

A Global Perspective of Fine Particulate Matter Pollution and Its Health Effects

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Abbreviations

CI	Confidence interval
CPCB	Central Pollution Control Board
HR	Hazard ratio
IQR	Interquartile range
NAAQS	National ambient air quality standard
OR	Odds ratio
PI	Posterior interval
PM	Particulate matters
PM _{2.5}	Particulate matters of 2.5 μm size or less
WHO	World Health Organization

1 Introduction

The particulate matter (PM) pollution is a global burden, which not only affects the physical atmosphere, but also has drastic effects on human civilization since the beginning of anthropogenic interferences to the natural ecosystems. The greatest concern lies over the negative impact on human health as exposure to ambient PM pollution is ranked 12th among the Global DALYs (disability-adjusted life-years) risk factors according to Global burden of disease study in 2013 (Forouzanfar et al. 2015). An aerosol is a suspension of a solid or liquid particle in the air. For health purposes, aerosol or particulate matter (PM) is typically defined by size, with the smaller particles having more health impacts. Particles with a diameter $<10 \mu\text{m}$ are called PM₁₀ and with a diameter $<2.5 \mu\text{m}$ PM_{2.5}.

Climate change combined with air pollution reduces the healing capacity (intrinsic ability to restore back to its earlier state in time) of natural systems, and causes extreme variability (variations over a wide range) leading to more serious health effects (Fig. 1). PM in air is cosmopolitan in distribution in all types of ecosystems from desert to oceans. PM has both direct and indirect effects on the earth by both cooling and heating of the atmosphere. Aerosols in the atmosphere impact both climate and biogeochemistry in the earth's surface after deposition (Von Schneidmesser et al. 2015). It is estimated that anthropogenic changes in aerosols contribute to $\sim 40.0\%$ change in short wave radiative forcing and $\sim 60.0\%$ increase in the number of Cloud Condensation Nuclei (CCN), the small particles on which water vapor condenses (Mahowald et al. 2011). Aerosols have a short atmospheric lifetime (about a week), so their effects are more evident than greenhouse gases (GHGs) (Kopp and Mauzerall 2010). Most PM occurs naturally in the environment as desert dust, forest fire, sea salt, and sulfates from volcanoes, but increasing anthropogenic interferences in the environment have significantly increased the PM burden (Zhang et al. 2015). Large cities with higher traffic and poor air pollutant dispersion are more susceptible to negative health effects due to PM. High traffic density in urban areas contributed up to 140 % higher PM_{2.5} levels compared to suburban background area (Dongarra et al. 2010).

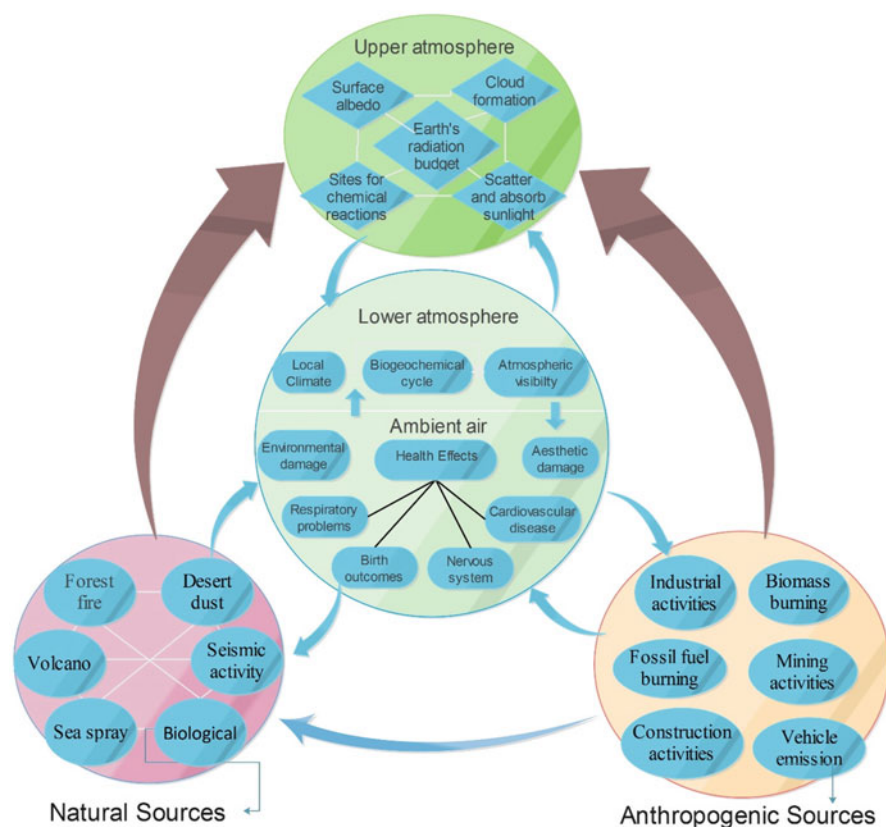


Fig. 1 Effects of particulate matter on different components of the atmosphere

The major component of PM having potential negative health effects are traffic and combustion related fine PM (Mills et al. 2009; Pope and Dockery 2013). Earlier reports have mostly highlighted PM exposure and short- or long-term effects with PM-related hospital admissions, mortality and morbidity (Samet et al. 2000; Pope et al. 2002; Dominici et al. 2006), but recent scientific findings have identified several health effects of PM as fine particles trigger inflammatory response, plaque formation in the blood vessels, lung cancer, term low birth weight, mutagenicity, changes in gene expression, and immune responses (Coronas et al. 2009; Wichmann et al. 2009; Raaschou-Nielsen et al. 2013; Dadvand et al. 2014; Ding et al. 2014; Hennig et al. 2014; Apte et al. 2015; Chafe et al. 2014).

As fine PM is one of the major air pollutants in the world and most severe to health, it is of utmost important to summarize a global scenario of PM status and its health effects. So considering these points the manuscript attempts to cover a global perspective of the current status of fine particulate matter pollution ($PM_{2.5}$) particles with an aerodynamic diameter of $2.5 \mu m$ or less. Their range of concentrations and

sources and their health effects in urban, suburban, rural, and remote areas of the world are well documented and analyzed. The major objective of this review is to collate the information on spatial variations in fine PM concentrations and their exceedances in different continents of the world, with special reference to country and city specific variations. Since fine PM is mainly responsible for negative health impacts, source apportionment of fine PM, its constituents, and different health impacts were also reviewed.

2 Method

World Wide Web was searched for combinations of key words such as fine particulate matter pollution, PM_{2.5}, source apportionment of fine PM, constituents of PM_{2.5}, health effects of fine PM in PubMed, Scopus, Google scholar, and SciVerse. We selected 500 peer-reviewed articles published after year 2000 containing information on fine PM concentrations and related health effects. Among the articles searched, only those satisfying the criteria such as measurement of fine PM and source apportionment by standard methodology, health effects using human subjects and relevant statistical analyses for health assessment were further screened for the review. Local studies with only limited data for specific season or location were excluded with exception of a few studies having no long-term data available for comparison at such locations. For global and country specific variations in fine PM, databases from government organizations like Central Pollution Control Board (CPCB) in India, United States Environmental Protection Agency (US EPA) in the USA, Chinese National Environmental Monitoring (CNEM) in China, European Environment Agency (EEA) for Europe, and World Health Organization (WHO) for global database were also screened (CPCB 2013; CNEM 2013; EEA 2013, US EPA 2015; WHO 2014). Relevant published reviews and meta-analysis were also searched to identify the health effects of PM_{2.5} and its constituents.

3 PM_{2.5} Guidelines and Standards

PM_{2.5} and below in ambient air significantly cause severe health concern, even in low concentrations. Several national and international monitoring agencies have proposed guideline values to reduce health effects caused by PM_{2.5}. The values above the guidelines are suggested to be toxic or causing severe negative effects on breathing for both short and long durations, but no concentration is identified at which no health effect is recorded (WHO 2005). These values are suggested based on scientific evidences of toxicity of specific pollutant, geographic location, emission sources, economic conditions, and monitoring duration. These guidelines are implemented to reduce or control the pollutant under the specific conditions of a country. Selected PM_{2.5} guideline values for 24-h average (short term) and annual average (long term) of national and international agencies are given in Table 1.

Table 1 PM_{2.5} ambient air quality guideline values ($\mu\text{g m}^{-3}$)

Agency/country	24-h	Annual	References
WHO	25.0	10.0	WHO (2005)
	75.0 (IT-1)	35.0 (IT-1)	
	50.0 (IT-2)	25.0 (IT-2)	
	37.5 (IT-1)	15.0 (IT-1)	
US EPA	35.0	12.0 (Primary)	US EPA (2015)
		15.0 (Secondary)	
China	35.0 Class 1	15.0 Class 1	GB-3095 (2012)
	75.0 Class 2	35.0 Class 2	
India	60.0	40.0	CPCB (2009)
Europe		25.0	EEA (2013)
Australia	25.0	8.0	NEPM (2002)
Canada	30.0 (Old)	10.0 (2015)	ECC (2013)
	28.0 (2015)	8.80 (2020)	
	27.0 (2020)		
Mexico	65.0	15.0	NOM (2005)
Bangladesh	65.0	15.0	Begum et al. (2013)
Thailand	50.0	25.0	PCD (2010)
Japan	35.0	15.0	EQSJ (2009)

IT-1 interim target-1, *IT-2* interim target-2, *IT-3* interim target-3

4 Global PM_{2.5} Status and Exceedance

4.1 Asia

Asia is the largest and most populated continent in the world with varying landscapes. Most of the developing countries in Asia have very high levels of air pollutants (WHO 2014). West and South Asia are the most populated and polluted zones in the world. In South Asia, Pakistan, Bangladesh, and Afghanistan have very high PM_{2.5} concentration and values were 8.00–10.0 times above the World Health Organization (WHO) annual mean standard of $10.0 \mu\text{g m}^{-3}$ (WHO 2005). In India, fine PM level was 5.00 times higher than the WHO annual mean standard. In West Asia, fine PM levels were very high in Qatar, Iran, and in United Arab Emirates although levels were significantly lower in Oman, Lebanon, and Israel (Fig. 2). In East Asia, Mongolia has very high PM level of $64.0 \mu\text{g m}^{-3}$ followed by China ($41.0 \mu\text{g m}^{-3}$), whereas Japan is the only country having PM_{2.5} levels below the WHO annual mean standard (Fig. 2d). South-East Asia is comparatively less polluted by fine PM as most of the countries showed values below $25.0 \mu\text{g m}^{-3}$ except Vietnam and Myanmar (WHO 2014).

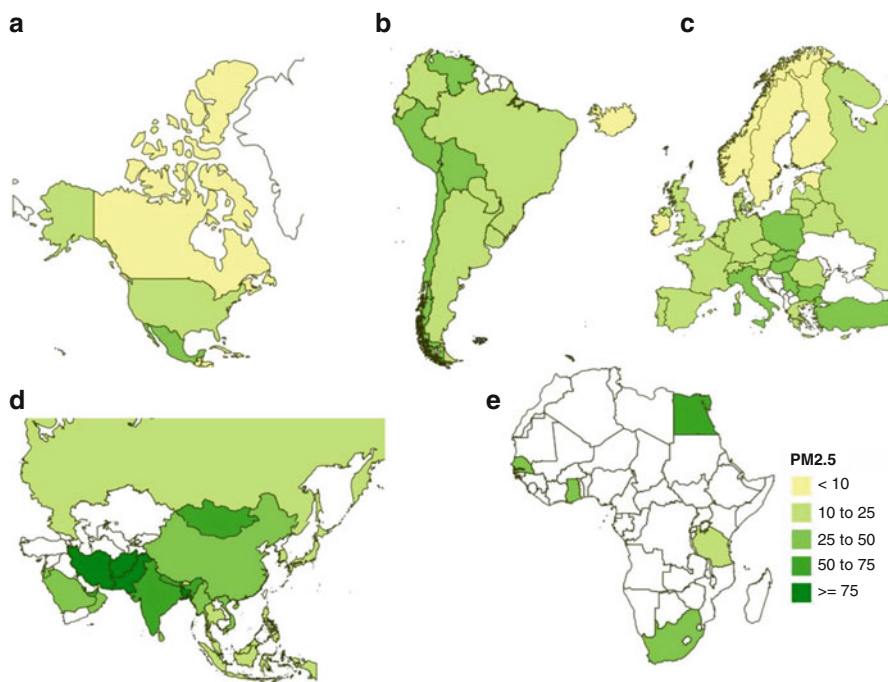


Fig. 2 Global distribution of $\text{PM}_{2.5}$ concentration ($\mu\text{g m}^{-3}$) in different continents of the world (a) North America, (b) South America, (c) Europe (d) Asia, and (e) Africa. (Data source WHO 2014)

4.1.1 India

Most of the major metropolitan cities in India violate the annual mean WHO and national ambient air quality standard (NAAQS) of India (CPCB 2009) for $\text{PM}_{2.5}$ (CPCB 2013) (Fig. 3). Major cities like Delhi, Ahmedabad, and Patna showed levels higher than $100 \mu\text{g m}^{-3}$ (CPCB 2013). Southern and northeastern part of India showed lower levels of $\text{PM}_{2.5}$ compared with other regions mostly due to higher vegetation cover, lower emissions, and planned urbanization (CPCB 2013; Tian et al. 2014). Most cities in north India are unplanned with huge population and poor road conditions that are ideal conditions for higher particulate episodes throughout the year. Seasonal variations were distinct with higher pollution load in winter and lower in rainy season (CPCB 2013). Chennai was found to be the least polluted with $\text{PM}_{2.5}$ and maximum levels were found in Delhi. Traffic is identified as a major cause of such higher $\text{PM}_{2.5}$ level in India (CPCB 2013).

Northern parts of India are highly polluted with PM levels in ambient air (Fig. 4). New Delhi, the capital of India, showed very high $\text{PM}_{2.5}$ levels ranging between 27.0 and $227 \mu\text{g m}^{-3}$ in urban atmosphere which exceeded the annual mean NAAQS of India (CPCB 2009) by 2.40 times (Tiwari et al. 2009). Further Tiwari et al. (2013) reported a higher annual mean of $122 \mu\text{g m}^{-3}$, with variations between

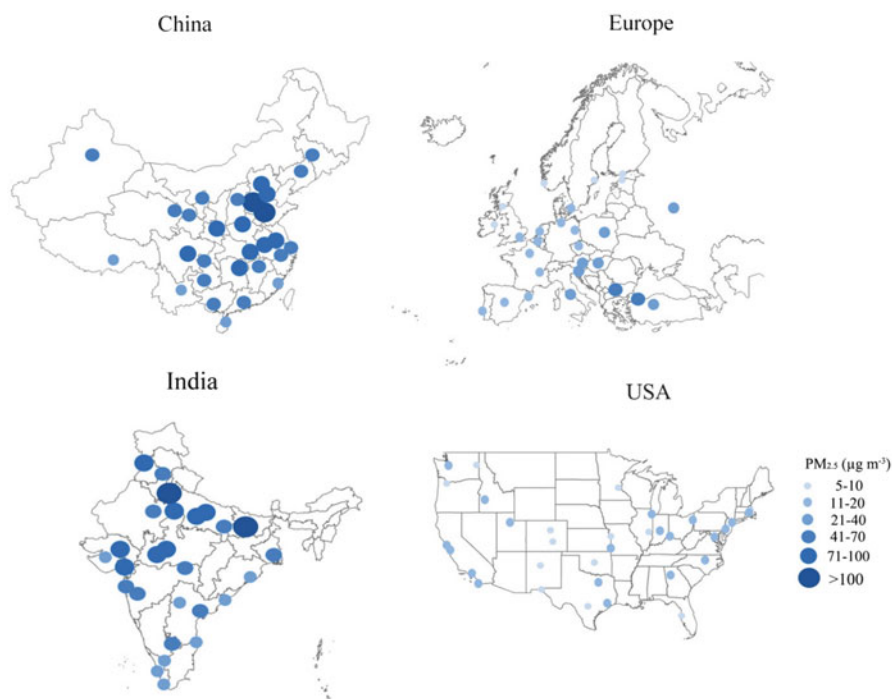


Fig. 3 Mean annual PM_{2.5} concentrations for year 2013 in China and the USA and for 2011 for India and Europe (Data Source CPCB 2013; CNEM 2013; EEA 2013, US EPA 2015, WHO 2014)

54.3 and 338 $\mu\text{g m}^{-3}$ in New Delhi. Higher concentration of fine PM in northern India is a direct result of excessive biomass burning, coal burning, traffic, and dust storms in summer from Thar Desert of Rajasthan (Kulshrestha et al. 2009; Tiwari et al. 2013). Mean PM_{2.5} concentrations in both urban and rural areas in Agra were more than double the annual mean NAAQS of India (CPCB 2009), and annual average levels were 10.5 and 9.10 times higher than annual mean WHO standard, respectively (Kulshrestha et al. 2009). Similarly, in Kanpur ambient PM_{2.5} levels in commercial, residential, and control sites were well above the annual mean national standard of India and 14.0, 9.50, and 6.00 times of WHO annual mean standard at respective sites (Sharma and Maloo 2005).

PM_{2.5} levels ranged between 34.5 and 112 $\mu\text{g m}^{-3}$ in the largest city of eastern India, Kolkata, with annual mean value, almost 1.80 times the annual mean national standard (CPCB 2009), and maximum exceedance occurred during winter season (2.30–2.80 times) (Chatterjee et al. 2012). In Raipur, another growing urban center in eastern India, PM_{2.5} levels ranging from 24.0 to 269 $\mu\text{g m}^{-3}$ with a mean value of 150 $\mu\text{g m}^{-3}$, which was more than three times higher than annual mean NAAQS (CPCB 2009). But the most concerning factor was that PM_{2.5} levels exceeded the annual mean standard for 94.0 % of sampling days (Deshmukh et al. 2013).

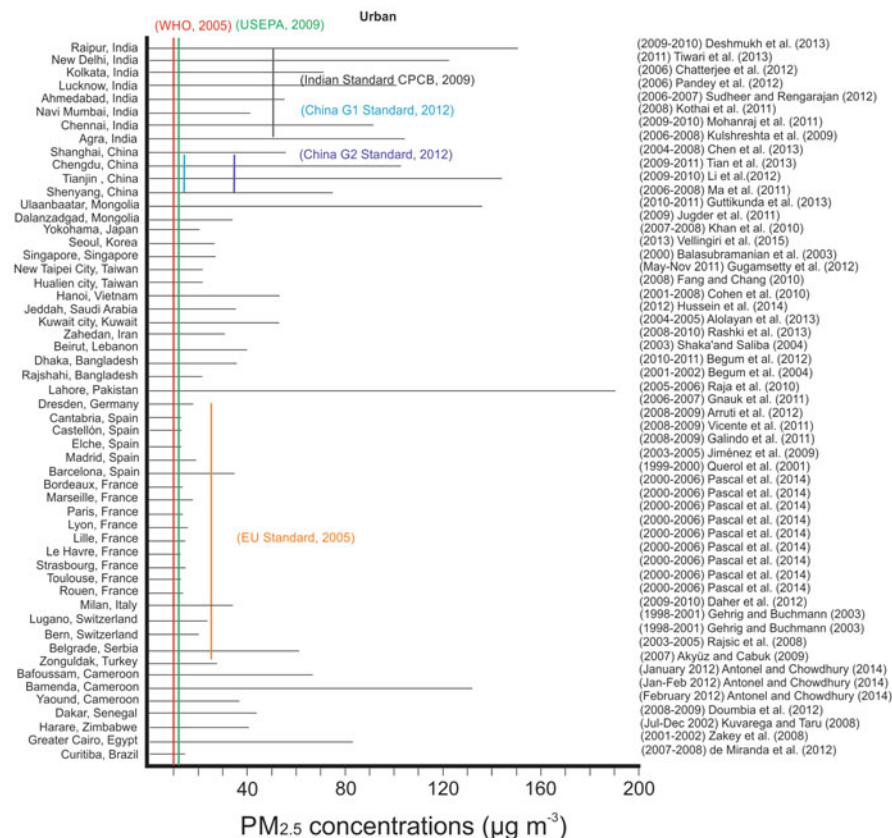


Fig. 4 Variations in PM_{2.5} concentrations at different urban locations throughout the world. Concentrations are mean values based on measurements performed in different years (varying from 1999 to 2013)

Monitoring data from heavily urbanized area of Ahmedabad, one of the largest city in western India, revealed PM_{2.5} concentrations to be 1.30 times the NAAQS and 5.00 times of WHO annual mean standard during the study period from 2006 to 2007 (Sudheer and Rengarajan 2012). A study based on different land use patterns in Mumbai city by Joseph et al. (2012) reported 59.0, 76.0, 67.0, and 80.0 % exceedance of NAAQS (CPCB 2009) for PM_{2.5} at control, kerb, residential, and industrial sites, respectively.

4.1.2 China

China showed higher PM_{2.5} concentrations in all major cities with violation of annual mean Class I Chinese ambient air quality standards (CAAQS) of $15.0 \mu\text{g m}^{-3}$ and Class II standard of $35.0 \mu\text{g m}^{-3}$ (GB3095 2012) (Fig. 3). Shijiazhuang and Jinan showed

values above $100 \mu\text{g m}^{-3}$ in north, whereas apart from Haikou and Fuzhou in south-east and Yinchuan, Lhasa, and Kunming in western China, all other cities showed $\text{PM}_{2.5}$ levels above $50.0 \mu\text{g m}^{-3}$ (WHO 2014). Seasonal variations were distinct with maximum concentration during winter and lowest during summer season. Higher exceedance levels of $\text{PM}_{2.5}$ concentrations were mostly in northern part compared to south-east and west and exceedance rates were more in winter than in summer season.

In urban and suburban areas in Tianjin, $\text{PM}_{2.5}$ mass concentration was above CAAQS (GB3095 2012) and almost 13.0 times the WHO annual mean standard (Li et al. 2012). $\text{PM}_{2.5}$ mean concentrations were just above 24-h CAAQS at suburban sites near two largest cities, Beijing and Shanghai (Zhou et al. 2009), however, at an urban site in Shanghai, concentrations varied from 8.00 to $389 \mu\text{g m}^{-3}$ with annual mean level exceeding annual mean CAAQ Class II standard by 1.60 times during the study period of 2004–2008 (Chen et al. 2013). Hu et al. (2014) studied short-term $\text{PM}_{2.5}$ levels during June–August 2013, in 13 cities located in North China Plain (NCP) and in 20 cities in Yangtze River Delta (YRD). Average $\text{PM}_{2.5}$ levels were $77.0 \mu\text{g m}^{-3}$ in NCP region and $42.8 \mu\text{g m}^{-3}$ in YRD region and in both the regions, annual mean CAAQ Class I standard was exceeded by 83.0 % and 51.0 % of times, respectively. Mean annual level of $\text{PM}_{2.5}$ in Chengdu was 10.0 times of the WHO annual mean standard and almost three times of annual mean CAAQ Class II standard of $35.0 \mu\text{g m}^{-3}$.

4.1.3 Other Asian Countries

$\text{PM}_{2.5}$ levels were recorded very high in Lahore, Pakistan with annual average concentration of $194 \mu\text{g m}^{-3}$ during 2007–2008, and $\text{PM}_{2.5}$ levels were above $100 \mu\text{g m}^{-3}$ for 84.0 % of sampling days (Stone et al. 2010). Khwaja et al. (2015) reported $\text{PM}_{2.5}$ concentrations varying between 30.0 and $279 \mu\text{g m}^{-3}$ with an annual mean of $101 \mu\text{g m}^{-3}$ in the megacity of Karachi, Pakistan during 2008–2009. Long-term monitoring results of $\text{PM}_{2.5}$ concentrations from 1996 to 2011 in Dhaka, Bangladesh showed values ranging between 5.26 and $240 \mu\text{g m}^{-3}$ with a mean value of $36.7 \mu\text{g m}^{-3}$, which was 3.70 times higher than WHO annual mean standard and 2.40 times of Bangladesh annual standard of $15.0 \mu\text{g m}^{-3}$ (Begum et al. 2013) (Fig. 4). In an urban traffic-influenced site in Dhaka, annual mean $\text{PM}_{2.5}$ concentration level was $82.5 \mu\text{g m}^{-3}$, which was ascribed to high traffic under urban influence (Begum et al. 2012). Alolayan et al. (2013) in Kuwait City reported $\text{PM}_{2.5}$ levels to be 5.00 times the WHO annual mean standard and exceedance of 78.0 % days above WHO 24-h mean value. In Jeddah, Saudi Arabia, $\text{PM}_{2.5}$ levels were 3.40 times of the WHO annual mean standard (Hussein et al. 2014). $\text{PM}_{2.5}$ concentrations varied between 23.0 and $186 \mu\text{g m}^{-3}$ with an average of $74.2 \mu\text{g m}^{-3}$ during 2000–2001 in Jeddah, Saudi Arabia (Hussain et al. 2014).

Compared to other Asian megacities, $\text{PM}_{2.5}$ levels were comparatively lower in Singapore with annual mean $\text{PM}_{2.5}$ concentrations of $27.2 \mu\text{g m}^{-3}$ and levels exceeded only 30.0 % times, the value of $30.0 \mu\text{g m}^{-3}$, although the mean level was 2.70 times higher than WHO annual mean standard (Balasubramanian et al. 2003). In urban residential site in Karachi, Pakistan, $\text{PM}_{2.5}$ concentration varied

from 27.4 to 175 $\mu\text{g m}^{-3}$ with annual mean level, 8.35 times higher than the annual mean WHO standards (Mansha et al. 2012). Han et al. (2011) monitored $\text{PM}_{2.5}$ levels at a rural site in Chuncheon, Korea and reported that about 37.0 % of samples exceeded 24-h US NAAQS of 35.0 $\mu\text{g m}^{-3}$ and annual mean level was 3.00 times the annual mean WHO standard (Fig. 6). Urban residential site in Kaohsiung City, Taiwan showed $\text{PM}_{2.5}$ levels ranging from 0.80 to 162 $\mu\text{g m}^{-3}$ with annual mean value three times of the annual mean WHO standard during 2006–2010 (Cheng et al. 2014).

4.2 Africa

In the African continent, fine PM levels were above 30 $\mu\text{g m}^{-3}$ in Senegal, Mauritius, and Ghana whereas in South Africa, level was below 30 $\mu\text{g m}^{-3}$ (Fig. 2e). Compared to other countries in Africa, Ghana showed high mean $\text{PM}_{2.5}$ value of around 49 $\mu\text{g m}^{-3}$ (WHO 2014). Due to lack of data in the most of the countries in this region, limited information is available on fine PM levels in Africa. Poor air quality in African continent is not new with excessive biomass burning for cooking and dusty roads. African dust storms add more concern to the already severely affected continent.

High $\text{PM}_{2.5}$ levels were observed by Doumbia et al. (2012) at a traffic-influenced site in Dakar, the capital and the largest city of Senegal in West Africa, where daily mean levels varied between 24.0 and 80.0 $\mu\text{g m}^{-3}$ and annual mean concentration was several times higher than WHO annual mean standard. Dionisio et al. (2010) reported about 8.00–14.0 $\mu\text{g m}^{-3}$ higher $\text{PM}_{2.5}$ levels in roadside compared to residential site in Accra, the capital of West African country of Ghana. Fine PM levels ranged between 3.00 and 53.0 $\mu\text{g m}^{-3}$ at an urban background site and from 1.90 to 36.0 $\mu\text{g m}^{-3}$ at a suburban site in Nairobi, Kenya, and both sites exceeded the 24-h WHO standard by 29.0 and 7.00 %, respectively (Gaita et al. 2014). $\text{PM}_{2.5}$ at a kerb side in Dares Salaam, the largest city of Tanzania ranged between 5.10 and 66.0 $\mu\text{g m}^{-3}$ (Mkoma et al. 2010). Around an urban industrial area in Harare, Zimbabwe Kuvarega and Taru (2008) found mean $\text{PM}_{2.5}$ concentration of 40.5 $\mu\text{g m}^{-3}$, which exceeded the WHO annual mean limit throughout the monitoring period. Owoade et al. (2015) found fine PM ranging between 14.4 and 986 $\mu\text{g m}^{-3}$ around scrap iron and steel smelting industries in South Western Nigeria during 2011–2012, with a mean value of 300 $\mu\text{g m}^{-3}$ around the main production unit.

4.3 Europe

Europe seems to have much better air quality related to fine PM concentration compared to other regions of the world primarily due to planned development, green technology, reduction in emissions, and lower population density (Maas and Grennfelt 2016). Fine PM levels were least in Northwestern Europe with only United Kingdom, Denmark, and Lithuania having values above WHO guideline (Fig. 2c). In Western Europe, most of the countries have PM_{2.5} values ranging between 15.0 and 20.0 $\mu\text{g m}^{-3}$ (WHO 2014). In Eastern Europe, fine PM values above 20.0 $\mu\text{g m}^{-3}$ were recorded in most of the countries except Romania and Belarus and maximum concentration was reported from Turkey, with annual average level of 39.0 $\mu\text{g m}^{-3}$ (WHO 2014). In Southern European countries, annual fine PM values were just above the annual mean WHO levels of 10.0 $\mu\text{g m}^{-3}$ in Spain, Portugal, and Malta, above 20.0 $\mu\text{g m}^{-3}$ in Slovenia and Italy, above 30.0 $\mu\text{g m}^{-3}$ in Serbia, and above 40.0 $\mu\text{g m}^{-3}$ in Bulgaria (WHO 2014).

PM_{2.5} levels in most European cities were well below the annual mean European Union (EU) PM_{2.5} standard of 25.0 $\mu\text{g m}^{-3}$ (Fig. 4). In some of the cities like Dublin (Ireland), Stavanger (Norway), Stockholm (Sweden), and Helsinki (Finland), the levels were even below the annual mean WHO PM_{2.5} standard of 10.0 $\mu\text{g m}^{-3}$. In Madrid (Spain), Lisbon (Portugal), and Glasgow (UK), levels were just above or around the annual mean WHO standard. Only few cities showed values above the annual mean EU PM_{2.5} standard like Sofia (Bulgaria) and Istanbul (Turkey), where annual mean levels were twice the EU permissible limit. Larger European cities like Paris, Berlin, Barcelona, Prague, Moscow, and London also showed PM_{2.5} values below EU permissible limit, but were well above WHO, PM_{2.5} annual mean standard.

Rajšić et al. (2008) reported PM_{2.5} concentration to be three times higher than the European Commission (EC) annual limit of 20.0 $\mu\text{g m}^{-3}$ and six times of the WHO annual limit in Belgrade, Serbia. Roadside monitoring of PM_{2.5} in Athens, Greece, showed levels ranging from 8.00 to 135 $\mu\text{g m}^{-3}$ with annual mean level almost four times of the annual mean WHO and 1.60 times of the annual mean European Union (EU) standards (Chaloulakou et al. 2005). In dense traffic area around a highway in Istanbul, Turkey, Onat et al. (2013) found PM_{2.5} levels ranged from 23.8 to 81.5 $\mu\text{g m}^{-3}$ whereas PM_{2.5} value was observed above 35.0 $\mu\text{g m}^{-3}$ during 60 % of the total monitoring period.

Mean annual PM_{2.5} concentration was twice the EU and five times of the WHO annual mean standards in industrialized city of Bursa, Turkey (Kendall et al. 2011). Mean PM_{2.5} concentrations were 24.11 and 64.3 $\mu\text{g m}^{-3}$, respectively, in suburban and urban areas in Izmir, Turkey during 2004–2005 (Yatkin and Bayram 2008). In three cities, Pavia, Verona, and Torino in Northern Italy, PM_{2.5} levels were 1.4, 1.5, and 1.9 times of the annual mean EU standard whereas exceedance in daily air quality value was more than 50 % in all these cities (Traversi et al. 2009). Lazaridis et al. (2008) found PM_{2.5} concentration close to the EU annual mean, but were 2.50–2.70 times higher than the annual WHO standard at Akrotiri station, an urban

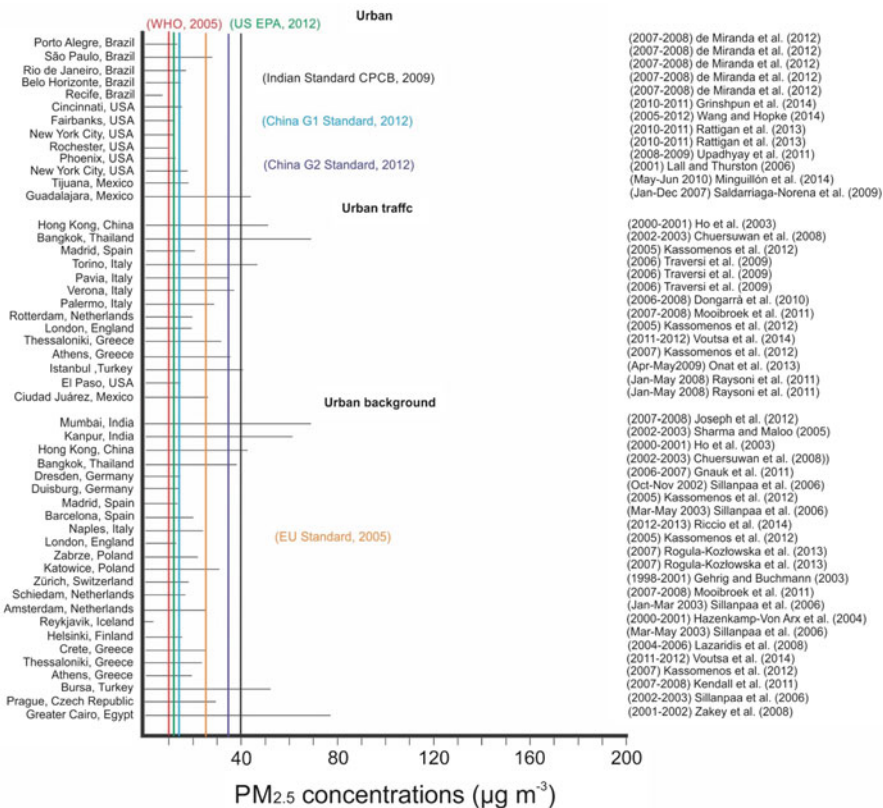


Fig. 5 Variations in PM_{2.5} concentrations at different urban, urban traffic, and urban background locations throughout the world. Concentrations are mean values based on measurements performed in different years (varying from 1998 to 2013)

background area of the island of Crete, Greece (Fig. 5). In suburban areas in Cartagena, Spain, PM_{2.5} concentrations ranged from 7.00 to 47.0 $\mu\text{g m}^{-3}$ with annual mean below the EU standard and 2.20 times the annual mean WHO standard (Fig. 6) (Negral et al. 2008). Long-term monitoring results from 2003 to 2012 at a rural site in Melpitz, Germany showed that PM_{2.5} levels were consistently above the annual mean WHO standard (Spindler et al. 2013) (Fig. 6). Hazenkamp-Von Arx et al. (2004) monitored PM_{2.5} levels in 21 cities in Europe for 1 year and found PM_{2.5} levels ranging from 3.70 to 44.9 $\mu\text{g m}^{-3}$ with lowest concentration in Reykjavik, the capital of Iceland and maximum in Turin, Italy. In most of the major cities like Paris, Barcelona, and Antwerp, PM_{2.5} levels were above the annual mean WHO standard. A similar study from 20 European sites during 2008–2011 found that 16 out of 20 sites showed PM_{2.5} levels above the WHO annual limit, whereas except Turin, Italy, all other cities were below the EU annual limit (Eeftens et al. 2012).

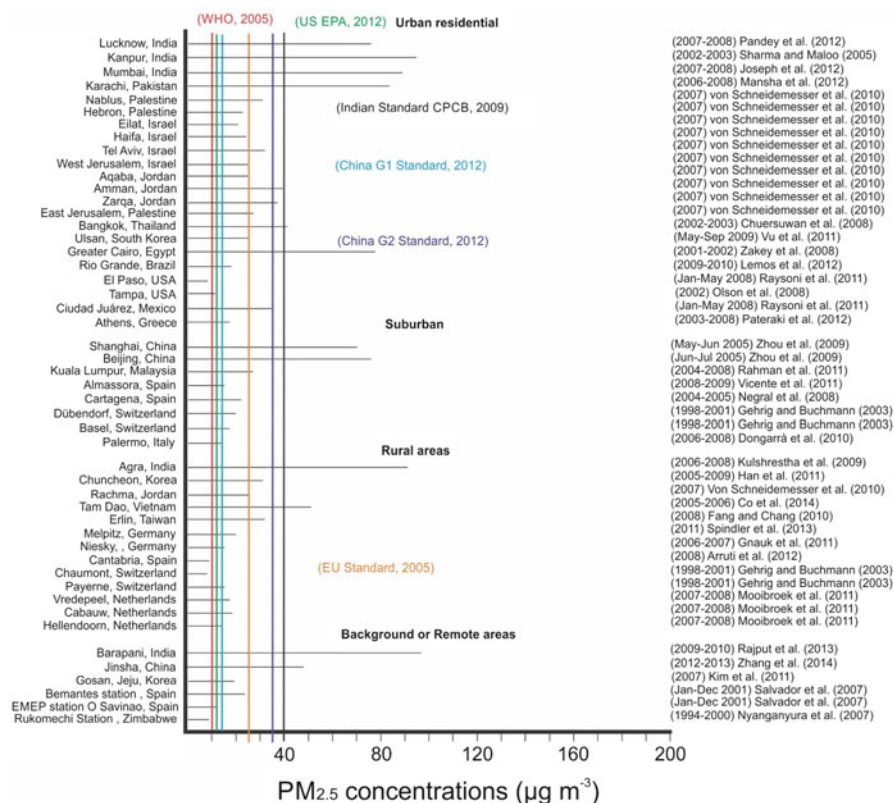


Fig. 6 Variations in PM_{2.5} concentrations at different urban residential, suburban, rural, and remote location throughout the world. Concentrations are mean values based on measurements performed in different years (varying from 1994 to 2013)

4.4 South America

Unlike Asia and Africa, South American countries have regulated fine PM levels up to a significant level (WHO 2014). In most of the countries, PM_{2.5} levels were below $30.0 \mu\text{g m}^{-3}$ with exception of Peru where concentration was $39.0 \mu\text{g m}^{-3}$. In Brazil and Columbia, values were below $25.0 \mu\text{g m}^{-3}$, whereas in Uruguay, Argentina, Ecuador, and Paraguay levels were even below $20.0 \mu\text{g m}^{-3}$ (Fig. 2b). PM_{2.5} estimation in highly traffic area in six Brazilian state capitals revealed that concentration was maximum in Sao Paulo ($28.1 \mu\text{g m}^{-3}$) and least in Recife ($7.10 \mu\text{g m}^{-3}$). In all the cities except Recife, levels were higher than the annual mean WHO and USEPA standards (de Miranda et al. 2012).

Mariani and de Mello (2007) reported PM_{2.5} concentration of $17.0 \mu\text{g m}^{-3}$ at a costal urban site in Rio de Janeiro, Brazil. Short-term study of PM_{2.5} levels by Lemos et al. (2012) in urban, industrial, and residential areas in Rio Grande city,

Brazil showed that levels were below the Brazilian standard, but higher than 24-h WHO standard on few occasions during the study period. $\text{PM}_{2.5}$ mean concentration was 71.0 and 61.0 $\mu\text{g m}^{-3}$ in urban and semiurban areas of the Córdoba, the second largest city of Argentina which was three times higher than the EU and seven times higher than the annual mean WHO standards during the period of July 2009–April 2010 (López et al. 2011).

4.5 North America

$\text{PM}_{2.5}$ levels in North American continent were variable with countries. Levels in Canada were below the WHO annual mean standard and were just above the standard in the USA (WHO 2014). In countries like Mexico levels were 2.50 times higher than the annual mean WHO standard (Fig. 2a).

According to US EPA dataset for $\text{PM}_{2.5}$ (Weighted Annual Mean) for year 2013 (US EPA 2015), levels in most of the areas in the USA were below NAAQS (US EPA 2012) annual mean standard of 15.0 $\mu\text{g m}^{-3}$ (Fig. 3). Only few areas having higher $\text{PM}_{2.5}$ levels are Bakersfield, CA; Fairbanks, AK; Fresno, CA based on Core Based Statistical Area (CBSA) in the USA. The database clearly showed that $\text{PM}_{2.5}$ is under control in most of the areas in the USA. In many areas values were even below 5.00 $\mu\text{g m}^{-3}$. When 98th percentiles of the daily average measurement values for the whole period were compared, exceedance of 24-h NAAQS of 35 $\mu\text{g m}^{-3}$ was more prominent (US EPA 2012). It indicates that short-term effects of $\text{PM}_{2.5}$ are more realized than the long-term ones. Results of 537 monitoring sites for $\text{PM}_{2.5}$ from 2000 to 2013 indicate a 34.0 % national decrease in fine PM concentration in the USA (US EPA 2014).

Saldarriaga-Noreña et al. (2009) in Guadalajara, Mexico found $\text{PM}_{2.5}$ levels to be 2.90 and 3.50 times the annual mean Mexican standard of 15.0 $\mu\text{g m}^{-3}$ in industrial and traffic-influenced sites, with values ranging from 2.00 to 181 $\mu\text{g m}^{-3}$ during the study period (Fig. 4). Raysoni et al. (2011) analyzed $\text{PM}_{2.5}$ levels outside schools in US-Mexican border and found higher concentrations at schools in Ciudad Juárez, Mexico compared to El Paso, USA during January to May 2008. A short-term study by Olson et al. (2008) at a residential outdoor in Tampa, USA showed that $\text{PM}_{2.5}$ level was three times below the USEPA 24-h standard during October and November 2002. Long-term monitoring results in both New York City and Rochester, USA showed decreasing pattern of $\text{PM}_{2.5}$ level from 2003 to 2012 with values within the annual mean WHO and US EPA levels in Rochester and just above WHO level in New York City (Rattigan et al. 2013). In four cities in Connecticut and one in Massachusetts, $\text{PM}_{2.5}$ levels ranged between 11.9 and 17.0 $\mu\text{g m}^{-3}$ with maximum concentration of 18.3 $\mu\text{g m}^{-3}$ during cold season in New Haven and lowest concentration of 11.6 $\mu\text{g m}^{-3}$ at Hartford (Lee et al. 2011). Qin et al. (2006) found average $\text{PM}_{2.5}$ concentration of 13.0–15.0 $\mu\text{g m}^{-3}$ in different urban commercial area in New York City.

5 Source Apportionment for PM_{2.5}

Source apportionment studies help to recognize or characterize those factors that contribute to pollution levels. The composition of PM depends upon its origin and transformations that happen under different environmental conditions (Hyder et al. 2014). An effect of PM is elicited based on its chemical nature and toxicity. PM_{2.5} is mostly generated due to combustion and emissions from automobiles (Fleisch et al. 2014). Major components of PM_{2.5} are black carbon (BC), polycyclic aromatic hydrocarbons (PAHs), heavy metals, organic carbon, hydrocarbons, volatile organic hydrocarbons (VOCs), minerals, inorganic ions, and biological material (Tiwari et al. 2009; Traversi et al. 2009; de Miranda et al. 2012; Fleisch et al. 2014).

5.1 Asia

In New Delhi, India, Tiwari et al. (2009) reported that PM_{2.5} was made up of undetermined fractions (40.9 %), secondary inorganic aerosols (27.1 %), salt aerosols (23.3 %), and mineral matter (8.70 %) indicating sources such as biomass and fossil fuel burning and soil derived particles. Source apportionment through principal component analysis (PCA) of PM_{2.5} revealed that major contributors were vehicular emission (38.0 %), biomass burning (27.0 %), dust aerosols (18.0 %), and secondary anthropogenic components (11 %) in the eastern Indian megacity of Kolkata in 2006 (Chatterjee et al. 2012). PM_{2.5} mass was mostly contributed by organic matter (36.0–52.0 %), secondary inorganic aerosols (21.0–27.0 %), crustal (6.00–12.0 %), non-crustal (4.0–8.00 %), and sea salt (6.00–11.0 %) in Mumbai, India (Joseph et al. 2012). Sudheer and Rengarajan (2012) in urban atmosphere of Ahmedabad, India reported that anthropogenic sources (80.0 %) contributed to PM_{2.5} mass and identified industrial emission, biomass burning, vehicular emissions, and resuspended or long range transported dust as the major sources of particulate pollution.

Major contributors to high PM_{2.5} in Karachi, Pakistan were industrial emissions (53.0 %), road dust (16.1 %), and secondary aerosols (12.4 %) (Mansha et al. 2012). Source apportionment study by Raja et al. (2010) in Lahore, Pakistan detected secondary particles (30.2 %), diesel emissions (28.3 %), biomass burning (14.8 %), coal combustion (13.3 %), and two-stroke vehicles (7.70 %) as the major contributors to observed PM_{2.5} mass. Alolayan et al. (2013) in Kuwait City found maximum contribution of soil/sand dust (54.0 %) followed by oil combustion (18.0 %), petrochemical industry (12.0 %), local traffic (11.0 %), and transported traffic/smelter emissions (5.0 %) to PM_{2.5}, indicating that major sources in Kuwait city are of crustal origin which is approximately 50.0 % mass of PM_{2.5}, and were attributed to dust storm events in this area (Alolayan et al. 2013). Major sources of fine particles in Kuala Lumpur, Malaysia were two-stroke engines (35.7 %), motor vehicles (31.9 %), smoke/biomass burning (17.5 %), soil dust (8.30 %), and

industry (16.7 %) (Rahman et al. 2011). At a traffic site in Bangkok Metropolitan Region, Thailand, Chuersuwan et al. (2008) observed maximum contribution from automobiles (32.0 %), followed by biomass burning (26.0 %). PCA analysis revealed six major sources of $PM_{2.5}$ in Singapore as soil dust, metallurgical industry, emissions from biomass fires and local traffic, sea spray, and fuel oil combustion processes (Balasubramanian et al. 2003).

5.2 *Europe and Africa*

Rogula-Kozłowska et al. (2013) found that apart from industrial, power plants, and soil/road dust, combustion of fuels in domestic uses and automobiles contributed to 36.0–78.0 % $PM_{2.5}$ mass at two sites in Poland. In urban traffic and background sites, Voutsas et al. (2014) found secondary inorganic aerosols (SIA) to be a major part of $PM_{2.5}$ mass and identified local sources and long range transport as major contributors to SIA formation in Thessaloniki, Greece. In Barcelona Metropolitan area, Spain, vehicular-secondary sources contributed 73.0 % of $PM_{2.5}$ mass (Querol et al. 2001). Rajšić et al. (2008) reported combustion sources from traffic and resuspended road dust mostly contribute to trace metals in coarse and fine fraction of PM in a central urban area in Belgrade, Serbia. Negral et al. (2008) identified sources of $PM_{2.5}$ as crustal, traffic, secondary, marine, Zn metallurgy industry, and shipyard in Cartagena, Spain. Source profile of fine PM using positive matrix factorization (PMF) technique, in Izmir, Turkey, showed contributions of unidentified fraction (38.0 %), fuels and steel (37.0 %), traffic (12.0 %), mineral (9.00 %), and marine (4.00 %) sources in suburban area whereas unidentified (48.0 %), fuels and steel (22.0 %), traffic (15.0 %), marine (14.0 %), and mineral (1.00 %) in urban area Yatkin and Bayram (2008).

Takey et al. (2008) identified waste burning to be a significant source of $PM_{2.5}$ in different environments of Greater Cairo area in Egypt. In Nairobi, Kenya, source apportionment by PMF identified five major sources of fine PM as traffic (39.0 %), mineral dust (35.0 %), mixed factor (13.0 %), industrial (7.0 %), and combustion (6.0 %) (Gaita et al. 2014). Around scrap iron and steel smelting industries in South Western Nigeria, Owoade et al. (2015) identified coking coal (83 %) as the major source of fine PM followed by soil (10.0 %), metallurgical industry (6.00 %), and electronic waste processing (1.00 %).

5.3 *North and South America*

Source apportionment analysis by Olson et al. (2008) in residential outdoors of Tampa, Florida identified sulfate (55.0 %), gasoline-powered motor vehicles (32.0 %), diesel-powered vehicles (8.00 %), and road dust (5.00 %) as major sources of $PM_{2.5}$ by using EPA Chemical Mass Balance Model. Pancras et al.

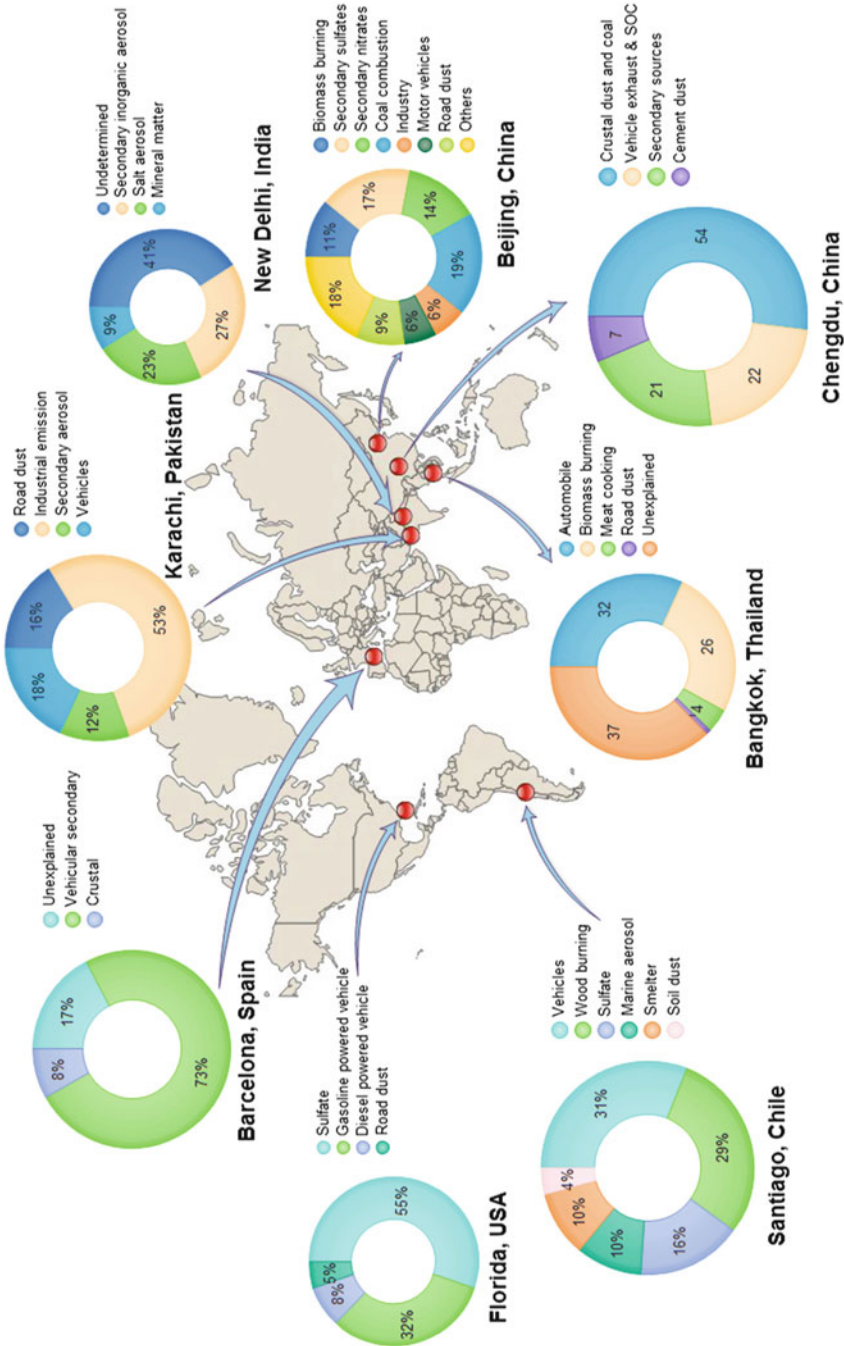


Fig. 7 Source apportionment of $PM_{2.5}$ in selected cities of the world

(2013) in Dearborn, Michigan, USA identified automobiles, road dust, and industries, secondary sources as the major sources of $PM_{2.5}$ pollution. Sources of $PM_{2.5}$ in four cities in Connecticut and one in Massachusetts in the USA were sulfur-related pollution, motor vehicle, road dust, oil combustion, and sea salt (Lee et al. 2011).

A study by Murillo et al. (2013) to identify sources of EC and OC in $PM_{2.5}$ mass characterized gasoline vehicles (10.0 %), on-road diesel emissions (16.0 %), rail-road traffic (4.0 %), industrial combustion (9.0 %), and wood smoke (5.0 %) as major contributors to $PM_{2.5}$ in Costa Rica, Central America. Major sources of $PM_{2.5}$ in urban area of Córdoba City, Argentina were traffic (32.0 %), urban dust (54.0 %), soil dust (1.00 %), metallurgical industries and diesel (13.0 %), whereas in suburban site major contributors were urban dust (56.0 %) followed by traffic (28.0 %) and metallurgical industries and diesel (11.0 %) (López et al. 2011).

Different sources of fine PM in different regions of the world mostly depend on local activities, population density, atmospheric condition, and land use pattern. In Fig. 7 a detailed source profile of $PM_{2.5}$ at selected cities of the world, where exceedances are high is shown.

6 Gas to Particle Conversion and Secondary Aerosol Formation in Atmosphere

PM in the atmosphere is either directly generated through primary sources or by secondary formation due to gas to particle conversion. Secondary generation of PM contributes significant portion of fine PM in the atmosphere, as most of the secondary aerosols are formed from particles of size 0.10–2.50 μm (Baek et al. 2006; Hallquist et al. 2009; Zhang et al. 2015; Yao et al. 2016).

Major secondary aerosols in the atmosphere are organics, sulfate, nitrate, ammonium, and other inorganic salts, which are formed either by gas to particle conversion or by particle phase reactions (Zhang et al. 2015; Huang et al. 2016). Primary emitted gases in the atmosphere such as sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ammonia (NH_3), ozone (O_3), and VOCs undergo photochemical oxidation, hydration, peroxyradical self-reaction, and condensation to produce various short lived intermediate molecules, which by process of nucleation, transformation, and coagulation ultimately produce secondary aerosols (Baek et al. 2006; Hallquist et al. 2009; Zhang et al. 2015). Secondary aerosol formation is a complex process controlled by concentrations of precursor atmospheric gases, particle size of existing PM, relative humidity, temperature, wind speed and direction, boundary layer height, and solar radiation (Huang et al. 2016). Higher relative humidity increases higher secondary formation by increasing oxidation of sulfur and nitrogen species in the atmosphere (Huang et al. 2016).

Secondary aerosol formation and their lifetimes in the atmosphere is variable with higher concentrations during day time which is correlated with higher

emissions of precursor gases and suitable atmospheric conditions for secondary aerosol formation. Mancilla et al. (2015) found 32.0 % higher organic carbonaceous aerosol during day hours compared to night time in an urban area of Monterrey, Mexico.

Inorganic aerosols such as sulfate and nitrate are formed through initial reactions of hydroxyl radicals (OH) with SO₂ and NO₂ by intermediate formation of sulfuric and nitric acid. Nitric acid may further react with ammonia gas to form ammonium nitrate aerosol. Sulfuric acid may further nucleate or may be absorbed at existing particle surfaces or form salts with other inorganic species such as ammonia (Baek et al. 2006; Hallquist et al. 2009; Zhang et al. 2015). In Beijing, China, secondary inorganic aerosols such as sulfate, nitrate, and ammonium contributed almost 82.8 % of total inorganic mass of water soluble inorganics in fine PM compared to 61.8 % contribution in coarse particle fraction, indicating higher secondary formation in fine mode aerosols (Zhang et al. 2015). Further, during polluted days contributions of secondary inorganic aerosols (sulfate, nitrate, and ammonium) were much higher (82.0–90.0 %) than in non-polluted days (64.0–81.0 %) in total PM inorganic mass.

In comparison to inorganic aerosols, reaction mechanisms of VOCs are much more complicated. Different VOCs released into the atmosphere undergo further oxidation to produce less volatile organics, such as aldehydes, carboxylic acids, ketones, and hydroperoxides, by photo-oxidation in the presence of hydroxyl radicals, ozone, nitrate, water vapor through several chemical reactions such as aldol condensation, ozonolysis, and peroxyradical self-reaction to form secondary organic species (Zhang et al. 2015). Significant contributions of around 11.0–52.0 % of secondary organic aerosols in PM_{2.5} were observed in an urban metropolitan area of Monterrey, Mexico by Mancilla et al. (2015), whereas in an urban area of Ahmedabad city, 34.6 % and 45.0 % contributions to the total carbonaceous aerosol mass in PM_{2.5} were reported by Sudheera et al. (2015), respectively, during day and night time.

7 Health Effects of PM_{2.5}

Among outdoor air pollutants, PM_{2.5} is the most prevailing contributor to the global health liability (Anenberg et al. 2010). PM_{2.5} has the ability to cross the alveoli of the lung and can finally enter into the blood stream to produce inflammatory or oxidative damage, leading to more drastic and long-term secondary effects on both cardiovascular and nervous systems (Araujo 2011; Breysse et al. 2013; Øvreivik et al. 2015). These health effects mostly depend upon age, lifestyle, health status, medical care, and exposure concentrations of the pollutants and climate. Percentage change in PM_{2.5} mean annual exposure data between 1990 and 2010 showed a global increase of PM_{2.5} exposure by 9.90 % (World Bank 2015). This global increase is directly correlated with 8.00 % increase in South Asia and 34.0 % increase in East Asia and Pacific. Significant reductions have, however, occurred in

European Union (42.0 %), Latin American countries (7.00 %), and the USA (29.0 %) in the last two decades (Fig. 8). A detailed summary of studies examining the association between $PM_{2.5}$ and health effects is presented in Table 2. It is beyond the scope of this article to assess all the health effects of $PM_{2.5}$, therefore in this section we have only discussed recent studies suggesting association of $PM_{2.5}$ and health effects from article published between 2004 and 2017.

7.1 *Evidences from Recent Reviews and Meta-Analysis of Health Effects of Fine PM*

Sun et al. (2015) assessed a relationship between fine particulate matter exposure during pregnancy and preterm birth based on 18 studies before 2014 and found a positive association between fine PM and preterm birth. Hamra et al. (2014) suggested outdoor air pollution and PM to be classified as Group-1 carcinogen based on the systematic review and meta-analysis of outdoor particulate matter exposure and lung cancer risk. In a systematic review and meta-analysis of short-term exposure to PM Bell et al. (2013) reported a higher risk of PM associated hospitalization and death for elderly persons and indicative evidence of higher risk of death for lower education and income group individuals. In a systematic review and meta-analysis of epidemiological time-series studies (61 studies, 40 investigated daily mortality, and 27 hospital admissions)

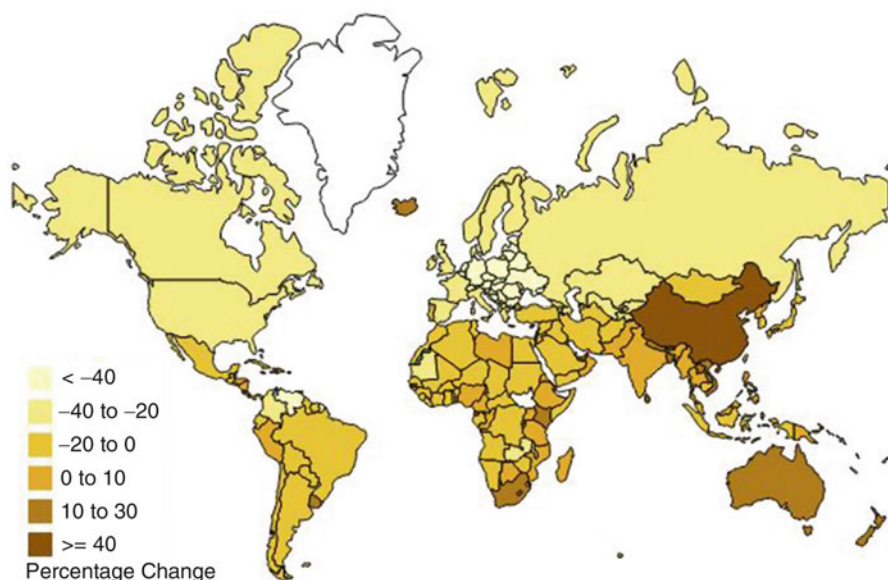


Fig. 8 Percentage change in mean annual exposure concentration of $PM_{2.5}$ between 1990 and 2010. Darker region indicates positive increase in $PM_{2.5}$ exposure, whereas lighter region indicates decrease in $PM_{2.5}$ exposure in the last two decades (World Bank 2015)

Table 2 Summary of studies examining the association between PM_{2.5} and health impacts

Study	Data collection period	Location	Sample size	Age	Method/model	Results	Findings	References
Global burden of mortality due to PM _{2.5}		Global		All ages	Global atmospheric chemical transport model	3.50 ± 0.90 million cardiopulmonary and 220,000 ± 80,000 lung cancer mortalities annually	Anthropogenic PM _{2.5} contributes substantially to global premature mortality	Anenberg et al. (2010)
Source-specific daily PM _{2.5} mass and hospital admissions	2001–2002	New York City, USA		≥65.0	Time-series generalized linear model	Source-related PM _{2.5} (specifically steel and traffic) was significantly associated with hospital admissions	Toxicity of PM _{2.5} depends on the source	Lall et al. (2011)
Mortality effects from PM size fractions	2005–2009	Beijing, China			Poisson generalized additive models	10.0 µg m ⁻³ increase in PM _{2.5} levels is associated with a 0.65 % (95 % CI: 0.29–0.80 %), 0.63 % (95 % CI: 0.25–0.83 %), and 1.38 % (95 % CI: 0.51–1.71 %) increase in non-accidental, respiratory and circulatory mortality, respectively	Significant associations between daily mortality with PM _{2.5}	Li et al. (2013)

(continued)

Table 2 (continued)

Study	Data collection period	Location	Sample size	Age	Method/model	Results	Findings	References
Short-term associations between $PM_{2.5}$ and mortality	2000–2006	9 French cities	548,474	15.0–74.0, >74.0	Poisson regression model	10.0 $\mu g m^{-3}$ increase in $PM_{2.5}$ results in increase of (+0.7 % [−0.1; 1.6]) on all-ages non-accidental mortality	Significant association between short-term impacts of $PM_{2.5}$ on mortality	Pascal et al. (2014)
Traffic related air toxics and low birth weight (LBW)	2004–2006	Los Angeles, USA	220,528		Logistic regression	Approximately 5.00 % increase in adjusted odds of term LBW per IQR increase was associated with exposures to $PM_{2.5}$	Significant association between $PM_{2.5}$ with fetal growth	Wilhelm et al. (2012)
PM exposure with arterial BP and hypertension	2000–2003	Bochum, Essen, and Mülheim, Germany	4291	45.0–75.0	Generalized additive models	IQR increase in $PM_{2.5}$ ($2.40 \mu g m^{-3}$) was associated with estimated increases in mean systolic and diastolic BP of 1.40 mmHg (95 % CI: 0.5, 2.3) and 0.9 mmHg (95 % CI: 0.4, 1.4), respectively	Significant association between long-term exposure to PM with increased arterial BP and hypertension	Fuks et al. (2011)

Ambient air and traffic pollution and the presence of depressive symptoms	2005–2008	Boston, USA	765	≥ 65.0	Epidemiological studies depression scale	OR of CES-D-R score ≥ 16.0 of 0.67 (95 % CI: 0.46, 0.98) per IQR (3.04 $\mu\text{g m}^{-3}$) increase in $\text{PM}_{2.5}$ over the 2 weeks preceding assessment	No association between exposure to ambient air pollutants with depressive symptoms	Wang et al. (2014)
Fine particulate air pollution and hospital admission	1999–2002	204 US urban counties	11.5 Million	> 65.0	Bayesian 2-stage hierarchical models and single lag and distributed lag over dispersed Poisson regression models	1.28 % (95 % CI, 0.78 %–1.78 %) increase in risk of heart failure per 10.0 $\mu\text{g m}^{-3}$ increase in same-day $\text{PM}_{2.5}$	Significant association between short-term exposure to $\text{PM}_{2.5}$ with increases the risk for hospital admission for cardiovascular and respiratory diseases	Dominici et al. (2006)
Association between ambient air pollution and stillbirth	1998–2004	New Jersey, USA	1719	20.0–40.0	Time-stratified case-crossover design and conditional logistic regression	The relative odds of stillbirth increased with IQR increases in the mean concentrations of $\text{PM}_{2.5}$ (OR = 1.07, 95 % CI = 0.93–1.22) 2 days before delivery	Significant association between increased risk of stillbirth with short-term increases in air pollutants in the previous few days	Faiz et al. (2013)

(continued)

Table 2 (continued)

Study	Data collection period	Location	Sample size	Age	Method/model	Results	Findings	References
Long- and short-term effects of $PM_{2.5}$ exposures on population mortality	2000–2008	Massachusetts, USA			Time-series analysis	$10.0 \mu g m^{-3}$ increase in $PM_{2.5}$ exposure was associated with a 2.80 % increase in PM-related mortality (95 % CI = 2.0–3.5) for short-term exposure and 1.6 (CI = 1.50–1.80) for long-term exposure	Short-term effects of $PM_{2.5}$ exposures was more severe on population mortality	Kloog et al. (2013)
Reduction in (traffic policy-related) air pollution concentrations with changes in respiratory health	2008, 2010		661		Eight busy urban and four suburban background locations were selected for air pollution and respiratory health measurement	Airway resistance decreased with a decline in PM (9.0 % per IQR)	Reduction in (traffic policy-related) air pollution concentrations may lead to small improvements in respiratory health	Boogaard et al. (2013)
Long-term exposure to ambient air pollution and lung cancer incidence	2008–2011	9 European countries	312,944		Cox regression models	Hazard ratio of 1.18 (0.96–1.46) with per $5.00 \mu g m^{-3}$ increase in $PM_{2.5}$	Significant association between long-term exposure to ambient air pollution and lung cancer	Raaschou-Nielsen et al. (2013)

Long-term exposure to fine PM and all cause, lung cancer and cardiovascular mortality	1979–1983 and 1999–2000	Columbia and Puerto Rico	1.2 Million	≥ 30.0	Cox proportional hazards survival model	Each $10.0 \mu\text{g m}^{-3}$ increase in fine particulate air pollution was associated with approximately a 4.0, 6.0, and 8.0 % increase risk of all cause, cardiopulmonary and lung cancer mortality	Significant association between long-term exposure to fine particulate air pollution and lung cancer and cardiovascular mortality	Pope et al. (2002)
Association between particulate air pollution and cardiovascular diseases	2008–2009	Karachi, Pakistan		$40.0 < \text{Age} < 60.0$	Generalized linear model (GLM) using negative binomial regression	1.60 % Increase in both hospital admissions and emergency room visits for cardiovascular disease with each $10.0 \mu\text{g m}^{-3}$ increase in $\text{PM}_{2.5}$ concentration	Higher levels of fine PM were associated with a striking elevation in rates of ER visits and hospitalizations for cardiovascular diseases	Khwaja et al. (2012)

IQR interquartile range, *CI* confidence interval, *HR* hazard ratio, *PI* posterior interval, *OR* odds ratio

Atkinson et al. (2015) assessed daily mortality and hospital admissions due to PM pollution. Morakinyo et al. (2016) identified the role of biological and chemical components of inhalable and respirable PM toxicity.

7.2 *Evidences from Recent Health Effects Studies of Fine PM*

In a recent study by Wu et al. (2017) in Chinese men, sperm concentration and count were found to be adversely effected by fine PM whereas no changes were observed in sperm motility. The study concludes that fine PM pollution effects sperm development specifically semen quality. Lu et al. (2017) identified a positive association between PM_{2.5} exposure and glucose homeostasis during pregnancy in 3589 pregnant women in Chiayi City, Taiwan although short-term effect was non-significant. Chen et al. (2017) reported association between ambient PM_{2.5} and influenza incidence in China and found that a 10.0 $\mu\text{g m}^{-3}$ increase in PM_{2.5} was associated with relative risk (RR) of 1.01 (95 % CI: 1.00, 1.02), of influenza incidence that appeared at lag day 2. The authors also estimated that exposure to fine PM may contribute to 10.7 % of incident influenza cases. Ajmani et al. (2016) reported significant association between worsening of olfactory function with PM_{2.5}, 6-month average exposure (per 1-IQR increase in PM_{2.5}: OR 1.28, 95 % CI 1.05, 1.55) in home-dwelling US adults age 57–85 years. Mehta et al. (2016) studied long-term PM_{2.5} exposure effects on renal function in a cohort of older men in the Boston Metropolitan area and found a 2.10 $\mu\text{g m}^{-3}$ interquartile range higher 1-year PM_{2.5} was associated with a 1.87 mL min⁻¹/1.73 m² lower estimated glomerular filtration rate [95 % confidence interval (CI): -2.99, -0.76] suggesting a reduction in renal function due to fine PM. In Seoul, Korea, Kim et al. (2016) assessed the relationship between long-term PM_{2.5} exposure and major depressive disorder in 27,270 participants 15–79 years of age from 2002 to 2010 and found risk of major depressive disorder was positively associated with long-term exposure to PM_{2.5}.

Results of meta-analysis by Flores-Pajot et al. (2016) found changes in autism spectrum disorder of 1.34 (95 % CI: 0.83, 2.17) with a 10.0 $\mu\text{g m}^{-3}$ increase in PM_{2.5} exposure. In a short-term PM_{2.5} exposure and infant mortality study in Japan Yorifuji et al. (2016) reported odds ratios of 1.06 (95 % confidence interval: 1.01–1.12) for infant mortality and 1.10 (1.02–1.19) for post-neonatal mortality for a 10.0 $\mu\text{g m}^{-3}$ increase in PM_{2.5}. Luo et al. (2015) in their systematic review and meta-analysis of short-term exposure to particulate air pollution and risk of myocardial infarction (MI) identified that 10.0 $\mu\text{g m}^{-3}$ increment in PM_{2.5} was associated with risk of MI (OR = 1.02; 95 % CI 1.01–1.03). Meta-analysis of 25 published epidemiological studies between maternal exposure to PM_{2.5} and pregnancy outcomes found a positive association between increase in PM_{2.5} concentrations and increase in the risk of low birth weight, preterm birth (PTB), and

small for gestational age (SGA) whereas the association was non-significant for stillbirth (Zhu et al. 2015). In a 5-year study from 2005 to 2009, in six counties in South-western Pennsylvania, authors found an increase in risk of childhood autism spectrum disorder for both prenatal and postnatal exposure to PM_{2.5} (Talbot et al. 2015).

7.3 Evidences from Global Health Effects Estimates of Fine PM

Evans et al. (2013) derived global PM_{2.5} exposure levels by utilizing remote sensing data of MODIS and MISR satellites, and found that the anthropogenic component of PM_{2.5} was responsible for a global fraction of adult mortality by 8.00 % (5.30–10.5) due to cardiopulmonary disease and 9.40 % (6.60–11.8) due to ischemic heart disease. Climate change can modify these effects more strongly than what was expected. Fang et al. (2013) found that twenty-first century climate change results in approximate global increases of 100,000 premature mortalities associated with PM_{2.5}. Satellite derived pollution data showed that the anthropogenic component of PM_{2.5} (95 % CI) was solely responsible for 12.8 % (5.90–18.5) increase in lung cancer-based adult mortality in the world (Evans et al. 2013). According to estimates of a global atmospheric chemical transport model (CTM) and health impact function, 75.0 % of excess mortalities in Asia and 17.0 % in Europe occurred because of high PM_{2.5} concentrations (Anenberg et al. 2010).

7.4 Evidences from Fine PM Exposure Health Effects Estimates

Hennig et al. (2014) found significant associations between long-term exposure to PM_{2.5} with high-sensitive C-reactive protein (hs-CRP), a marker of systemic inflammation with 4.53 % increase in hs-CRP concentration (95 % CI: 2.76, 6.33 %) per 1.00 µg m⁻³ increase in total PM_{2.5} in three German cities (Table 1). The study on short-term associations between PM_{2.5} and mortality in nine French cities by Pascal et al. (2014) clearly indicated that PM_{2.5} had a significant impact on cardiovascular mortality and the impact was highest during summer season. Fleisch et al. (2014) evaluated the association of second trimester and PM_{2.5} exposure in pregnant women in Boston, USA and found that PM_{2.5} exposure was significantly associated with impaired glucose tolerance (IGT), but not with gestational diabetes mellitus (GDM) and indicated the direct effect of PM in abnormal glycemia during pregnancy.

Harrison et al. (2004) found a significant association between long-term exposures to PM_{2.5} and mortality due to lung cancer based on American Cancer Society

cohort study. A significant association was found between long-term exposures to $\text{PM}_{2.5}$ with cardiovascular events in women in 36 US metropolitan areas (Miller et al. 2007). A 24.0 % increase in the risk of a cardiovascular event [Hazard Ratio (HR) = 1.24; 95 % CI, 1.09 to 1.41] and 76.0 % increase in the risk of death from cardiovascular disease (HR = 1.76; 95 % CI, 1.25 to 2.47) were associated with a $10 \mu\text{g m}^{-3}$ increase in $\text{PM}_{2.5}$ (Miller et al. 2007). In Karachi, Pakistan, Khwaja et al. (2012) found an increase in hospital admissions by 1.60 % and emergency room visits for cardiovascular disease by 1.60 % with each $10.0 \mu\text{g m}^{-3}$ increase in $\text{PM}_{2.5}$ concentrations. The Heinz Nixdorf Recall Study, a population-based prospective cohort in Germany found a positive association between long-term urban background $\text{PM}_{2.5}$ and arterial BP and hypertension, which may induce atherosclerosis (Fuks et al. 2011). Short-term effects of $\text{PM}_{2.5}$ on cause-specific hospital admission were investigated by Dominici et al. (2006) in 204 US urban counties and they reported that reducing $\text{PM}_{2.5}$ concentrations by $10.0 \mu\text{g m}^{-3}$ would reduce the number of hospitalizations for heart failure by 3156.

7.5 Evidences from Genotoxic Health Effects Estimates of Fine PM

Human bronchial epithelial (HBE) cells when exposed to $\text{PM}_{2.5}$ samples collected from Wuhan, China showed changes in 970 and 492 genes at lower and higher exposures (Ding et al. 2014). These changes were mostly associated with genes of inflammatory and immune responses, oxidative stress response, and response to DNA damage. Similar variable responses in inflammation-related genes in the bronchial epithelial cell line were also observed by Øvrevik et al. (2009). Baulig et al. (2009) and Cachon et al. (2014) also found associations between pro-inflammatory response in airway epithelial cells and aqueous extracts of $\text{PM}_{2.5}$. Cachon et al. (2014) observed cell cycle alterations by fine PM in human bronchial epithelial cells.

8 Health Effects of Chemical Constituents of $\text{PM}_{2.5}$

Major components present in the fine PM are typically similar in different geographic regions of the world but proportions of different components vary significantly with different emissions and local sources (Chatterjee et al. 2012; Gaita et al. 2014; Raja et al. 2010). The composition and properties of PM influence its toxicity apart from its aerodynamic diameter, exposure pathway, and alveolar deposition capacity (Mazzoli-Rocha et al. 2010). PM contains a diverse array of chemical and biological constituents such as carbonaceous (organic and elemental carbon, aldehydes, PAH, nitro-PAH, ketones, quinones, hydrocarbons), inorganic

(sulfate, nitrate, ammonia, quartz, silica, sea salt, mineral oxides), and biological (bacteria, pollen, fungi, virus, plant debris) components (Ghio et al. 2012) (Fig. 9).

Rohr and Wyzga (2012) reviewed 48 independent epidemiological studies to assess different health effects of PM components. Among different PM components studied, carbonaceous components of PM were found to be maximally associated with negative health effects. The study also identified that effects were more prominent for cardiovascular anomalies. Metals such as nickel (Ni), vanadium (V), zinc (Zn), copper (Cu), silicon (Si), and potassium (K) showed maximum negative health effects. V and Ni were found to be more toxic for both respiratory and cardiovascular diseases whereas Al and Si were more prominent in respiratory anomalies. Iron (Fe), Zn, sulfur (S), and lead (Pb) showed the least associations with negative health effects amongst the metals studied. Authors also found contradictions in health effects related to sulfate which were probably due to spatial variations in those studies. Cakmak et al. (2014) reported acute changes in cardiovascular and respiratory physiology with metals in PM_{2.5}. There were significant increases in heart rate and blood pressure and decrease in lung function with IQR increases in calcium (Ca), cadmium (Cd), Pb strontium (Sr), tin (Sn), V, and Zn levels in fine PM. Greene and Morris (2006) found lifetime excess lung cancer risk due to chromium (Cr) and arsenic (As) in PM_{2.5} in Washington, DC, USA (Fig. 9).

Mazzoli-Rocha et al. (2010) reviewed the association between oxidative stress and impairment of the function of the lung exposed to PM and found PM constituents specifically PAH and HM as the major elicitors of oxidative stress and respiratory diseases. Breyse et al. (2013) identified BC, OC, SVOCs, Ni, and V as important components of PM toxicity and also concluded that the secondary aerosol formation may enhance PM toxicity. Miyata and van Eeden (2011) identified major soluble metals such as Fe, V, Ni, and Zn to be mainly responsible for ROS generation. de Kok et al. (2006) reported PAH concentration as the most important factor in determining the radical generating capacity compared to total metal content or transition metal content in PM.

In a review Valavanidis et al. (2008) identified almost 500 different organic compounds with mutagenic potential. Mutagenicity was mostly due to the presence of polar or highly polar compounds with an aromatic nitro group, amines, and aromatic ketones. DNA-reactivity was found to be positively correlated with concentrations of total PAH and transition metals indicating that chemical components of PM directly influence DNA strand break (de Kok et al. 2006). Significant quantitative and qualitative differences in cytokine/chemokine responses to PM components in bronchial epithelial cell line were observed when effects of PM components were assessed on the expression of 84 inflammation-related genes Øvreivik et al. (2009).

Stanek et al. (2011) examined relationships between PM components and health effects from 29 epidemiological studies and found vehicular emission, biomass burning, and road dust sources contributing maximally to cardiovascular diseases but no such clear associations were observed with metals, secondary sulfate, and salt components in PM. SOA represents a significant part of PM_{2.5}, as we have already discussed in Section 6 but health effects of SOA are limited and only

reported from a few regions of the world. Rappazzo et al. (2015) studied associations between chemical component of $PM_{2.5}$ with risk of preterm birth (PTB) in three cities of the USA and found consistent associations between EC and sulfate with risk of PTB, whereas for nitrate, an association was only found for the first trimester and no significant association was observed for organic carbon. Nitrate levels in $PM_{2.5}$ showed an association with an increase in monthly mortality in a 7-year study of 12.5 million Medicare enrollees in eastern USA for the age group above 65 years (Chung et al. 2015). In a traffic related air toxic study, major components of SOAs such as organic carbon and ammonium nitrate in $PM_{2.5}$ showed a positive association with preterm birth in women living in Southern California, USA (Wilhelm et al. 2011).

Exposure to airborne PAHs was found to be adversely affecting the children's cognitive development by 5 years of age in a prospective cohort study in Krakow, Poland (Edwards et al. 2010). Results of a New York City cohort study showed distinct effects in child behavior with prenatal exposure to PAH (Perera et al. 2012) (Fig. 9). Perera et al. (2009) observed a significant inverse relationship between high/low PAH exposure and full-scale and verbal IQ score (Fig. 10). Hong et al. (2016) estimated the lung cancer risk of atmospheric PAH in five Asian countries and found a higher lung cancer risk in China and Vietnam compared to India, Japan, and South Korea.

Biological components in PM can also cause severe health effects directly through allergic pollens in PM or by the presence of bacterial or fungal toxin in PM that after inhalation can induce several inflammatory responses (Morakinyo et al. 2016). Degobbi et al. (2011) reviewed the role of endotoxin (cell wall component of gram-negative bacteria) in PM toxicity and found endotoxin as a modulator for immunological response by increasing pro-inflammatory cytokine expression. The study also identified an increment in response of several cytokines and chemokine's expressions due to the presence of endotoxin in PM.

Krall et al. (2015) reviewed the current methods and challenges in epidemiological studies on associations between PM constituents and health effects and found three major challenges like spatial-temporal variations in exposure, identifying effects of individual PM components and error in measurement techniques. Based on the epidemiological and toxicological findings it is clear that the toxicity of PM depends on the combination of all PM components and their interactions with each other. The information is still limited due to heterogeneity in PM components concentrations in different studies, exposure doses, and specific measurement techniques. Therefore, further researches are required to identify stronger associations between PM components and health effects.

9 Mechanism of Fine PM Toxicity

Most of the studies have reported oxidative stress as the primary effect of PM-related toxicity (Salvi and Holgate 1999; Øvrevik et al. 2015). Reactive oxygen species (ROS) cause significant damage to tissues and further induce different signaling cascades in this process. The mechanism follows the activation of transcription factors which induce genes of pro-inflammatory response in most cases. The intensity of oxidative stress depends upon the size of fine PM as well as its chemical constituents. PAH, organic constituents, and heavy metals severely intensify these effects (Bai et al. 2007; Øvrevik et al. 2015). When PM interacts with airway epithelial cells and alveolar macrophages it raises the levels of cytokine like IL-interleukin-6 (IL-6), granulocyte-macrophage colony-stimulating factor (GMCSF), IL-1 β which results in local or systematic inflammation (Block and Calderón-Garcidueñas 2009).

Araujo (2011) reviewed the association of PM and its components with systemic oxidative stress, inflammation, and atherosclerosis based on epidemiological and experimental evidences. The author suggested three different mechanisms by which PM causes systemic cardiovascular irregularities; pulmonary and systemic inflammation; activation of receptors of pulmonary receptors that alters the autonomic nervous system, and entree of PM components to systemic circulation. It was further suggested that the effects of PM and its components are collaborating and even gaseous pollutants play a significant role in health effects.

Mazzoli-Rocha et al. (2010) reviewed the role of oxidative stress in signaling and inflammation by PM. It was found that PM-induced generation of ROS activates the redox responsive signaling pathway (mitogen-activated protein kinase (MAPK) family) and Ca^{++} influx, which further stimulate transcription factor and expression of genes related to inflammation or DNA damage in inflammatory cells and alveolar macrophages that ultimately result in cell injury or apoptosis, leading to respiratory morbidity and mortality. Similar observations were also made by Ghio et al. (2012) and Breyse et al. (2013) in their studies of uncertainties in the health effects caused by airborne particulate matter. Major outcomes of the studies were (1) identification of the role of epigenetic mechanisms in PM-induced toxicity, (2) role of respiratory inflammation by fine PM induces asthmatic responses, (3) ROS as an important modulator in induction of cellular response, (4) increment in pro-inflammatory and immune responses, (5) changes in concentrations of plasma high-density lipoprotein, (6) increase in coronary vascular resistance and decrease in myocardial perfusion causing acute myocardial infarction, (7) induction in expression of genes related to innate immunity, genes of complement system pathways and chemotaxis, (8) intrusion of eosinophil and neutrophil to airways, and (9) elevated secretions of cytokines (Breyse et al. 2013).

Ghio et al. (2012) further identified PM-induced responses such as the role of kinase cascade (ERK, p38, and Jun kinases) in PM-induced cell responses, oxidant generation by activation of NADPH oxidases, the role of metals in electron transport and ROS generation, induction in expression of stress response enzymes

(glutathione transferase, heme oxygenase, and superoxide dismutase) in epithelial cells and macrophages, mitochondrial dysfunction, and increased expression of cytochrome P-450.

In their review on the toxicological assessment of airborne PM, Valavanidis et al. (2008) identified several different mechanisms of toxicity such as ROS generation, DNA oxidative damage, mutagenicity, and induction of pro-inflammatory factors (cytokines and chemokines). Valavanidis et al. (2008) in their review concluded that all the studied relationships between PM and genotoxicity showed positive DNA damage, oxidative DNA damage, micronuclei sister chromatid exchange, and single-strand breaks. de Kok et al. (2006) also found fine PM to exert high DNA-reactivity.

Miyata and van Eeden (2011) reviewed the immunological interactions between alveolar macrophages and PM and found fine PM to induce innate immune responses by ROS generation through activation of different transcription factors. After particles are internalized, adaptive immunity is induced through expression of major histocompatibility complex Class II and modulation in response of T helper cells. Feng et al. (2016) identified oxidative stress and inflammation with alteration in immune responses as the major mechanism behind fine PM-induced respiratory effects. Oxidative stress and inflammation in hypothalamus may cause alteration in its neuroendocrine function and ultimately may lead to neuroendocrine disorders. Alteration in immune response in pregnant women due to PM exposure may induce adverse effects in childhood or at maturity (Feng et al. 2016). Increase in expression of phase I xenobiotic-metabolizing cytochrome P450 enzymes due to organic components of fine PM and inflammatory response in other organs such as spleen, heart, kidney, and liver due to fine PM are reported (Feng et al. 2016) (Fig. 9).

Øvrevik et al. (2015) identified interactions of particles with lipid bilayer, cell surface receptors, intracellular molecules, and direct formation of ROS, resulting in activation of genes responsible for process of inflammation in airway mucosa cells. This inflammation itself can trigger cardiovascular effects by transport of inflammatory mediators through circulation (Øvrevik et al. 2015). Laing et al. (2010) observed that PM_{2.5} exposure resulted in endoplasmic reticulum stress and unfolded protein response (UPR) signaling pathway in lung and liver tissues and in mouse macrophage cells and they concluded that PM_{2.5} can activate UPR-related branches which further lead to apoptosis by the PERK-eIF2-CHOP pathway. The mechanisms of toxicity of PM and their effects are shown in Fig. 10.

Based on the above evidences, it can be concluded that the actual mechanisms and components of fine PM responsible for such health effects after exposure of biological systems to PM are still not completely known and further evidences at gene and molecular levels should be studied.

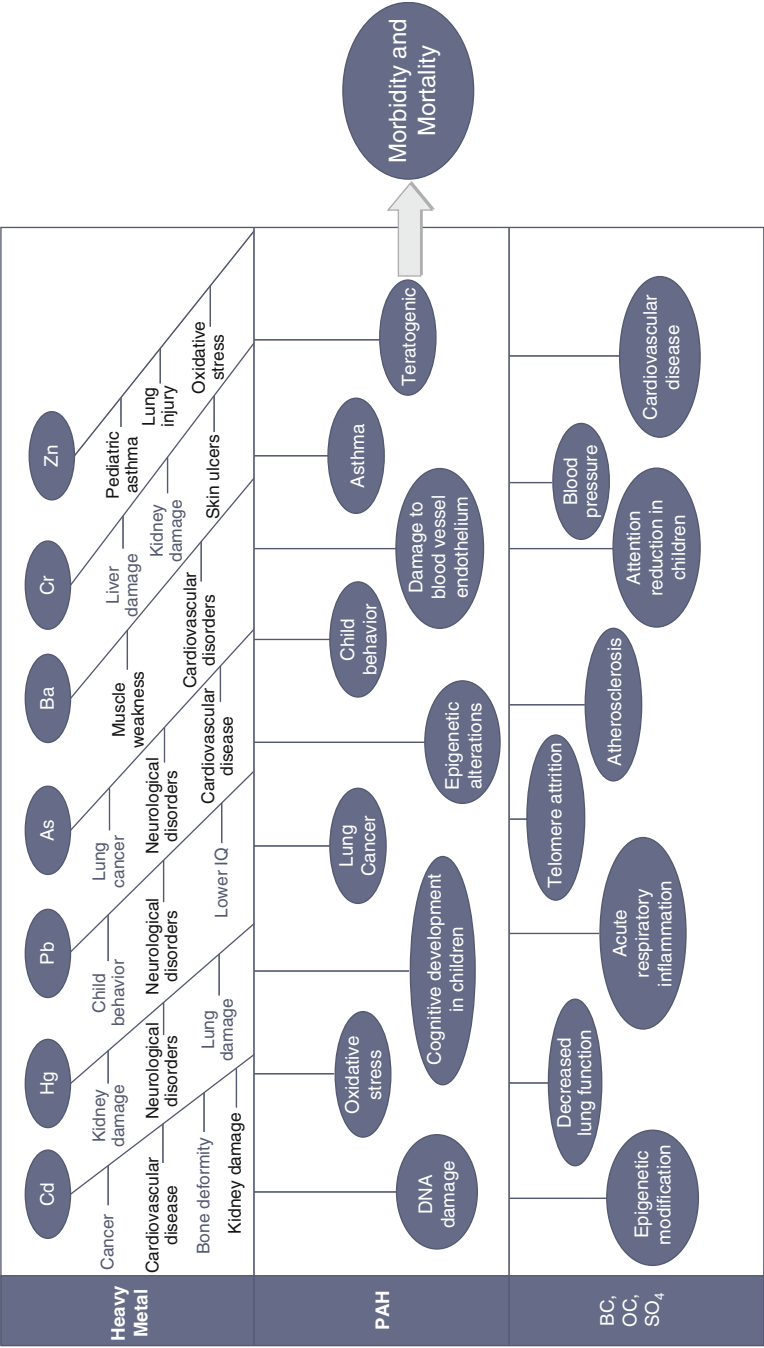


Fig. 9 Health effects of different PM_{2.5} chemical constituents

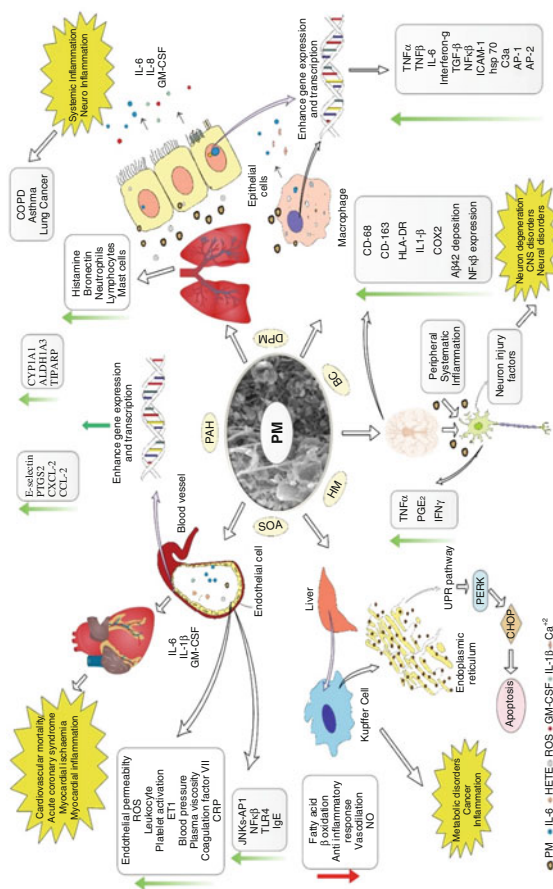


Fig. 10 Different pathways of PM toxicity and their effects in different organ systems. PM particulate matter, PAH polycyclic aromatic hydrocarbons, DPM diesel particulate matter, SOA secondary organic aerosol, HM heavy metal, BC black carbon, ROS reactive oxygen species, ET-1 endothelin-1, CRP C-reactive protein, JNKs-AP1 c-Jun N-terminal kinases activation protein 1, NO nitric oxide, NF-κB nuclear factor-κB, TLR-4 toll-like receptor 4, IgE immunoglobulin E, UPR unfolded protein response, PERK protein kinase-like ER kinase, CHOP C/EBP homologous protein, IL-6 interleukin 6, IL-1β interleukin 1β, GM-CSF granulocyte-macrophage colony stimulating factor, TNF-α tumor necrosis factor α, PGE₂ prostaglandin E2, IFN-γ interferon γ, CCL-2 chemokine (C-C motif) ligand 2, CXCL-2 chemokine (C-X-C motif) ligand 2, PTGS2 prostaglandin-endoperoxide synthase 2, CYP1A1 cytochrome P450, family 1, subfamily A, polypeptide 1, ALDH1A3 aldehyde dehydrogenase 1 family, member A3, TIPARP TCDD-inducible poly(ADP-ribose) polymerase, AP-1 activator protein 1, AP-2 activator protein 2, C3a complement component 3a, HSP70 heat shock protein 70, ICAM1 intercellular adhesion molecule-1, TGF-β transforming growth factor beta, NF-κB nuclear factor κB, β₂ amyloid beta-42, COX₂ cyclooxygenase 2, HLA-DR human leukocyte antigen-D related, HETE hydroxyicosatetraenoic acid. (Modified from Bai et al. 2007; Block and Calderón-Garcidueñas 2009; Laing et al. 2010; Aung et al. 2011)

10 Suggestions

Cleaning our environment is the biggest and topmost priority in the current scenario; choices should be economical and implementable with long-term solutions. Identifying problems is the first step to solution. We know the consequences that we are facing every day, so we must bring new ideas to overcome this burden. The following suggestions can help to mitigate the PM levels and consequently improve human health. Suggestions are proposed from best to least effective measure:

1. Strict emission standards for fine PM emission from vehicles and industries.
2. Prevention of biomass burning.
3. More emphasis should be given on health effects study especially in Asian and African countries.
4. School building should not be near highway or any major roadway.
5. More scientific research on passive monitoring techniques should be conducted as it is economical and easy to implement in developing countries.
6. Large urban forestry development programs.
7. Use of bio-filters to reduce emissions from industry or around traffic sites.
8. Creating a global PM_{10} , $PM_{2.5}$, and PM_1 standards for different regions based on its topography and history.
9. Epidemiological monitoring of air quality effects as a part of national ambient air quality monitoring program especially for developing countries.
10. Creating a global database of PM levels and their sources.

11 Conclusion

Fine PM has become a major public health issue, particularly in large cities. Most of the large cities in the world showed PM levels above the respective standards of their own countries as well as the WHO standards. Levels were less elevated in the USA and European cities. Asian cities are mostly critical with consistently higher $PM_{2.5}$ levels and exposure. Most of the major cities in Asia have exceeded the threshold levels. Even rural and remote areas have higher PM levels, which indicate a rapid dispersion from urban centers. High population density and urbanization are the major drivers of poor air quality around the world. Automobiles along with combustion activities are major sources of $PM_{2.5}$.

Epidemiological studies provided evidence that there is an increasing trend of fine PM-related health issues all over the world. Traffic seems to be the major factor behind health anomalies around the globe with children and pregnant women being most vulnerable. Several studies indicated the carcinogenic or mutagenic nature of chemical constituents associated with fine PM that have severe health consequences even at lower concentrations. African and Asian countries require more epidemiological studies to provide a broader perspective of health effects. More scientific

studies on gene and metabolic levels will further be able to discover the yet hidden health effects of fine PM.

12 Summary

Global status, trend, and health effects of fine particulate matter (PM_{2.5}) were reviewed. Asian and African continents have higher exceedance of fine PM than the standards compared to Europe and the USA. Traffic, biomass burning, road dust, and local sources affect fine PM concentrations. Epidemiological studies provided clear evidence of increasing fine PM-related health issues all over the world. Fine PM causes several health effects such as increase in inflammatory responses, lung cancer, heart rate variability, term low birth weight, mutagenicity, changes in gene expression, and immune responses. Chemical constituents of fine PM such as polycyclic aromatic hydrocarbons, heavy metals, and elemental carbon are mostly responsible for PM_{2.5} toxicity. Emission reduction policies should be more emphasized with epidemiological studies to combat negative effect of fine PM.

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References

- Ajmani GS, Suh HH, Wroblewski KE et al (2016) Fine particulate matter exposure and olfactory dysfunction among urban-dwelling older US adults. *Environ Res* 151:797–803. doi:[10.1016/j.envres.2016.09.012](https://doi.org/10.1016/j.envres.2016.09.012)
- Akyüz M, Çabuk H (2009) Meteorological variations of PM_{2.5}/PM₁₀ concentrations and particle-associated polycyclic aromatic hydrocarbons in the atmospheric environment of Zonguldak, Turkey. *J Hazard Mater* 170:13–21. doi:[10.1016/j.jhazmat.2009.05.029](https://doi.org/10.1016/j.jhazmat.2009.05.029)
- Alolayan MA, Brown KW, Evans JS et al (2013) Source apportionment of fine particles in Kuwait City. *Sci Total Environ* 448:14–25. doi:[10.1016/j.scitotenv.2012.11.090](https://doi.org/10.1016/j.scitotenv.2012.11.090)
- Anenberg SC, Horowitz LW, Tong DQ, West JJ (2010) An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. *Environ Health Perspect* 118:1189–1195. doi:[10.1289/ehp.0901220](https://doi.org/10.1289/ehp.0901220)
- Antonel J, Chowdhury Z (2014) Measuring ambient particulate matter in three cities in Cameroon, Africa. *Atmos Environ* 95:344–354. doi:[10.1016/j.atmosenv.2014.06.053](https://doi.org/10.1016/j.atmosenv.2014.06.053)
- Apte JS, Marshall JD, Cohen AJ, Brauer M (2015) Addressing global mortality from ambient PM_{2.5}. *Environ Sci Technol* 49:8057–8066. doi:[10.1021/acs.est.5b01236](https://doi.org/10.1021/acs.est.5b01236)
- Araujo JA (2011) Particulate air pollution, systemic oxidative stress, inflammation, and atherosclerosis. *Air Qual Atmos Health* 4:79–93. doi:[10.1007/s11869-010-0101-8](https://doi.org/10.1007/s11869-010-0101-8)

- Arruti A, Fernández-Olmo I, Irabien Á (2012) Evaluation of the urban/rural particle-bound PAH and PCB levels in the northern Spain (Cantabria region). *Environ Monit Assess* 184:6513–6526. doi:[10.1007/s10661-011-2437-4](https://doi.org/10.1007/s10661-011-2437-4)
- Atkinson RW, Mills IC, Walton HA, Anderson HR (2015) Fine particle components and health—a systematic review and meta-analysis of epidemiological time series studies of daily mortality and hospital admissions. *J Expo Sci Environ Epidemiol* 25:208–214. doi:[10.1038/jes.2014.63](https://doi.org/10.1038/jes.2014.63)
- Aung HH, Lame MW, Gohil K et al (2011) Comparative gene responses to collected ambient particles in vitro: endothelial responses. *Physiol Genomics* 43:917–929. doi:[10.1152/physiolgenomics.00051.2011](https://doi.org/10.1152/physiolgenomics.00051.2011)
- Baek BH, Koziel JA, Aneja VP (2006) A preliminary review of gas-to-particle conversion monitoring and modelling efforts in the USA. *Int J Glob Environ Issues* 6:204–230
- Bai N, Khazaei M, van Eeden SF, Laher I (2007) The pharmacology of particulate matter air pollution-induced cardiovascular dysfunction. *Pharmacol Ther* 113:16–29
- Balasubramanian R, Qian W-B, Decesari S et al (2003) Comprehensive characterization of PM_{2.5} aerosols in Singapore. *J Geophys Res Atmos* 108:4523. doi:[10.1029/2002JD002517](https://doi.org/10.1029/2002JD002517)
- Baulig A, Singh S, Marchand A et al (2009) Role of Paris PM_{2.5} components in the pro-inflammatory response induced in airway epithelial cells. *Toxicology* 261:126–135. doi:[10.1016/j.tox.2009.05.007](https://doi.org/10.1016/j.tox.2009.05.007)
- Begum BA, Hopke PK, Markwitz A (2013) Air pollution by fine particulate matter in Bangladesh. *Atmos Pollut Res* 4:75–86. doi:[10.5094/APR.2013.008](https://doi.org/10.5094/APR.2013.008)
- Begum BA, Hossain A, Nahar N et al (2012) Organic and black carbon in PM_{2.5} at an urban site at Dhaka, Bangladesh. *Aerosol Air Qual Res* 12:1062–1072. doi:[10.4209/aaqr.2012.05.0138](https://doi.org/10.4209/aaqr.2012.05.0138)
- Bell ML, Zanobetti A, Dominici F (2013) Evidence on vulnerability and susceptibility to health risks associated with short-term exposure to particulate matter: a systematic review and meta-analysis. *Am J Epidemiol* 178:865–876. doi:[10.1093/aje/kwt090](https://doi.org/10.1093/aje/kwt090)
- Block ML, Calderón-Garcidueñas L (2009) Air pollution: mechanisms of neuroinflammation & CNS disease. *Trends Neurosci* 29:506–516. doi:[10.1016/j.tins.2009.05.009](https://doi.org/10.1016/j.tins.2009.05.009)
- Boogaard H, Fischer PH, Janssen NAH et al (2013) Respiratory effects of a reduction in outdoor air pollution concentrations. *Epidemiology* 24:753–761. doi:[10.1097/EDE.0b013e31829e1639](https://doi.org/10.1097/EDE.0b013e31829e1639)
- Breyse PN, Delfino RJ, Dominici F et al (2013) US EPA particulate matter research centers: summary of research results for 2005–2011. *Air Qual Atmos Health* 6:333–355. doi:[10.1007/s11869-012-0181-8](https://doi.org/10.1007/s11869-012-0181-8)
- Cachon BF, Firmin S, Verdin A et al (2014) Proinflammatory effects and oxidative stress within human bronchial epithelial cells exposed to atmospheric particulate matter (PM_{2.5} and PM_{>2.5}) collected from Cotonou, Benin. *Environ Pollut* 185:340–351. doi:[10.1016/j.envpol.2013.10.026](https://doi.org/10.1016/j.envpol.2013.10.026)
- Cakmak S, Dales R, Kauri LM et al (2014) Metal composition of fine particulate air pollution and acute changes in cardiorespiratory physiology. *Environ Pollut* 189:208–214. doi:[10.1016/j.envpol.2014.03.004](https://doi.org/10.1016/j.envpol.2014.03.004)
- Chafe ZA, Brauer M, Klimont Z et al (2014) Household cooking with solid fuels contributes to ambient PM_{2.5} air pollution and the burden of disease. *Environ Health Perspect* 122:1314–1320. doi:[10.1289/ehp.1206340](https://doi.org/10.1289/ehp.1206340)
- Chaloulakou A, Kassomenos P, Grivas G, Spyrellis N (2005) Particulate matter and black smoke concentration levels in Central Athens, Greece. *Environ Int* 31:651–659. doi:[10.1016/j.envint.2004.11.001](https://doi.org/10.1016/j.envint.2004.11.001)
- Chatterjee A, Dutta C, Jana TK, Sen S (2012) Fine mode aerosol chemistry over a tropical urban atmosphere: characterization of ionic and carbonaceous species. *J Atmos Chem* 69:83–100. doi:[10.1007/s10874-012-9231-8](https://doi.org/10.1007/s10874-012-9231-8)
- Chen G, Zhang W, Li S et al (2017) The impact of ambient fine particles on influenza transmission and the modification effects of temperature in China: a multi-city study. *Environ Int* 98:82–88. doi:[10.1016/j.envint.2016.10.004](https://doi.org/10.1016/j.envint.2016.10.004)

- Chen R, Wang X, Meng X et al (2013) Communicating air pollution-related health risks to the public: an application of the air quality health index in Shanghai, China. *Environ Int* 51:168–173. doi:[10.1016/j.envint.2012.11.008](https://doi.org/10.1016/j.envint.2012.11.008)
- Cheng YH, Lin CC, Liu JJ, Hsieh CJ (2014) Temporal characteristics of black carbon concentrations and its potential emission sources in a southern Taiwan industrial urban area. *Environ Sci Pollut Res* 21:3744–3755. doi:[10.1007/s11356-013-2373-7](https://doi.org/10.1007/s11356-013-2373-7)
- Chuersuwan N, Nimrat S, Lekphet S, Kerdumrai T (2008) Levels and major sources of PM_{2.5} and PM₁₀ in Bangkok metropolitan region. *Environ Int* 34:671–677. doi:[10.1016/j.envint.2007.12.018](https://doi.org/10.1016/j.envint.2007.12.018)
- Chung Y, Dominici F, Wang Y et al (2015) Associations between long-term exposure to chemical constituents of fine particulate matter (PM_{2.5}) and mortality in medicare enrollees in the eastern United States. *Environ Health Perspect* 123:467–474. doi:[10.1289/ehp.1307549](https://doi.org/10.1289/ehp.1307549)
- CNEM (2013) China National Environmental Monitoring Center. <http://www.cnemc.cn>. Accessed 20 Jan 2017
- Co HX, Dung NT, Oanh NTK et al (2014) Levels and composition of ambient particulate matter at a mountainous rural site in Northern Vietnam. *Aerosol Air Qual Res* 14:1917–1928. doi:[10.4209/aaqr.2013.09.0300](https://doi.org/10.4209/aaqr.2013.09.0300)
- Cohen DD, Crawford J, Stelcer E, Bac VT (2010) Characterisation and source apportionment of fine particulate sources at Hanoi from 2001 to 2008. *Atmos Environ* 44:320–328. doi:[10.1016/j.atmosenv.2009.10.037](https://doi.org/10.1016/j.atmosenv.2009.10.037)
- Coronas MV, Pereira TS, Rocha JAV et al (2009) Genetic biomonitoring of an urban population exposed to mutagenic airborne pollutants. *Environ Int* 35:1023–1029. doi:[10.1016/j.envint.2009.05.001](https://doi.org/10.1016/j.envint.2009.05.001)
- CPCB (2009) National Ambient Air Quality Standards. Central Pollution Control Board India. http://cpcb.nic.in/National_Ambient_Air_Quality_Standards.php. Accessed 21 Jan 2017
- CPCB (2013) CPCB Annual Report 2011–2012. Central Pollution Control Board Ministry of Environment & Forest, New Delhi. 284 p
- Dadvand P, Ostro B, Figueras F et al (2014) Residential proximity to major roads and term low birth weight: the roles of air pollution, heat, noise, and road-adjacent trees. *Epidemiology* 25:518–525. doi:[10.1097/EDE.0000000000000107](https://doi.org/10.1097/EDE.0000000000000107)
- Daher N, Ruprecht A, Invernizzi G et al (2012) Characterization, sources and redox activity of fine and coarse particulate matter in Milan, Italy. *Atmos Environ* 49:130–141. doi:[10.1016/j.atmosenv.2011.12.011](https://doi.org/10.1016/j.atmosenv.2011.12.011)
- de Kok TCM, Driessens HAL, Hogervorst JGF, Briedé JJ (2006) Toxicological assessment of ambient and traffic-related particulate matter: a review of recent studies. *Mutat Res* 613:103–122. doi:[10.1016/j.mrrev.2006.07.001](https://doi.org/10.1016/j.mrrev.2006.07.001)
- de Miranda RM, de Fatima AM, Fornaro A et al (2012) Urban air pollution: a representative survey of PM 2.5 mass concentrations in six Brazilian cities. *Air Qual Atmos Health* 5:63–77. doi:[10.1007/s11869-010-0124-1](https://doi.org/10.1007/s11869-010-0124-1)
- Degobbi C, Saldiva PHN, Rogers C (2011) Endotoxin as modifier of particulate matter toxicity: a review of the literature. *Aerobiologia* 27:97–105. doi:[10.1007/s10453-010-9179-6](https://doi.org/10.1007/s10453-010-9179-6)
- Deshmukh DK, Deb MK, Suzuki Y, Kouvarakis GN (2013) Water-soluble ionic composition of PM_{2.5-10} and PM_{2.5} aerosols in the lower troposphere of an industrial city Raipur, the eastern Central India. *Air Qual Atmos Health* 6:95–110. doi:[10.1007/s11869-011-0149-0](https://doi.org/10.1007/s11869-011-0149-0)
- Ding X, Wang M, Chu H et al (2014) Global gene expression profiling of human bronchial epithelial cells exposed to airborne fine particulate matter collected from Wuhan, China. *Toxicol Lett* 228:25–33. doi:[10.1016/j.toxlet.2014.04.010](https://doi.org/10.1016/j.toxlet.2014.04.010)
- Dionisio KL, Arku RE, Hughes AF et al (2010) Air pollution in Accra neighborhoods: spatial, socioeconomic, and temporal patterns. *Environ Sci Technol* 44:2270–2276. doi:[10.1021/es903276s](https://doi.org/10.1021/es903276s)
- Dominici F, Peng RD, Bell ML et al (2006) Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA* 295:1127–1134. doi:[10.1001/jama.295.10.1127](https://doi.org/10.1001/jama.295.10.1127)

- Dongarra G, Manno E, Varrica D et al (2010) Study on ambient concentrations of PM₁₀, PM_{10-2.5}, PM_{2.5} and gaseous pollutants. Trace elements and chemical speciation of atmospheric particulates. *Atmos Environ* 44:5244–5257. doi:[10.1016/j.atmosenv.2010.08.041](https://doi.org/10.1016/j.atmosenv.2010.08.041)
- Doumbia EHT, Lioussé C, Galy-Lacaux C et al (2012) Real time black carbon measurements in West and Central Africa urban sites. *Atmos Environ* 54:529–537. doi:[10.1016/j.atmosenv.2012.02.005](https://doi.org/10.1016/j.atmosenv.2012.02.005)
- ECC (2013) Environment and Climate Change Canada. Canadian Ambient Air Quality Standards. Government of Canada. <http://www.ec.gc.ca/default.asp?lang=En&n=56D4043B-1&news=A4B2C28A-2DFB-4BF4-8777-ADF29B4360BD>. Accessed 19 Jan 2017
- Edwards SC, Jedrychowski W, Butscher M et al (2010) Prenatal exposure to airborne polycyclic aromatic hydrocarbons and children's intelligence at 5 years of age in a prospective cohort study in Poland. *Environ Health Perspect* 118:1326–1331. doi:[10.1289/ehp.0901070](https://doi.org/10.1289/ehp.0901070)
- EEA (2013) AirBase: public air quality database – air pollution. European Environment Agency. <http://www.eea.europa.eu/themes/air/air-quality/map/airbase>. Accessed 5 Jan 2017
- Eeftens M, Tsai MY, Ampe C et al (2012) Spatial variation of PM_{2.5}, PM₁₀, PM_{2.5} absorbance and PM_{coarse} concentrations between and within 20 European study areas and the relationship with NO₂ – results of the ESCAPE project. *Atmos Environ* 62:303–317. doi:[10.1016/j.atmosenv.2012.08.038](https://doi.org/10.1016/j.atmosenv.2012.08.038)
- EQSJ (2009) Environmental quality standards in Japan – air quality. Ministry of the Government of Japan. <http://www.env.go.jp/en/air/aq/aq.html>. Accessed 28 Jan 2017
- Evans J, van Donkelaar A, Martin RV et al (2013) Estimates of global mortality attributable to particulate air pollution using satellite imagery. *Environ Res* 120:33–42. doi:[10.1016/j.envres.2012.08.005](https://doi.org/10.1016/j.envres.2012.08.005)
- Faiz AS, Rhoads GG, Demissie K et al (2013) Does ambient air pollution trigger stillbirth? *Epidemiology* 24:538–544. doi:[10.1097/EDE.0b013e3182949ce5](https://doi.org/10.1097/EDE.0b013e3182949ce5)
- Fang G, Chang S (2010) Atmospheric particulate (PM₁₀ and PM_{2.5}) mass concentration and seasonal variation study in the Taiwan area during 2000–2008. *Atmos Res* 98:368–377. doi:[10.1016/j.atmosres.2010.07.005](https://doi.org/10.1016/j.atmosres.2010.07.005)
- Fang Y, Mauzerall DL, Liu J et al (2013) Impacts of 21st century climate change on global air pollution-related premature mortality. *Clim Change* 121:239–253. doi:[10.1007/s10584-013-0847-8](https://doi.org/10.1007/s10584-013-0847-8)
- Feng S, Gao D, Liao F et al (2016) The health effects of ambient PM_{2.5} and potential mechanisms. *Ecotoxicol Environ Saf* 128:67–74. doi:[10.1016/j.ecoenv.2016.01.030](https://doi.org/10.1016/j.ecoenv.2016.01.030)
- Fleisch AF, Gold DR, Rifas-Shiman SL et al (2014) Air pollution exposure and abnormal glucose tolerance during pregnancy: the project viva cohort. *Environ Health Perspect* 122:378–383. doi:[10.1289/ehp.1307065](https://doi.org/10.1289/ehp.1307065)
- Flores-Pajot M-C, Ofner M, Do MT et al (2016) Childhood autism spectrum disorders and exposure to nitrogen dioxide, and particulate matter air pollution: a review and meta-analysis. *Environ Res* 151:763–776. doi:[10.1016/j.envres.2016.07.030](https://doi.org/10.1016/j.envres.2016.07.030)
- Forouzanfar MH, Alexander L, Anderson HR et al (2015) Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990–2013: a systematic analysis for the global burden of disease study 2013. *Lancet* 386:2287–2323. doi:[10.1016/S0140-6736\(15\)00128-2](https://doi.org/10.1016/S0140-6736(15)00128-2)
- Fuks K, Moebus S, Hertel S et al (2011) Long-term urban particulate air pollution, traffic noise, and arterial blood pressure. *Environ Health Perspect* 119:1706–1711. doi:[10.1289/ehp.1103564](https://doi.org/10.1289/ehp.1103564)
- Gaita SM, Boman J, Gatari MJ et al (2014) Source apportionment and seasonal variation of PM_{2.5} in a sub-Saharan African city: Nairobi, Kenya. *Atmos Chem Phys* 14:9977–9991. doi:[10.5194/acp-14-9977-2014](https://doi.org/10.5194/acp-14-9977-2014)
- Galindo N, Varea M, Gil-Moltó J et al (2011) The influence of meteorology on particulate matter concentrations at an urban mediterranean location. *Water Air Soil Pollut* 215:365–372. doi:[10.1007/s11270-010-0484-z](https://doi.org/10.1007/s11270-010-0484-z)

- GB 3095 (2012) Ambient air quality standards in China. TransportPolicy.net. http://transportpolicy.net/index.php?title=China:_Air_Quality_Standards. Accessed 11 Jan 2017
- Gehrig R, Buchmann B (2003) Characterising seasonal variations and spatial distribution of ambient PM₁₀ and PM_{2.5} concentrations based on long-term Swiss monitoring data. *Atmos Environ* 37:2571–2580. doi:[10.1016/S1352-2310\(03\)00221-8](https://doi.org/10.1016/S1352-2310(03)00221-8)
- Ghio AJ, Carraway MS, Madden MC (2012) Composition of air pollution particles and oxidative stress in cells, tissues, and living systems. *J Toxicol Environ Health B* 15:1–21. doi:[10.1080/10937404.2012.632359](https://doi.org/10.1080/10937404.2012.632359)
- Gnauk T, Müller K, Brüggemann E et al (2011) A study to discriminate local, urban and regional source contributions to the particulate matter concentrations in the city of Dresden, Germany. *J Atmos Chem* 68:199–231. doi:[10.1007/s10874-012-9216-7](https://doi.org/10.1007/s10874-012-9216-7)
- Greene NA, Morris VR (2006) Assessment of public health risks associated with atmospheric exposure to PM_{2.5} in Washington, DC, USA. *Int J Environ Res Public Health* 3:86–97. doi:[10.3390/ijerph2006030010](https://doi.org/10.3390/ijerph2006030010)
- Grinshpun SA, Yermakov M, Reponen T et al (2014) Traffic particles in ambient air of a major US urban area: has anything changed over a decade? *Aerosol Air Qual Res* 14:1344–1351. doi:[10.4209/aaqr.2013.11.0334](https://doi.org/10.4209/aaqr.2013.11.0334)
- Gugamsetty B, Wei H, Liu CN et al (2012) Source characterization and apportionment of PM₁₀, PM_{2.5} and PM_{0.1} by using positive matrix factorization. *Aerosol Air Qual Res* 12:476–491. doi:[10.4209/aaqr.2012.04.0084](https://doi.org/10.4209/aaqr.2012.04.0084)
- Guttikunda SK, Lodoysamba S, Bulgansaikhan B, Dashdondog B (2013) Particulate pollution in Ulaanbaatar, Mongolia. *Air Qual Atmos Health* 6:589–601. doi:[10.1007/s11869-013-0198-7](https://doi.org/10.1007/s11869-013-0198-7)
- Hallquist M, Wenger JC, Baltensperger U et al (2009) The formation, properties and impact of secondary organic aerosol: current and emerging issues. *Atmos Chem Phys* 9:5155–5236. doi:[10.5194/acp-9-5155-2009](https://doi.org/10.5194/acp-9-5155-2009)
- Hamra GB, Guha N, Cohen A et al (2014) Outdoor particulate matter exposure and lung cancer: a systematic review and meta-analysis. *Environ Health Perspect* 122:906–911. doi:[10.1289/ehp.1408092](https://doi.org/10.1289/ehp.1408092)
- Han Y-J, Kim S-R, Jung J-H (2011) Long-term measurements of atmospheric PM_{2.5} and its chemical composition in rural Korea. *J Atmos Chem* 68:281–298. doi:[10.1007/s10874-012-9225-6](https://doi.org/10.1007/s10874-012-9225-6)
- Harrison RM, Smith DJ, Kibble AJ (2004) What is responsible for the carcinogenicity of PM_{2.5}? *Occup Environ Med* 61:799–805. doi:[10.1136/oem.2003.010504](https://doi.org/10.1136/oem.2003.010504)
- Hazenkamp-Von Arx ME, Gotschi T, Ackermann-Liebrich U et al (2004) PM_{2.5} and NO₂ assessment in 21 European study centres of ECRHS II: annual means and seasonal differences. *Atmos Environ* 38:1943–1953. doi:[10.1016/j.atmosenv.2004.01.016](https://doi.org/10.1016/j.atmosenv.2004.01.016)
- Hennig F, Fuks K, Moebus S et al (2014) Association between source-specific particulate matter air pollution and hs-CRP: local traffic and industrial emissions. *Environ Health Perspect* 122:703–710. doi:[10.1289/ehp.1307081](https://doi.org/10.1289/ehp.1307081)
- Ho KF, Lee SC, Chan CK et al (2003) Characterization of chemical species in PM_{2.5} and PM₁₀ aerosols in Hong Kong. *Atmos Environ* 37:31–39. doi:[10.1016/S1352-2310\(02\)00804-X](https://doi.org/10.1016/S1352-2310(02)00804-X)
- Hong W, Jia H, Ma W et al (2016) Distribution, fate, inhalation exposure and lung cancer risk of atmospheric polycyclic aromatic hydrocarbons in some Asian countries. *Environ Sci Technol*. acs.est.6b01090. doi: [10.1021/acs.est.6b01090](https://doi.org/10.1021/acs.est.6b01090)
- Hu J, Wang Y, Ying Q, Zhang H (2014) Spatial and temporal variability of PM_{2.5} and PM₁₀ over the North China plain and the Yangtze River Delta, China. *Atmos Environ* 95:598–609. doi:[10.1016/j.atmosenv.2014.07.019](https://doi.org/10.1016/j.atmosenv.2014.07.019)
- Huang X, Liu Z, Zhang J et al (2016) Seasonal variation and secondary formation of size-segregated aerosol water-soluble inorganic ions during pollution episodes in Beijing. *Atmos Res* 168:70–79. doi:[10.1016/j.atmosres.2015.08.021](https://doi.org/10.1016/j.atmosres.2015.08.021)
- Hussain MM, Aburizaiza OS, Khwaja HA et al (2014) The state of ambient air quality of Jeddah, Saudi Arabia. Presented at the 2014 American Geophysical Union (AGU) meeting, San Francisco, CA

- Hussein T, Alghamdi MA, Khoder M et al (2014) Particulate matter and number concentrations of particles larger than 0.25 μm in the urban atmosphere of Jeddah, Saudi Arabia. *Aerosol Air Qual Res* 14:1383–1391. doi:[10.4209/aaqr.2014.02.0027](https://doi.org/10.4209/aaqr.2014.02.0027)
- Hyder A, Lee HJ, Ebisu K et al (2014) PM_{2.5} exposure and birth outcomes. *Epidemiology* 25:58–67. doi:[10.1097/EDE.0000000000000027](https://doi.org/10.1097/EDE.0000000000000027)
- Jiménez E, Linares C, Rodríguez LF et al (2009) Short-term impact of particulate matter (PM_{2.5}) on daily mortality among the over-75 age group in Madrid (Spain). *Sci Total Environ* 407:5486–5492. doi:[10.1016/j.scitotenv.2009.06.038](https://doi.org/10.1016/j.scitotenv.2009.06.038)
- Joseph AE, Unnikrishnan S, Kumar R (2012) Chemical characterization and mass closure of fine aerosol for different land use patterns in Mumbai city. *Aerosol Air Qual Res* 12:61–72. doi:[10.4209/aaqr.2011.04.0049](https://doi.org/10.4209/aaqr.2011.04.0049)
- Jugder D, Shinoda M, Sugimoto N et al (2011) Spatial and temporal variations of dust concentrations in the Gobi Desert of Mongolia. *Global Planet Change* 78:14–22. doi:[10.1016/j.gloplacha.2011.05.003](https://doi.org/10.1016/j.gloplacha.2011.05.003)
- Kassomenos P, Vardoulakis S, Chaloulakou A et al (2012) Levels, sources and seasonality of coarse particles (PM 10-PM 2.5) in three European capitals – implications for particulate pollution control. *Atmos Environ* 54:337–347. doi:[10.1016/j.atmosenv.2012.02.051](https://doi.org/10.1016/j.atmosenv.2012.02.051)
- Kendall M, Pala K, Ucakli S, Gucer S (2011) Airborne particulate matter (PM_{2.5} and PM₁₀) and associated metals in urban Turkey. *Air Qual Atmos Health* 4:235–242. doi:[10.1007/s11869-010-0129-9](https://doi.org/10.1007/s11869-010-0129-9)
- Khan MF, Shirasuna Y, Hirano K, Masunaga S (2010) Characterization of PM_{2.5}, PM_{2.5-10} and PM_{> 10} in ambient air, Yokohama, Japan. *Atmos Res* 96:159–172. doi:[10.1016/j.atmosres.2009.12.009](https://doi.org/10.1016/j.atmosres.2009.12.009)
- Khwaja HA, Fatmi Z, Malashock D et al (2012) Effect of air pollution on daily morbidity in Karachi, Pakistan. *J Local Glob Heal Sci* 3. doi: [10.5339/jlghs.2012.3](https://doi.org/10.5339/jlghs.2012.3)
- Khwaja HA, Kelly L, Fatmi Z et al (2015) Spatial and temporal variability in chemical composition of ambient fine particulate matter in the megacity of Karachi, Pakistan. Presented at the American Association of Aerosol Research (AAAR) 34th annual conference, Minneapolis, MN
- Kim KN, Lim YH, Bae HJ et al (2016) Long-term fine particulate matter exposure and major depressive disorder in a community-based urban cohort. *Environ Health Perspect* 124:1547–1553. doi:[10.1289/EHP192](https://doi.org/10.1289/EHP192)
- Kim NK, Kim YP, Kang CH (2011) Long-term trend of aerosol composition and direct radiative forcing due to aerosols over Gosan: TSP, PM₁₀, and PM_{2.5} data between 1992 and 2008. *Atmos Environ* 45:6107–6115. doi:[10.1016/j.atmosenv.2011.08.051](https://doi.org/10.1016/j.atmosenv.2011.08.051)
- Kloog I, Ridgway B, Koutrakis P et al (2013) Long- and short-term exposure to PM_{2.5} and mortality: using novel exposure models. *Epidemiology* 24:555–561. doi:[10.1097/EDE.0b013e318294beaa](https://doi.org/10.1097/EDE.0b013e318294beaa)
- Kopp RE, Mauzerall DL (2010) Assessing the climatic benefits of black carbon mitigation. *Proc Natl Acad Sci U S A* 107:11703–11708. doi:[10.1073/pnas.0909605107](https://doi.org/10.1073/pnas.0909605107)
- Kothai P, Saradhi IV, Pandit GG et al (2011) Chemical characterization and source identification of particulate matter at an urban site of Navi Mumbai, India. *Aerosol Air Qual Res* 11:560–569. doi:[10.4209/aaqr.2011.02.0017](https://doi.org/10.4209/aaqr.2011.02.0017)
- Krall JR, Chang HH, Sarnat SE et al (2015) Current methods and challenges for epidemiological studies of the associations between chemical constituents of particulate matter and health. *Curr Environ Health Rep* 2:388–398. doi:[10.1007/s40572-015-0071-y](https://doi.org/10.1007/s40572-015-0071-y)
- Kulshrestha A, Satsangi PG, Masih J, Taneja A (2009) Metal concentration of PM_{2.5} and PM₁₀ particles and seasonal variations in urban and rural environment of Agra, India. *Sci Total Environ* 407:6196–6204. doi:[10.1016/j.scitotenv.2009.08.050](https://doi.org/10.1016/j.scitotenv.2009.08.050)
- Kuvarega AT, Taru P (2008) Ambient dust speciation and metal content variation in TSP, PM₁₀ and PM_{2.5} in urban atmospheric air of Harare (Zimbabwe). *Environ Monit Assess* 144:1–14. doi:[10.1007/s10661-008-0436-x](https://doi.org/10.1007/s10661-008-0436-x)

- Laing S, Wang G, Briazova T et al (2010) Airborne particulate matter selectively activates endoplasmic reticulum stress response in the lung and liver tissues. *Am J Physiol Cell Physiol* 299:C736–C749. doi:[10.1152/ajpcell.00529.2009](https://doi.org/10.1152/ajpcell.00529.2009)
- Lall R, Ito K, Thurston GD (2011) Distributed lag analyses of daily hospital admissions and source-apportioned fine particle air pollution. *Environ Health Perspect* 119:455–460. doi:[10.1289/ehp.1002638](https://doi.org/10.1289/ehp.1002638)
- Lall R, Thurston GD (2006) Identifying and quantifying transported vs. local sources of New York City PM_{2.5} fine particulate matter air pollution. *Atmos Environ* 40:333–346. doi:[10.1016/j.atmosenv.2006.04.068](https://doi.org/10.1016/j.atmosenv.2006.04.068)
- Lazaridis M, Dzumbova L, Kopanakis I et al (2008) PM₁₀ and PM_{2.5} levels in the eastern Mediterranean (Akrotiri research station, Crete, Greece). *Water Air Soil Pollut* 189:85–101. doi:[10.1007/s11270-007-9558-y](https://doi.org/10.1007/s11270-007-9558-y)
- Lee HJ, Gent JF, Leaderer BP, Koutrakis P (2011) Spatial and temporal variability of fine particle composition and source types in five cities of Connecticut and Massachusetts. *Sci Total Environ* 409:2133–2142. doi:[10.1016/j.scitotenv.2011.02.025](https://doi.org/10.1016/j.scitotenv.2011.02.025)
- Lemos AT, Coronas MV, Rocha JAV, Vargas VMF (2012) Mutagenicity of particulate matter fractions in areas under the impact of urban and industrial activities. *Chemosphere* 89:1126–1134. doi:[10.1016/j.chemosphere.2012.05.100](https://doi.org/10.1016/j.chemosphere.2012.05.100)
- Li P, Xin J, Wang Y et al (2013) Time-series analysis of mortality effects from airborne particulate matter size fractions in Beijing. *Atmos Environ* 81:253–262. doi:[10.1016/j.atmosenv.2013.09.004](https://doi.org/10.1016/j.atmosenv.2013.09.004)
- Li PH, Han B, Huo J et al (2012) Characterization, meteorological influences and source identification of carbonaceous aerosols during the autumn-winter period in Tianjin, China. *Aerosol Air Qual Res* 12:283–294. doi:[10.4209/aaqr.2011.09.0140](https://doi.org/10.4209/aaqr.2011.09.0140)
- López ML, Ceppi S, Palancar GG et al (2011) Elemental concentration and source identification of PM₁₀ and PM_{2.5} by SR-XRF in Córdoba City, Argentina. *Atmos Environ* 45:5450–5457. doi:[10.1016/j.atmosenv.2011.07.003](https://doi.org/10.1016/j.atmosenv.2011.07.003)
- Lu MC, Wang P, Cheng TJ et al (2017) Association of temporal distribution of fine particulate matter with glucose homeostasis during pregnancy in women of Chiayi City, Taiwan. *Environ Res* 152:81–87. doi:[10.1016/j.envres.2016.09.023](https://doi.org/10.1016/j.envres.2016.09.023)
- Luo C, Zhu X, Yao C et al (2015) Short-term exposure to particulate air pollution and risk of myocardial infarction: a systematic review and meta-analysis. *Environ Sci Pollut Res* 22:14651–14662. doi:[10.1007/s11356-015-5188-x](https://doi.org/10.1007/s11356-015-5188-x)
- Ma Y, Chen R, Pan G et al (2011) Fine particulate air pollution and daily mortality in Shenyang, China. *Sci Total Environ* 409:2473–2477. doi:[10.1016/j.scitotenv.2011.03.017](https://doi.org/10.1016/j.scitotenv.2011.03.017)
- Maas R, Grennfelt P (2016) Towards cleaner air. Scientific Assessment Report 2016
- Mahowald N, Ward DS, Kloster S et al (2011) Aerosol impacts on climate and biogeochemistry. *Annu Rev Env Resour* 36:45–74. doi:[10.1146/annurev-environ-042009-094507](https://doi.org/10.1146/annurev-environ-042009-094507)
- Mancilla Y, Herckes P, Fraser MP, Mendoza A (2015) Secondary organic aerosol contributions to PM_{2.5} in Monterrey, Mexico: temporal and seasonal variation. *Atmos Res* 153:348–359. doi:[10.1016/j.atmosres.2014.09.009](https://doi.org/10.1016/j.atmosres.2014.09.009)
- Mansha M, Ghauri B, Rahman S, Amman A (2012) Characterization and source apportionment of ambient air particulate matter (PM_{2.5}) in Karachi. *Sci Total Environ* 425:176–183. doi:[10.1016/j.scitotenv.2011.10.056](https://doi.org/10.1016/j.scitotenv.2011.10.056)
- Mariani RL, de Mello WZ (2007) PM_{2.5-10}, PM_{2.5} and associated water-soluble inorganic species at a coastal urban site in the metropolitan region of Rio de Janeiro. *Atmos Environ* 41:2887–2892. doi:[10.1016/j.atmosenv.2006.12.009](https://doi.org/10.1016/j.atmosenv.2006.12.009)
- Mazzoli-Rocha F, Fernandes S, Einicker-Lamas M, Zin WA (2010) Roles of oxidative stress in signaling and inflammation induced by particulate matter. *Cell Biol Toxicol* 26:481–498. doi:[10.1007/s10565-010-9158-2](https://doi.org/10.1007/s10565-010-9158-2)
- Mehta AJ, Zanobetti A, Bind M-AC et al (2016) Long-term exposure to ambient fine particulate matter and renal function in older men: the VA normative aging study. *Environ Health Perspect* 124:1353–1360. doi:[10.1289/ehp.1510269](https://doi.org/10.1289/ehp.1510269)

- Miller KA, Siscovick DS, Sheppard L et al (2007) Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 356:447–458. doi:[10.1056/NEJMoa054409](https://doi.org/10.1056/NEJMoa054409)
- Mills NL, Donaldson K, Hadoke PW et al (2009) Adverse cardiovascular effects of air pollution. *Nat Clin Pract Cardiovasc Med* 6:36–44. doi:[10.1038/npcardio1399](https://doi.org/10.1038/npcardio1399)
- Minguillón MC, Campos AA, Cárdenas B et al (2014) Mass concentration, composition and sources of fine and coarse particulate matter in Tijuana, Mexico, during Cal-Mex campaign. *Atmos Environ* 88:320–329. doi:[10.1016/j.atmosenv.2013.09.032](https://doi.org/10.1016/j.atmosenv.2013.09.032)
- Miyata R, van Eeden SF (2011) The innate and adaptive immune response induced by alveolar macrophages exposed to ambient particulate matter. *Toxicol Appl Pharmacol* 257:209–226. doi:[10.1016/j.taap.2011.09.007](https://doi.org/10.1016/j.taap.2011.09.007)
- Mkoma SL, Chi X, Maenhaut W (2010) Characteristics of carbonaceous aerosols in ambient PM10 and PM2.5 particles in Dar es Salaam, Tanzania. *Sci Total Environ* 408:1308–1314. doi:[10.1016/j.scitotenv.2009.10.054](https://doi.org/10.1016/j.scitotenv.2009.10.054)
- Mohanraj R, Solaraj G, Dhanakumar S (2011) Fine particulate phase PAHs in ambient atmosphere of Chennai metropolitan city, India. *Environ Sci Pollut Res* 18:764–771. doi:[10.1007/s11356-010-0423-y](https://doi.org/10.1007/s11356-010-0423-y)
- Mooibroek D, Schaap M, Weijers EP, Hoogerbrugge R (2011) Source apportionment and spatial variability of PM2.5 using measurements at five sites in the Netherlands. *Atmos Environ* 45:4180–4191. doi:[10.1016/j.atmosenv.2011.05.017](https://doi.org/10.1016/j.atmosenv.2011.05.017)
- Morakinyo OM, Mokgobu MI, Mukhola MS, Hunter RP (2016) Health outcomes of exposure to biological and chemical components of inhalable and respirable particulate matter. *Int J Environ Res Public Health* 13:1–22
- Murillo JH, Roman SR, Rojas Marin JF et al (2013) Chemical characterization and source apportionment of PM10 and PM2.5 in the metropolitan area of Costa Rica, Central America. *Atmos Pollut Res* 4:181–190. doi:[10.5094/APR.2013.018](https://doi.org/10.5094/APR.2013.018)
- Negrál L, Moreno-Grau S, Moreno J et al (2008) Natural and anthropogenic contributions to PM10 and PM2.5 in an urban area in the western Mediterranean coast. *Water Air Soil Pollut* 192:227–238. doi:[10.1007/s11270-008-9650-y](https://doi.org/10.1007/s11270-008-9650-y)
- NEPM (2002) National Environment Protection Measure for Ambient Air Quality. Department of the Environment and Energy Australian Government. <https://www.environment.gov.au/protection/air-quality/air-quality-standards>. Accessed 21 Jan 2017
- NOM (2005) Norma Oficial Mexicana, Mexico's environmental and natural resources ministry, Secretaría de Medio Ambiente y Recursos Naturales (SEMARNAT). <http://www.salud.gob.mx/unidades/cdi/nom/025ssa193.html>. Accessed 18 January 2017
- Nyanganyura D, Maenhaut W, Mathuthu M et al (2007) The chemical composition of tropospheric aerosols and their contributing sources to a continental background site in northern Zimbabwe from 1994 to 2000. *Atmos Environ* 41:2644–2659. doi:[10.1016/j.atmosenv.2006.11.015](https://doi.org/10.1016/j.atmosenv.2006.11.015)
- Olson DA, Turlington J, Duval RM et al (2008) Indoor and outdoor concentrations of organic and inorganic molecular markers: source apportionment of PM2.5 using low-volume samples. *Atmos Environ* 42:1742–1751. doi:[10.1016/j.atmosenv.2007.11.035](https://doi.org/10.1016/j.atmosenv.2007.11.035)
- Onat B, Sahin UA, Akyuz T (2013) Elemental characterization of PM2.5 and PM1 in dense traffic area in Istanbul, Turkey. *Atmos Pollut Res* 4:101–105. doi:[10.5094/APR.2013.010](https://doi.org/10.5094/APR.2013.010)
- Øvreik J, Låg M, Holme JA et al (2009) Cytokine and chemokine expression patterns in lung epithelial cells exposed to components characteristic of particulate air pollution. *Toxicology* 259:46–53. doi:[10.1016/j.tox.2009.01.028](https://doi.org/10.1016/j.tox.2009.01.028)
- Øvreik J, Refsnes M, Låg M et al (2015) Activation of proinflammatory responses in cells of the airway mucosa by particulate matter: oxidant- and non-oxidant-mediated triggering mechanisms. *Biomol Ther* 5:1399–1440. doi:[10.3390/biom5031399](https://doi.org/10.3390/biom5031399)
- Owoade KO, Hopke PK, Olise FS et al (2015) Chemical compositions and source identification of particulate matter (PM2.5 and PM2.5-10) from a scrap iron and steel smelting industry along the Ife–Ibadan highway, Nigeria. *Atmos Pollut Res* 6:107–119. doi:[10.5094/APR.2015.013](https://doi.org/10.5094/APR.2015.013)

- Pancras JP, Landis MS, Norris GA et al (2013) Source apportionment of ambient fine particulate matter in Dearborn, Michigan, using hourly resolved PM chemical composition data. *Sci Total Environ* 448:2–13. doi:[10.1016/j.scitotenv.2012.11.083](https://doi.org/10.1016/j.scitotenv.2012.11.083)
- Pandey P, Khan AH, Verma AK et al (2012) Seasonal trends of PM 2.5 and PM 10 in ambient air and their correlation in ambient air of Lucknow City, India. *Bull Environ Contam Toxicol* 88:265–270. doi:[10.1007/s00128-011-0466-x](https://doi.org/10.1007/s00128-011-0466-x)
- Pascal M, Falq G, Wagner V et al (2014) Short-term impacts of particulate matter (PM10, PM10-2.5, PM2.5) on mortality in nine French cities. *Atmos Environ* 95:175–184. doi:[10.1016/j.atmosenv.2014.06.030](https://doi.org/10.1016/j.atmosenv.2014.06.030)
- Pateraki S, Asimakopoulou DN, Flocas HA et al (2012) The role of meteorology on different sized aerosol fractions (PM 10, PM 2.5, PM 2.5-10). *Sci Total Environ* 419:124–135. doi:[10.1016/j.scitotenv.2011.12.064](https://doi.org/10.1016/j.scitotenv.2011.12.064)
- PCD (2010) Pollution Control Department. Ambient Air Standards. Ministry of Natural Resources and Environment Thailand. http://www.pcd.go.th/info_serv/en_reg_std_airsnd01.html. Accessed 22 Jan 2017
- Perera FP, Li Z, Whyatt R et al (2009) Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. *Pediatrics* 124:e195–e202. doi:[10.1542/peds.2008-3506](https://doi.org/10.1542/peds.2008-3506)
- Perera FP, Tang D, Wang S et al (2012) Prenatal polycyclic aromatic hydrocarbon (PAH) exposure and child behavior at age 6–7 years. *Environ Health Perspect* 120:921–926. doi:[10.1289/ehp.1104315](https://doi.org/10.1289/ehp.1104315)
- Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287:1132–1141. doi:[10.1001/jama.287.9.1132](https://doi.org/10.1001/jama.287.9.1132)
- Pope CA, Dockery DW (2013) Air pollution and life expectancy in China and beyond. *Proc Natl Acad Sci U S A* 110:12861–12862. doi:[10.1073/pnas.1310925110](https://doi.org/10.1073/pnas.1310925110)
- Qin Y, Kim E, Hopke PK (2006) The concentrations and sources of PM2.5 in metropolitan New York City. *Atmos Environ* 40:312–332. doi:[10.1016/j.atmosenv.2006.02.025](https://doi.org/10.1016/j.atmosenv.2006.02.025)
- Querol X, Alastuey A, Rodriguez S et al (2001) PM10 and PM2.5 source apportionment in the Barcelona metropolitan area, Catalonia, Spain. *Atmos Environ* 35:6407–6419. doi:[10.1016/S1352-2310\(01\)00361-2](https://doi.org/10.1016/S1352-2310(01)00361-2)
- Raaschou-Nielsen O, Andersen ZJ, Beelen R et al (2013) Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European study of cohorts for air pollution effects (ESCAPE). *Lancet Oncol* 14:813–822. doi:[10.1016/S1470-2045\(13\)70279-1](https://doi.org/10.1016/S1470-2045(13)70279-1)
- Rahman SA, Hamzah MS, Wood AK et al (2011) Sources apportionment of fine and coarse aerosol in Klang Valley, Kuala Lumpur using positive matrix factorization. *Atmos Pollut Res* 2:197–206. doi:[10.5094/APR.2011.025](https://doi.org/10.5094/APR.2011.025)
- Raja S, Biswas KF, Husain L, Hopke PK (2010) Source apportionment of the atmospheric aerosol in Lahore, Pakistan. *Water Air Soil Pollut* 208:43–57. doi:[10.1007/s11270-009-0148-z](https://doi.org/10.1007/s11270-009-0148-z)
- Rajput P, Sarin M, Kundu SS (2013) Atmospheric particulate matter (PM2.5), EC, OC, WSOC and PAHs from NE-Himalaya: abundances and chemical characteristics. *Atmos Pollut Res* 4:214–221. doi:[10.5094/APR.2013.022](https://doi.org/10.5094/APR.2013.022)
- Rajšić S, Mijić Z, Tasić M et al (2008) Evaluation of the levels and sources of trace elements in urban particulate matter. *Environ Chem Lett* 6:95–100. doi:[10.1007/s10311-007-0115-0](https://doi.org/10.1007/s10311-007-0115-0)
- Rappazzo KM, Daniels JL, Messer LC et al (2015) Exposure to elemental carbon, organic carbon, nitrate, and sulfate fractions of fine particulate matter and risk of preterm birth in New Jersey, Ohio, and Pennsylvania (2000–2005). *Environ Health Perspect* 123:1059–1065. doi:[10.1289/ehp.1408953](https://doi.org/10.1289/ehp.1408953)
- Rashki A, dew Rautenbach CJ, Eriksson PG et al (2013) Temporal changes of particulate concentration in the ambient air over the city of Zahedan, Iran. *Air Qual Atmos Health* 6:123–135. doi:[10.1007/s11869-011-0152-5](https://doi.org/10.1007/s11869-011-0152-5)
- Rattigan OV, Civerolo K, Doraiswamy P et al (2013) Long term black carbon measurements at two urban locations in New York. *Aerosol Air Qual Res* 13:1181–1196. doi:[10.4209/aaqr.2013.02.0060](https://doi.org/10.4209/aaqr.2013.02.0060)

- Raysoni AU, Sarnat JA, Sarnat SE et al (2011) Binational school-based monitoring of traffic-related air pollutants in El Paso, Texas (USA) and Ciudad Juárez, Chihuahua (Mexico). *Environ Pollut* 159:2476–2486. doi:[10.1016/j.envpol.2011.06.024](https://doi.org/10.1016/j.envpol.2011.06.024)
- Riccio A, Chianese E, Agrillo G et al (2014) Source apportion of atmospheric particulate matter: a joint Eulerian/Lagrangian approach. *Environ Sci Pollut Res* 21:13160–13168. doi:[10.1007/s11356-013-2367-5](https://doi.org/10.1007/s11356-013-2367-5)
- Rogula-Kozłowska W, Błaszczak B, Szopa S et al (2013) PM_{2.5} in the central part of upper Silesia, Poland: concentrations, elemental composition, and mobility of components. *Environ Monit Assess* 185:581–601. doi:[10.1007/s10661-012-2577-1](https://doi.org/10.1007/s10661-012-2577-1)
- Rohr AC, Wyzga RE (2012) Attributing health effects to individual particulate matter constituents. *Atmos Environ* 62:130–152. doi:[10.1016/j.atmosenv.2012.07.036](https://doi.org/10.1016/j.atmosenv.2012.07.036)
- Saldarriaga-Noreña H, Hernández-Mena L, Ramírez-Muñoz M et al (2009) Characterization of trace metals of risk to human health in airborne particulate matter (PM_{2.5}) at two sites in Guadalajara, Mexico. *J Environ Monit* 11:887–894. doi:[10.1039/b815747b](https://doi.org/10.1039/b815747b)
- Salvador P, Artíñano B, Querol X et al (2007) Characterisation of local and external contributions of atmospheric particulate matter at a background coastal site. *Atmos Environ* 41:1–17. doi:[10.1016/j.atmosenv.2006.08.007](https://doi.org/10.1016/j.atmosenv.2006.08.007)
- Salvi S, Holgate S (1999) Mechanisms of particulate matter toxicity. *Clin Exp Allergy* 29:1187–1194. doi:[10.1046/j.1365-2222.1999.00576.x](https://doi.org/10.1046/j.1365-2222.1999.00576.x)
- Samet JM, Dominici F, Currier FC et al (2000) Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N Engl J Med* 343:1742–1749. doi:[10.1056/NEJM200012143432401](https://doi.org/10.1056/NEJM200012143432401)
- Shaka' H, Saliba NA (2004) Concentration measurements and chemical composition of PM_{10-2.5} and PM_{2.5} at a coastal site in Beirut, Lebanon. *Atmos Environ* 38:523–531. doi:[10.1016/j.atmosenv.2003.10.009](https://doi.org/10.1016/j.atmosenv.2003.10.009)
- Sharma M, Maloo S (2005) Assessment of ambient air PM₁₀ and PM_{2.5} and characterization of PM₁₀ in the city of Kanpur, India. *Atmos Environ* 39:6015–6026. doi:[10.1016/j.atmosenv.2005.04.041](https://doi.org/10.1016/j.atmosenv.2005.04.041)
- Sillanpää M, Hillamo R, Saarikoski S et al (2006) Chemical composition and mass closure of particulate matter at six urban sites in Europe. *Atmos Environ* 40:212–223. doi:[10.1016/j.atmosenv.2006.01.063](https://doi.org/10.1016/j.atmosenv.2006.01.063)
- Spindler G, Grüner A, Müller K et al (2013) Long-term size-segregated particle (PM₁₀, PM_{2.5}, PM₁) characterization study at Melpitz – influence of air mass inflow, weather conditions and season. *J Atmos Chem* 70:165–195. doi:[10.1007/s10874-013-9263-8](https://doi.org/10.1007/s10874-013-9263-8)
- Stanek LW, Sacks JD, Dutton SJ, Dubois JJB (2011) Attributing health effects to apportioned components and sources of particulate matter: an evaluation of collective results. *Atmos Environ* 45:5655–5663. doi:[10.1016/j.atmosenv.2011.07.023](https://doi.org/10.1016/j.atmosenv.2011.07.023)
- Stone E, Schauer J, Quraishi TA, Mahmood A (2010) Chemical characterization and source apportionment of fine and coarse particulate matter in Lahore, Pakistan. *Atmos Environ* 44:1062–1070. doi:[10.1016/j.atmosenv.2009.12.015](https://doi.org/10.1016/j.atmosenv.2009.12.015)
- Sudheer AK, Rengarajan R (2012) Atmospheric mineral dust and trace metals over urban environment in western India during winter. *Aerosol Air Qual Res* 12:923–933. doi:[10.4209/aaqr.2011.12.0237](https://doi.org/10.4209/aaqr.2011.12.0237)
- Sudheera K, Rengarajan R, Sheel V (2015) Secondary organic aerosol over an urban environment in a semi-arid region of western India. *Atmos Pollut Res* 6:11–20. doi:[10.5094/APR.2015.002](https://doi.org/10.5094/APR.2015.002)
- Sun X, Luo X, Zhao C et al (2015) The association between fine particulate matter exposure during pregnancy and preterm birth: a meta-analysis. *BMC Pregnancy Childbirth* 15:300. doi:[10.1186/s12884-015-0738-2](https://doi.org/10.1186/s12884-015-0738-2)
- Talbott EO, Arena VC, Rager JR et al (2015) Fine particulate matter and the risk of autism spectrum disorder. *Environ Res* 140:414–420. doi:[10.1016/j.envres.2015.04.021](https://doi.org/10.1016/j.envres.2015.04.021)
- Tian H, Banger K, Bo T, Dadhwal VK (2014) History of land use in India during 1880–2010: Large-scale land transformations reconstructed from satellite data and historical archives. *Glob Planet Change* 121:78–88. doi: [10.1016/j.gloplacha.2014.07.005](https://doi.org/10.1016/j.gloplacha.2014.07.005)

- Tian YZ, Wu JH, Shi GL et al (2013) Long-term variation of the levels, compositions and sources of size-resolved particulate matter in a megacity in China. *Sci Total Environ* 463–464:462–468. doi:[10.1016/j.scitotenv.2013.06.055](https://doi.org/10.1016/j.scitotenv.2013.06.055)
- Tiwari S, Srivastava AK, Bisht DS et al (2009) Black carbon and chemical characteristics of PM₁₀ and PM_{2.5} at an urban site of North India. *J Atmos Chem* 62:193–209. doi:[10.1007/s10874-010-9148-z](https://doi.org/10.1007/s10874-010-9148-z)
- Tiwari S, Srivastava AK, Bisht DS et al (2013) Diurnal and seasonal variations of black carbon and PM_{2.5} over New Delhi, India: influence of meteorology. *Atmos Res* 125–126:50–62. doi:[10.1016/j.atmosres.2013.01.011](https://doi.org/10.1016/j.atmosres.2013.01.011)
- Traversi D, Degan R, De Marco R et al (2009) Mutagenic properties of PM_{2.5} urban pollution in the Northern Italy: the nitro-compounds contribution. *Environ Int* 35:905–910. doi:[10.1016/j.envint.2009.03.010](https://doi.org/10.1016/j.envint.2009.03.010)
- Upadhyay N, Clements A, Fraser M, Herckes P (2011) Chemical speciation of PM_{2.5} and PM₁₀ in south Phoenix, AZ, USA. *J Air Waste Manag Assoc* 61:302–310. doi:[10.3155/1047-3289.61.3.302](https://doi.org/10.3155/1047-3289.61.3.302)
- US EPA (2012) The National Ambient Air Quality Standards for particle pollution. United States Environmental Protection Agency. www.epa.gov/airquality/particulatepollution/2012/decfsstandards.pdf. Accessed 10 Jan 2017
- US EPA (2014) National trends in particulate matter levels. United States Environmental Protection Agency. <http://www.epa.gov/airtrends/pm.html>. Accessed 22 Jan 2017
- US EPA (2015) Annual summary and daily summary air quality data. United States Environmental Protection Agency. http://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html. Accessed 21 Jan 2017
- Valavanidis A, Fiotakis K, Vlachogianni T (2008) Airborne particulate matter and human health: toxicological assessment and importance of size and composition of particles for oxidative damage and carcinogenic mechanisms. *J Environ Sci Health C* 26:339–362. doi:[10.1080/10590500802494538](https://doi.org/10.1080/10590500802494538)
- Vellingiri K, Kim K-H, Ma C-J et al (2015) Ambient particulate matter in a central urban area of Seoul, Korea. *Chemosphere* 119:812–819. doi:[10.1016/j.chemosphere.2014.08.049](https://doi.org/10.1016/j.chemosphere.2014.08.049)
- Vicente AB, Pallares S, Soriano A et al (2011) Toxic metals (As, Cd, Ni and Pb) and PM_{2.5} in air concentration of a model ceramic cluster. *Water Air Soil Pollut* 222:149–161. doi:[10.1007/s11270-011-0813-x](https://doi.org/10.1007/s11270-011-0813-x)
- Von Schneidmesser E, Monks PS, Allan JD et al (2015) Chemistry and the linkages between air quality and climate change. *Chem Rev* 115:3856–3897. doi:[10.1021/acs.chemrev.5b00089](https://doi.org/10.1021/acs.chemrev.5b00089)
- von Schneidmesser E, Zhou J, Stone EA et al (2010) Seasonal and spatial trends in the sources of fine particle organic carbon in Israel, Jordan, and Palestine. *Atmos Environ* 44:3669–3678. doi:[10.1016/j.atmosenv.2010.06.039](https://doi.org/10.1016/j.atmosenv.2010.06.039)
- Voutsas D, Samara C, Manoli E et al (2014) Ionic composition of PM_{2.5} at urban sites of northern Greece: secondary inorganic aerosol formation. *Environ Sci Pollut Res* 21:4995–5006. doi:[10.1007/s11356-013-2445-8](https://doi.org/10.1007/s11356-013-2445-8)
- Vu VT, Lee BK, Kim JT et al (2011) Assessment of carcinogenic risk due to inhalation of polycyclic aromatic hydrocarbons in PM₁₀ from an industrial city: a Korean case-study. *J Hazard Mater* 189:349–356. doi:[10.1016/j.jhazmat.2011.02.043](https://doi.org/10.1016/j.jhazmat.2011.02.043)
- Wang Y, Eliot MN, Koutrakis P et al (2014) Ambient air pollution and depressive symptoms in older adults: results from the MOBILIZE Boston study. *Environ Health Perspect* 122:553–558. doi:[10.1289/ehp.1205909](https://doi.org/10.1289/ehp.1205909)
- Wang Y, Hopke PK (2014) Is Alaska truly the great escape from air Pollution? – Long term source apportionment of fine particulate matter in Fairbank, Alaska. *Aerosol Air Qual Res* 14:1875–1882. doi:[10.4209/aaqr.2014.03.0047](https://doi.org/10.4209/aaqr.2014.03.0047)
- WHO (2005) WHO air quality guidelines global update published by World Health Organization on the internet. http://www.euro.who.int/__data/assets/pdf_file/0005/78638/E90038.pdf?ua=1. Accessed 31 Jan 2016

- WHO (2014) Ambient (outdoor) air pollution in cities database 2014. World Health Organization. http://www.who.int/phe/health_topics/outdoorair/databases/cities/en/. Accessed 20 Jan 2017
- Wichmann G, Franck U, Herbarth O et al (2009) Different immunomodulatory effects associated with sub-micrometer particles in ambient air from rural, urban and industrial areas. *Toxicology* 257:127–136. doi:[10.1016/j.tox.2008.12.024](https://doi.org/10.1016/j.tox.2008.12.024)
- Wilhelm M, Ghosh JK, Su J et al (2011) Traffic-related air toxics and preterm birth: a population-based case-control study in Los Angeles county, California. *Environ Health* 10:1–12
- Wilhelm M, Ghosh JK, Su J et al (2012) Traffic-related air toxics and term low birth weight in Los Angeles County, California. *Environ Health Perspect* 120:132–138. doi:[10.1289/ehp.1103408](https://doi.org/10.1289/ehp.1103408)
- World Bank (2015) The World Bank Group. PM2.5 pollution, mean annual exposure (micrograms per cubic meter). <http://data.worldbank.org/indicator/EN.ATM.PM25.MC.M3>. Accessed 20 Nov 2016
- Wu L, Jin L, Shi T et al (2017) Association between ambient particulate matter exposure and semen quality in Wuhan, China. *Environ Int* 98:219–228. doi:[10.1016/j.envint.2016.11.013](https://doi.org/10.1016/j.envint.2016.11.013)
- Yao L, Yang L, Chen J et al (2016) Science of the total environment characteristics of carbonaceous aerosols: impact of biomass burning and secondary formation in summertime in a rural area of the North China plain. *Sci Total Environ* 557–558:520–530. doi:[10.1016/j.scitotenv.2016.03.111](https://doi.org/10.1016/j.scitotenv.2016.03.111)
- Yatkin S, Bayram A (2008) Source apportionment of PM10 and PM2.5 using positive matrix factorization and chemical mass balance in Izmir, Turkey. *Sci Total Environ* 390:109–123. doi:[10.1016/j.scitotenv.2007.08.059](https://doi.org/10.1016/j.scitotenv.2007.08.059)
- Yorifuji T, Kashima S, Doi H (2016) Acute exposure to fine and coarse particulate matter and infant mortality in Tokyo, Japan (2002–2013). *Sci Total Environ* 551–552:66–72. doi:[10.1016/j.scitotenv.2016.01.211](https://doi.org/10.1016/j.scitotenv.2016.01.211)
- Zakey AS, Abdel-Wahab MM, Pettersson JBC et al (2008) Seasonal and spatial variation of atmospheric particulate matter in a developing megacity, the greater Cairo, Egypt. *Atmosfera* 21:171–189
- Zhang F, Cheng H-r, Wang Z-w et al (2014) Fine particles (PM2.5) at a CAWNET background site in Central China: chemical compositions, seasonal variations and regional pollution events. *Atmos Environ* 86:193–202. doi:[10.1016/j.atmosenv.2013.12.008](https://doi.org/10.1016/j.atmosenv.2013.12.008)
- Zhang R, Wang G, Guo S et al (2015) Formation of urban fine particulate matter. *Chem Rev* 115:3803–3855
- Zhou X, Gao J, Wang T et al (2009) Measurement of black carbon aerosols near two Chinese megacities and the implications for improving emission inventories. *Atmos Environ* 43:3918–3924. doi:[10.1016/j.atmosenv.2009.04.062](https://doi.org/10.1016/j.atmosenv.2009.04.062)
- Zhu X, Liu Y, Chen Y et al (2015) Maternal exposure to fine particulate matter (PM2.5) and pregnancy outcomes: a meta-analysis. *Environ Sci Pollut Res* 22:3383–3396. doi:[10.1007/s11356-014-3458-7](https://doi.org/10.1007/s11356-014-3458-7)

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