development was rapid. However, there are a lot of relevant problems to be further studied. Firstly, in china high-rise buildings and super high-rise buildings have been increasing, of which the airtightness has become better. Meantime, due to outdoor air frequently polluted, people prefer not to open the window. Unfortunately, the public isn't aware of this trend as a whole. Given that indoor air quality don't match buildings function and in china no detailed standards about indoor $PM_{2.5}$ is accessible for reference, the general awareness about indoor air quality should be improved and government and industry should formulate detailed control standards on the base of abundant investigation as soon as possible. Secondly, research scope of indoor $PM_{2.5}$ needs to be broadened. Now most of current researches focus on cities and towns and very little is on rural areas. But it was reported on WHO Indoor Air Quality Guideline in 2014 that over the world about 3 billion the poorest of the population used solid fuels for cooking and heating in stoves with low efficiency and high pollution. On account of inadequate study for a variety of airtight space, including urban residences, offices, commercial establishments, cultural and educational establishments and public transportation space, statistical analysis can't be conducted with a large number of data, which undoubtedly hinders the development of indoor $PM_{2.5}$ study. Last but not least, indoor $PM_{2.5}$ study needs to be deepened. So far adverse health effects have been determined, but the specific mechanism, contribution of particles in different sizes and of different components and the synergistic effects of other air pollutants still remain largely undetermined. Moreover, the relationship between $PM_{2.5}$ exposure and some diseases needs to be clarified, the short-term and long-term exposure and the effect of high efficiency filtration facilities in the actual use process also need to be studied further.

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