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REVIEW ARTICLE

Exposure to urban particulate matter and its association with human health risks

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Abstract

Human health and environmental risks are increasing following air pollution associated with vehicular and industrial emissions in which particulate matter is a constituent. The purpose of this review was to assess studies on the health effects and mortality induced by particles published for the last 15 years. The literature survey indicated the existence of strong positive associations between fine and ultrafine particles' exposure and cardiovascular, hypertension, obesity and type 2 diabetes mellitus, cancer health risks, and mortality. Its exposure is also associated with increased odds of hypertensive and diabetes disorders of pregnancy and premature deaths. The ever increasing hospital admission and mortality due to heart failure, diabetes, hypertension, and cancer could be due to long-term exposure to particles in different countries. Therefore, its effect should be communicated for legal and scientific actions to minimize emissions mainly from traffic sources.

Keywords Cancer · Cardiovascular · Diabetes · Exposure · Hypertension · Particulate matter

Introduction

Air pollution is becoming the major global risk factor connected with developmental activities such as industrialization, urbanization, and agricultural mechanization. Though different gaseous and particulate air pollutants are emitted by natural and anthropogenic sources, six pollutants have been identified by the US Environmental Protection Agency (EPA) as criteria air pollutants based on the way in which they are regulated, or their legal status (Esworthy and Mccarthy 2010). These are as follows: particulate matter (PM), sulfur dioxide (SO_2), carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), and lead (Pb) both common and detrimental to human health.

PM is one of the contributing pollutants directly emitted or formed as a result of atmospheric chemical interactions and transformations of gaseous emissions such as oxides of sulfur (SOx), oxides of nitrogen (NOx), and volatile organic compounds (VOCs). It is a mixture of solid and/or liquid particles in the atmosphere that remains dispersed in the air, having characteristics varying by location (Baltensperger and Prevot 2008).

The deposition and toxicity characteristics of PM are dependent on the sources. Burnett et al. (2014) reported the model of the fine particulate mass that covered the different emission sources and the global exposure range. For urban PM, the major sources are reported as vehicular emissions (Thorpe and Harrison 2008; Pant and Harrison 2013; Amato et al. 2014; Hikmat Ramdhan et al. 2018; Padoan et al. 2018; Elisabet Butarbutar et al. 2019), industrial (Alastuey et al. 2006; Hieu and Lee 2010; Stone et al. 2010; Mansha et al. 2012), and domestic biomass burning (Karagulian et al. 2015). The global contributions for these three sources are 25%, 15%, and 20%, respectively, and the other 40% is emitted by see sprays and unknown sources (Karagulian et al. 2015). Globally, the average anthropogenic PM accounts for about 10% of the total aerosol amount (Perrino 2010), and atmospheric levels of $PM_{2.5}$ is increased by the rate of 2.1%/year (Tagliabue et al. 2016).

Apart from the sources, different countries have conducted and reported studies on the health effects of urban PM. For example, there are study reports in cities of China (Chen et al. 2015, 2016; Wu et al. 2018), in Europe (Sillanpää et al. 2006; Viana et al. 2008; Putaud et al. 2010), USA (Peel et al.

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2005; Wellenius et al. 2005; Cheung et al. 2011), Australia and New Zealand (Barnett et al. 2005), Germany (Ruckerl et al. 2006), Pennsylvania countries (Sagiv et al. 2005), Brazil (Mateus et al. 2013; da Silva et al. 2015), and Pakistan (Stone et al. 2010; Javed et al. 2015; Shahid et al. 2016), among others are some of the areas where extensive study on short- and long-term effects of PM were studied. Despite considerable efforts, it is difficult to practically minimize particulate emission. For example, in China, urban air pollution remains high, with average $PM_{2.5}$ concentrations of 58 and 39 μg/m^3 in Beijing and Shanghai, respectively, which are far from 10 μg/m^3, the WHO annual mean threshold (Ji 2018). Globally, air pollution alone causes 6.5 million deaths each year of which PM took the largest share (WHO 2018).

Both epidemiological (prospective cohorts) (Babisch et al. 2014; Song et al. 2017; Tagliabue et al. 2016) and biomarker (Jia et al. 2017; Liu et al. 2014; Vineis and Husgafvel-Pursiainen 2005) studies reported existence of strong positive association between PM exposure and mortality. Conversely, a decrease in $PM_{2.5}$ is associated with an estimated increase in mean life expectancy (Pope et al. 2009).

Although traffic congestion, industrialization, and rapid urbanization is being practiced in African and Asian cities, there are limited scientific reports related to the effects of PM exposure on human health, especially on mortality rate due to cardiovascular, hypertension, diabetes, cancer, and all cause. Despite vehicles cause adverse health effects in urban areas (Elisabet Butarbutar et al. 2019; Hikmat Ramdhan et al. 2018; Kim et al. 2014), it remains neglected in Asian and African cities. For example, in Addis Ababa, Ethiopia, high traffic congestion, thereby diesel exhaust is mostly observed in the morning and at noon. This also affects national economy due to losses to medical expenditures, low productivity due to related disease, and environmental degradation costs (Landrigan 2017). Nevertheless, little actions have been done to prevent the public from PM and other pollutants' exposure; one reason could be lack of dissemination of comprehensive scientific findings to the public for awareness and precaution, and the government for policy formulation.

Therefore, critical review of existing research works conducted in urban areas of developed nations, and appropriate translation and implementation are necessary for the wellbeing of citizens. The aim of this review is to conduct literature survey on PM-induced specific cause mortality due to cardiovascular diseases, hypertension, obesity and diabetes mellitus, and cancer, and total (all) cause mortality to play its part in communicating existing problems.

Methodology

In order to conduct this task, published articles on related journals that are indexed and abstracted on Google scholar are systematically searched and reviewed. The search is performed using phrases like "PM and health," "PM exposure effects," "PM exposure and cardiovascular," "hypertension," "obesity," "diabetes" and "cancer," and "air pollution and health effects" each separately and categorized as "general" and "specific." The "general" labeled folder contained those articles referring to the property, composition, size distribution, and way of particle deposition. The specific one had subfolders of cardiovascular, hypertension, cancer, diabetes, and all-cause mortality. Publication year and PM-induced health effect are the main inclusion and exclusion criteria. Journal articles and reports published for the last 15 years were included in the review. Accordingly, 16, 10, 15, 9, and 16 articles were used for tabulation of PM exposure effects on cardiovascular disease, hypertension, diabetes, cancer, and all-cause mortality, respectively. For reference management and citation, articles in the respective folders were exported to Mendeley Desktop 1.19.4, checked for duplication and accuracy. The associations between PM and the respective health morbidity and mortality risks published by different authors are presented in tabular forms.

Size and composition of particulate matter

The physical property of PM can mainly be seen in terms of size which is a very important factor when related to composition of PM, health, and environmental effects (Franck et al. 2011; Karottki et al. 2014). PM has been differentiated as PM_{10} and $PM_{2.5}$ and groups particle pollution into the two categories by the USEPA. The former includes all particles less than 10 μm, while the later includes all particles having diameters less than 2.5 μm (fine particle) (Araujo et al. 2008). There are also others such as ultrafine particles (UFPs) which have diameters less than 0.1 μm, and total suspended particles (TSPs), which include all particles that are less than 40–50 μm in diameter (WHO 2013; Kim et al. 2014).

Apart from size, the chemical property and toxicity of PM are also highly depending on chemical composition (Bell et al. 2009; Kelly and Fussell 2012; Heo et al. 2014). The variation in composition is due to the absorption, adsorption, transportation, and transference of various pollutants (Kampa and Castanas 2008). Generally, PM is composed of inorganic and organic masses, elemental carbon mass, metals and non-metals, semi-volatile species crystal, and biological materials by which the chemical property relays on (Heo et al. 2014). The mass composition along with the specific constituent is presented in Table 1.

The organic and elemental masses contain both toxic components that affect human health depending on intensity and duration of exposure.

Table 1 Constituents of PM that affect its toxicity properties

PM mass constituent	Specific constituents/ primarily emitted/	Reference
Organic mass	Hydrocarbons and their derivatives: PAHs, PFCs, pesticides, dioxins, furans, and PCBs	Kampa and Castanas (2008), Falcon-Rodriguez et al. (2016)
Elemental mass	C, Si, Be, As, Na, Ni, V, Pb, Cd, Se, Zn, Br, Co, Mn, and others	Bell (2012), Kastury et al. (2017)
Atmospheric semi-volatile species	H_2O, NH_4NO_3, semi-volatile organic compounds	Falcon-Rodriguez et al. (2016)
Crustal materials	Sand, soil, and dusts	Hui et al. (2015)
Biological materials	Bacteria, spores, pollen, debris, and plant fragments	Kampa and Castanas (2008)

PAHs polycyclic aromatic hydrocarbons, *PFCs* polyfluorinated carbons, *PCBs* polychlorinated biphenyls

Route, intensity of exposure, and particle deposition

The most common route of human exposure to PM is inhalation through the mouth and nose followed by ingestion (Martin et al. 2014). The volume of air inspired with each normal breath (tidal volume) is about 500 ml in the adult male. But in women pulmonary volumes and capacities are about 20–25% less than men, and they are greater in large size and elite people than in small size and inactive people (Stieb et al. 2017). The metabolic demands of exercise increase minute of ventilation and therefore the rate of inhalation of pollutants increase. Breathing through the nose filter some percent of inhaled pollutants, but during exercise air intake usually comes through the mouth that enables air pollutants more directly get in to the lungs (Stieb et al. 2017).

The deposition of particles in the lung is governed by particle characteristics, anatomy of the respiratory tract, tidal volume, and breathing pattern (Carvalho et al. 2011). The main deposition mechanisms are impaction, sedimentation, and diffusion (Falcon-Rodriguez et al. 2016). Impaction is thought to be the principal mechanism of large PM deposition, and sedimentation happens to particles that are allowed to fall under their own force of gravity. Diffusion affects the smallest particles as they are displaced by random gas motion and will principally occur in the small airways and gas exchange regions of the lung (Hussain et al. 2011; Pražnikar and Pražnikar 2012). Fine and ultrafine particles are deposited directly in the alveolar regions of the lungs (Kawanaka et al. 2011), and may even be able to cross into the blood if not removed (Terzano et al. 2010). Alveolar macrophages and lung epithelial cells work to remove the toxics to be cleared by the lymphatic system (Tournier et al. 2007) depending on the pollutant and antioxidant ratio.

Associations between urban PM exposure and health risks

Almost all of the health risks induced by PM exposure can be mainly categorized into two ways: oxidative stress and genetic changes (mutagenicity and genotoxicity). PM exerts its health effects acting as oxidants of lipids and proteins or as free radical generators, promoting oxidative stress and inducing inflammation to cells (Pinkerton et al. 2008; Chen et al. 2016). Free radicals (reactive oxygen (ROS) and nitrogen species) are harmful to cellular lipids, proteins, and nuclear or mitochondrial DNA, inhibiting their normal function (Kampa and Castanas 2008). The extent of the oxidative damage is depending on the type and concentration of PM constituents.

The adverse health effects associated with oxidative stress posed by PM exposure can be seen in three different responses based on the proportion of free radicals to their scavengers in the body (Valavanidis et al. 2013; Billah 2016). First, when free radicals are lower, a series of antioxidant and detoxification enzymes are stimulated to scavenge ROS (Valko et al. 2007). Second, when ROS concentration is greater than the antioxidant concentration, it will lead to the formation of pro-inflammatory situation and lead to cytotoxicity of the cells. Third, when the oxidative stress level is high, it overwhelmed the antioxidant defenses and subsequently induces cytotoxic effects (Billah 2016). The free radical to scavengers' ratio is depending on the constituent of the particle exposed to an organism.

The other health effect due to PM exposure is through genetic changes which is associated with the presence of organic components like polycyclic aromatic hydrocarbons (PAH) and their derivatives (Vincent-Hubert et al. 2012; Jia et al. 2017). Among these, the nitro-PAHs have often been implicated in mutagenic and genotoxic activity (Vincent-Hubert et al. 2012; Jia et al. 2017; Mannucci and Franchini 2017). Another study showed that both the organic and inorganic components of PM contributed to the induction of DNA damage, but about 75% of the damage is induced by the organic component (Lodovici and Bigagli 2011) of PM through direct toxicity. The direct effect implies that the particles themselves could be toxic as particles may contain toxic metals and non-metals, such as Pb, Cd, Ni, Hg, As, and radionuclides (Yu 2005). It also has indirect effects through adsorbing toxic chemicals mostly organics, such as carcinogens like PAH, and enhances their effect by either increasing their penetration into the lungs, or prolonging their residence time in the respiratory tract (Kampa and Castanas 2008).

Urban PM is considered as one of the most hazardous pollutants for human health. Studies indicated a causal relationship between inhalable particles (PM_{10}, $PM_{2.5}$, and UFP) in the atmosphere and human morbidity and mortality, especially in people with preexisting respiratory or cardiovascular diseases (Brook et al. 2010; Mills et al. 2008; Bell 2012; Kim et al. 2015). It induced different health effects by causing

chronic and acute inflammation by realizing a protein that may modify cells to abnormality (Kurniawan et al. 2018), and initiate and/or promote mechanisms of carcinogenesis (Valavanidis et al. 2013). Brief exposures (within hours) to UFP and $PM_{2.5}$ can induce acute pathophysiological responses (Breitner et al. 2019).

In this regard, traffic-related PM-induced health effects in different metropolitan areas have been reported and the effect is more than industrial and other sources (Beevers and Carslaw 2005; Mcentee and Ogneva-himmelberger 2008; Fan et al. 2009; Levy et al. 2010; De Coensel et al. 2012; Laumbach and Kipen 2012). Quantitatively, for an increase of $PM_{2.5}$ by 1 mg/m^3, the adjusted odd ratio (OR) of street retailer reporting symptoms increases 1.022 for eye irritation and 1.027 for dizziness (Kongtip et al. 2006).

A study report conducted in 272 Chinese cities indicated that each 10 μg/m^3 increase in 2 days moving average in $PM_{2.5}$ concentrations is significantly associated with increments in mortality of 0.22% from total causes, 0.27% from cardiovascular diseases, 0.39% from hypertension, 0.30% from coronary heart diseases, 0.23% from stroke, 0.29% from respiratory diseases, and 0.38% from COPD (Chen et al. 2018a, b). The mortality hazard ratios (HRs) (95% confidence interval (CI)) per 10 μg/m^3 increase in $PM_{2.5}$ are 1.09 (1.08–1.09) for non-accidental causes, 1.09 (1.08–1.10) for cardiovascular, 1.12 (1.10–1.13) for COPD, and 1.12 (1.07–1.14) for lung cancer (Yin et al. 2017a, b).

This is an evidence that long-term exposure to particulate pollution is associated with different health and mortality risks: pulmonary, cardiopulmonary, cardiovascular, hypertension, obesity, diabetes, and cancer. This review presents beyond pulmonary health effects.

Associations between PM exposure and cardiovascular health risks

The heart can be affected by PM through uptake of particles into the circulation or the subsequent release of soluble substances into circulation. Three main mechanisms have been reported for effects of PM on cardiovascular system. The first is the inflammatory response induced in the lungs leads to release cytokines and other mediators that reach to the systemic circulation (Miller et al. 2012). Second, UFPs themselves may translocate across the alveolar wall and directly interact with the cardiovascular system (Jia et al. 2017). Third, particles may activate the autonomic nervous system through sensory receptors on the alveolar surface. This process is reported as a rapid way of affecting cardiovascular function (Miller et al. 2012). All these ways cause oxidative stress as the main for cardiovascular disease (CVD), death or hospitalization as it is central to the initiation, progression, and destabilization of atherosclerotic plaques and involves cholesterol in its well established contribution to risk of CVD (Chen et al. 2016; Levy 2007).

Simkhovich et al. (2008) and Kim et al. (2015) also reported the effect of inhalation of fine ($PM_{2.5}$) and UFPs ($PM_{0.1}$) and other air pollutants affecting the heart rate, heart rate variability, blood pressure, vascular tone, blood coagulability, and the progression of atherosclerosis in the general population, and those with preexisting cardiovascular disease and diabetic and elderly individuals. Exposure to $PM_{2.5}$ is associated with an increase in the long-term risk of cardiopulmonary mortality by 6–13% per 10 μg/m^3 of $PM_{2.5}$ (Perrino 2010) through developing coronary artery disease (McGuinn et al. 2017). At low exposure levels, $PM_{2.5}$ exposure causes CVD than carcinogenic effects (Pope et al. 2011). Studies on CV health effects and mortality are compiled as presented in Table 2.

An epidemiological study reports in a multi-country level also showed that an increase of 10 μg/m^3 in $PM_{2.5–10}$ size, even for short-term exposure, is associated with a 0.76% increase in CV deaths (Analitis et al. 2006), mostly through clogging of arteries and heart failure (Bartoli et al. 2009). In addition, a 0.12% increase (95% CI, 0.001–0.25) in CVD, a 0.17% (95% CI, − 0.04–0.40) increase in coronary heart disease, and a 0.13% (95% CI, − 0.12–0.33) increase in stroke in association with a 10 μg/m^3 increase in $PM_{2.5}$ concentrations on the same day have been reported (Chen et al. 2018a, b). Among the different effects, the largest $PM_{2.5}$ association reported for cardiovascular is for heart failure which is a 1.28% (95% CI, 0.78–1.78%) increase in risk per 10 μg/m^3 increase in same day (Dominici et al. 2006). In general, about 3% of cardiopulmonary deaths is attributable to PM globally (WHO 2013).

All these are evidences indicated associations between CV morbidity and mortality cases and PM exposure. These are mostly caused by vehicular emissions, and those who live very near to transportation route, especially highways, are highly susceptible (Brugge et al. 2007). Due to its high prevalence, mostly due to traffic, the American Heart Association's Strategic Impact Goal through 2020 and beyond set a criterion to reduce the prevalence. "By 2020, to improve the cardiovascular health of all Americans by 20% while reducing deaths from cardiovascular diseases and stroke by 20%" (Lloyd-Jones et al. 2010). All countries should be encouraged to set their own criteria and encourage the public to reduce emissions and associated health risks.

Associations between PM exposure and hypertension risks

The effect of PM exposure on the CV system could also be implicated effects to high blood pressure. For example, if a subject with preexisting cardiac disease is exposed to particle, it may be at increased risk of cardiac morbidity and mortality

Table 2 Associations of PM exposure and cardiovascular (CV) health effects

Study design	Country	Main finding (mean at 95% CI)	Reference
Time-series study on 12 million Medicare enrollees	USA	A 10 μg/m^3 increase in $PM_{10-2.5}$ was associated with a 0.36% increase in CVD admissions on the same day.	Peng et al. (2008)
Prospective cohort study of 71,431 middle aged	China	Each 10 μg/m^3 increase in PM_{10} is associated with a 1.8% increased risk of CV mortality.	Zhou et al. (2014)
Cross-sectional study of 669,046 participants	USA	Long-term exposure may contribute to the development or exacerbation of CVD and cardio-metabolic mortality	Pope et al. (2009)
Time-series study of aged persons (> = 65 years)	USA	An IQR increase in same day $PM_{2.5}$ road dust (1.71 μg/m^3) is associated with a 2.11% increase in CVD admissions.	Bell et al. (2014)
Time-series study of aged persons (> = 40 years)	USA	$PM_{2.5}$ chemical components are correlated with CVD hospitalizations and CVD mortality.	Ito et al. (2011)
Time-series regression analyses of California residents	USA	$PM_{2.5}$ is associated with a 3–5% increase in CV daily mortality among non-high school graduates.	Ostro et al. (2008)
Cohort study of 100,166 persons of 11.5 years	European countries	A 100 ng/m^3 increase in PM_{10} and a 50 ng/m^3 increase in $PM_{2.5}$ are associated with a 6% and 18% increase in coronary events, respectively.	Wolf et al. (2015)
Cohort study of 8000 persons	England	A 10 g/m^3 increase in $PM_{2.5}$ is associated with 28% increase in CVD risk of mortality.	Laden et al. (2006)
Cohort study of 660,000 persons	Great Britain	A 10 g/m^3 increase in $PM_{2.5}$ is associated with a 1.7% increase in CVD risk of mortality.	Elliott et al. 2007
Biomarker study	China	Daily consecutive $PM_{2.5}$ higher than 105 μg/m^3, risk of CV mortality	Wang et al. (2017a)
Time-series analysis of hospitalization	USA	A 10 μg/m^3 increment in $PM_{2.5}$ concentration accounted for a 1.37% increase in CVD	Hsu et al. (2017)
Meta-analysis commentary	China	A 10 μg/m^3 increment in $PM_{2.5}$ concentration accounted for a 5.91% increase in CVD	Liu et al. (2018)
Narrative review	Different	Higher CV mortality for both PM_{10} (0.6–1.8% for an increase of 20 μg/m^3) and $PM_{2.5}$ (0.6–1.3% for an increase of 10 μg/m^3)	Brook et al. (2010), Martinelli et al. (2013)
Time-stratified-case crossover	China	A 10 μg/m^3 increment in 2-day moving average of $PM_{2.5}$ corresponded to 0.53% increase in CV mortality	Ma et al. (2011)
Cohort study of 500,715 adults	USA	Odds of CVD increases by1.32 and 1.15 times per 10 mg/m^3 increase in $PM_{2.5}$ and PM_{10}, respectively.	Feng and Yang (2012)

through short-term increases in systemic arterial vascular narrowing, as manifested by increased peripheral blood pressure (Auchincloss et al. 2008; Bartoli et al. 2009).

Abundant studies in developed regions also reflected the association between PM exposure and health risks due to hypertension. To mention, Guo et al. (2010) and Coogan et al. (2012) reported that exposure to elevated level of particles is associated with emergency hospital visits due to hypertension, and increment of hypertension cases due to particle exposure, respectively. Brook and Rajagopalan (2009) reported the possibility of increasing blood pressure (BP) with a few days exposure of $PM_{2.5}$, and development of chronic hypertension with its long-term exposure. Reviewed literatures along with their main finding in the area are indicated in Table 3.

Kearney et al. (2005) reported the global burden of hypertension as 26.4% for the adult population in 2000 of which hypertension prevalence for men and women are 26.6% and 26.1%, respectively. These numbers are projected to be 29.2%

by 2025 (29.0% of men and 29.5% of women), increased by about 60%. Zhang et al. (2016) reported that long-term exposure to PM is associated with an increased risk of incident hypertension, particularly among younger women and the obese. Such an increase risk in women also affects pregnancy. For example, Van Den Hooven et al. (2011) reported that PM_{10} is associated with a steeper rise in SBP throughout pregnancy, and each increment in 10 g/m^3 is associated with an increased risk of pregnancy-induced hypertension (OR = 1.72 [95% CI 1.12–2.63]. In addition, a 1.6 (95% CI = 0.02, 3.3; p = 0.048) (Sears et al. 2018), 1.88 (95% CI = 0.84–2.93) (Lee et al. 2012) mmHg increase in SBP, and 0.63 (Sears et al. 2018) mmHg DBP associated with traffic attributed elemental carbon concentration.

The maternal exposure also associated with lower placenta weight (van den Hooven et al. 2012). Vinikoor-Imler et al. (2012) and Savitz et al. (2016) in their part reported both PM_{10} and $PM_{2.5}$ are positively associated with gestational hypertension that causes pregnancy disorder or elevations in BP during pregnancy. The risk ratios reported for an IQR increase in exposure are 1.07 [95% confidence interval (CI) 1.04, 1.11] for PM_{10} (IQR 3.92 mg/m^3) and 1.11 [95% CI 1.08, 1.15] for $PM_{2.5}$ (IQR 2.24 mg/m^3) (Vinikoor-Imler et al. 2012). The exposure to high levels of $PM_{2.5}$ in the third trimester of pregnancy is associated with 42% increased stillbirth risk [AOR = 1.42 (1.06, 1.91)] (DeFranco et al. 2015), and the greatest risk of being very low birth weight (AOR = 1.27, 95% CI = 1.08–1.49) (Salihu et al. 2012). Each 0.52 μg/m^3 increase in Delta-C (wood burning) concentration during the seventh gestational month is associated with an increased odds of hypertensive disorders of pregnancy (OR = 1.21; 95% CI = 1.01–1.45) (Assibey-Mensah et al. 2019). In addition, exposure to $PM_{2.5}$ in the 1st trimester is significantly associated with hypertensive disorders of pregnancy [ORs per 2-standard deviation increase in $PM_{2.5}$ (7 mg/m^3) is 9.10 [95% CI 3.33–24.6]] (Mobasher et al. 2013). The number of $PM_{2.5}$ associated preterm births is estimated as 2.7 million with a concentration of 10 μg/m^3 (Malley et al. 2017).

All these studies revealed that long-term exposure to PM is strongly and positively associated with BP. The association is higher for pregnancy and preterm births which needs international communications and policy actions for mitigation.

Associations between PM exposure with weight gain and diabetes

In addition to cardiovascular and hypertension effects, inhaled particles could also cause diabetes (Liu et al. 2014; Rajagopalan and Brook 2012) and non-diet-induced weight gain (Shamy et al. 2018; Wei et al. 2016) through different biologic pathways. The diabetes pathways include endothelial dysfunction, dysregulation of the visceral adipose tissue through inflammation, hepatic insulin resistance, elevated

Table 3 Association of PM exposure and hypertension

Method	Country	Main finding (mean ratio at 95% CI)	Reference
Cross-sectional study of 5112 persons, 45–84 years of age	USA	A 10 μg/m^3 increase in $PM_{2.5}$ 30-day mean is associated with 1.12 mmHg higher pulse pressure and 0.99 mmHg higher systolic BP.	Auchincloss et al. (2008)
A stratified survey of 347 participants and biomarker	USA	A 10 g/m^3 increase in daily $PM_{2.5}$ is associated with a 3.2 mmHg increase in systolic blood pressure (SBP).	Dvonch et al. (2009)
Crossover trial of controlled-human exposure to conc. ambient particles	USA	Both $PM_{2.5}$ and PM_{10} significantly increase SBP (2.53 mmHg and 1.56 mmHg, respectively).	Bellavia et al. (2013)
A cohort study on 4291 participants, 45–75 years of age	Germany	An IQ increase in $PM_{2.5}$ (2.4 μg/m^3) is associated with estimated increases in mean SBP and DBP of 1.4 mmHg and 0.9 mmHg, respectively.	Fuks et al. (2011)
Experimental study on $PM_{2.5}$ and particle-free air in 23 healthy adults	Canada	Significant increase in DBP (6 mmHg (9.3%)) is reported at 2 h of $PM_{2.5}$ exposure.	Urch et al. (2005)
Case-crossover study on 1491 emergency hospital visits (EHVs)	China	An increase in 10 μg/m^3 in $PM_{2.5}$ and PM_{10} is associated with EHVs for hypertension with OR of 1.084 and 1.060%, respectively.	Guo et al. (2010)
A cohort study of 35,303 non-hypertensive adults	Canada	For every 10 μg/m^3 increase of $PM_{2.5}$, the HR of incident hypertension is 1.13.	Chen et al. (2014)
A cohort study of black women	USA	A 10-g/m^3 increase in $PM_{2.5}$ is associated with hypertension (RR = 1.48).	Coogan et al. (2012)
Cross-sectional study with a total of 4166 participants	Germany	Traffic $PM_{2.5}$ is associated with a higher prevalence of hypertension. The adjusted OR for a 1-μg/m^3 increase in $PM_{2.5}$ was 1.15.	Babisch et al. (2014)
	Brazil	The RR of hospitalizations due	Nascimento and

Table 3 (continued)

Method	Country	Main finding (mean ratio at 95% CI)	Reference
Time-series study on hospital admissions due to hypertension		to hypertension increased 11% with a 10-μg/m^3 increment in PM_{10} concentrations.	Francisco (2013)

BP blood pressure, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

Table 4 Association of PM exposure and type 2 diabetes mellitus

Method	Country	Main finding (mean at 95% CI)	Reference
Cohort study on 24, 174 nurses	Denmark	Existence of significant positive association between $PM_{2.5}$ and diabetes incidence (HR = 1.11 per IQR 3.1 μg/m^3) and weaker for PM_{10} (HR = 1.06 per IQR 2.8 μg/m^3)	Hansen et al. (2016)
Meta-analysis of 55 articles	Different	With a 10-μg/m^3 increase in $PM_{2.5}$, the risk of T2DM would increase by 25% in the long-term exposure (RR = 1.25).	He et al. (2017)
Cross-sectional study 15, 477 subjects	China	PM_1, $PM_{2.5}$, and PM_{10} are significantly associated with increased diabetes prevalence (OR = 1.13, 1.14, and 1.20, respectively).	Yang et al. (2018)
Two years study by multivariate regression	USA	A 1% increase in diabetes prevalence seen with a 10 g/m^3 increase in $PM_{2.5}$ exposure (PR = 0.77)	Pearson et al. (2010)
Cohort study of 2.1 million adults	Canada	A 10 mg/m^3 elevation in $PM_{2.5}$ exposure is associated with an increase in risk for diabetes-related mortality (HR = 1.49)	Brook et al. (2013)
Cohort study of 62,012 non-diabetic adults	Canada	The HR for a 10 μg/m^3 increase in $PM_{2.5}$ is 1.11 which may contribute to the development of diabetes.	Chen et al. (2013)
Cohort analysis 3607 non-diabetics	German	Living closer than 100 m to a busy road has more than 30% higher risk (1.37) than those living further than 200 m away.	Weinmayr et al. (2015)
Mini review of literatures	Different	Risk of future diabetes associated with exposure to 10 μg/m^3 increase of $PM_{2.5}$ is 10–27%, and its mortality around 1%	Esposito et al. (2016)
Cross-sectional study on 11,847 adults	China	An IQR increase in $PM_{2.5}$ (41.1 μg/m^3) is significantly associated with increased T2DM prevalence (PR = 1.14).	Liu et al. (2016)
Experimental on mice exposure to $PM_{2.5}$	China	Early-life exposure to high levels of $PM_{2.5}$ is a risk factor for subsequent development of insulin resistance, adiposity, and inflammation.	Xu et al. (2010)
Cross-sectional study of 6392 participants	Switzerland	PM_{10} is associated with prevalent diabetes with respective OR of 1.40 per 10 μg/m^3 increase in outdoor concentration	Eze et al. (2014)
Cohort-case control	Switzerland	Odds of diabetes increased 35% per 10 μg/m^3 exposure to PM_{10}	Eze et al. (2016)
Meta-analysis of 3 studies	Different	Increased risk of T2DM by 8–10% per 10 μg/m^3 increase in exposure (OR = 1.10)	Eze et al. (2015)
Cohort study of 4121 older people	USA	IQR of 3.9 μg/m^3 increase in 1-year moving average $PM_{2.5}$ is associated with increased diabetes prevalence (OR = 1.35).	Honda et al. (2017)
A cohort study of black women	USA	The corresponding IRR for diabetes mellitus for a 10 g/m^3 increase in $PM_{2.5}$ was 1.63.	Coogan et al. (2012)

hemoglobin level, elevated blood pressure, and alterations in autonomic tone which may increase insulin resistance (Esposito et al. 2016; Liu et al. 2014).

Meo and Suraya (2015) also reported air pollution as a leading cause of insulin resistance and incidence of type 2 diabetes mellitus (T2DM), contributing to the global prevalence rate. Based on the International Diabetes Federations (IDF 2017), the global prevalence rate is 8.3% which means that 425 million adults are diabetics, and the number is expected to rise to 629 million by 2045. For Africa, a total of 16 million people live with diabetes as of 2017 and the number could reach to 41 million (156% increase) by 2045, and Ethiopia is the leading country having 2,652,129 by 2017 with a prevalence rate of 7.5%. The fact that 75% of those with diabetes are living in low- and middle-income countries (IDF 2017; Ogurtsova et al. 2017) could exacerbate the situation as little control on air pollution is practiced in the regions.

Almost all reports (Table 4) indicated the existence of strong positive associations between particulate exposure

Table 5 Association of PM Exposure and cancer

Method and study population	Country	Main finding (mean at 95% CI)	Reference
Meta-analysis and systemic review	Different	The meta-relative risk for lung cancer associated with $PM_{2.5}$ and PM_{10} is 1.09 and 1.08, respectively.	Hamra et al. (2014)
Cohort study of 188,699 participants	Canada	Each 10 mg/m^3 increase in $PM_{2.5}$ is associated with a 15–27% increase in lung cancer mortality.	Turner et al. (2011)
Meta-analysis of 14 cohort studies	European countries	Statistically significant associations were found for $PM_{2.5}$ Cu (HR, 1.25; per 5 ng/m^3), PM_{10} Zn (1.28, per 20 ng/m^3), PM_{10} S (1.58, per 200 ng/m^3), PM_{10} Ni (1.59, per 2 ng/m^3), and PM_{10} K (1.17, per 100 ng/m^3).	Raaschou-nielsen et al. (2016)
Ecological time series study	Brazil	PM_{10} is associated with incidence and mortality for some types of cancer (Pearson correlation up to 0.86).	Yanagi et al. (2012)
Cohort study of 103,650 women	USA	A 10 μg/m^3 increase in 72-month average PM_{10} (HR = 1.04), $PM_{2.5}$ (HR = 1.06), or $PM_{2.5–10}$ (HR = 1.05) is positively associated with lung cancer.	Puett et al. (2014)
Cohort study on all female breast cancer cases (255,128)	USA	Exposure to higher PM_{10} (HR 1.13, per 10 μg/m^3) or $PM_{2.5}$ (HR 1.86, per 5 μg/m^3) is significantly associated with early mortality among female breast cancer cases.	Hu et al. (2013)
Cohort study on all breast cancer cases	Italy	Risk of BC death is significantly higher due to $PM_{2.5}$ exposure (HRs of death 1.82)	Tagliabue et al. (2016)
		A statistically significant association between risk for lung cancer and PM_{10} (HR = 1·22 per 10 μg/m^3). For $PM_{2·5}$ the HR is 1·18 per 5 μg/m^3.	Raaschou-nielsen et al. (2016)
Meta-analysis of 17 cohort studies	European countries		
Cohort study of 8096 participants	England	A 10 g/m^3 increase in $PM_{2.5}$ exposure is associated with lung cancer (RR, 1.27).	Laden et al. (2006)

and risk of diabetes and its cause mortality. Traffic is also reported as the main risk factor for diabetes. There are other reports that also support traffic PM-induced diabetes. For example, the hazards for diabetes are reported as increased by 15–42% per IQR of PM from traffic-related exposure (Krämer et al. 2010), and the association of diabetes with distance to road (traffic related pollution) is shown among women (Puett et al. 2011). This can also be interlinked with pregnancy and premature health effects.

Similar to hypertension, the association of PM exposure is significantly higher on pregnancy and premature health effects than other individuals. A 10-μg/m^3 increase of $PM_{2.5}$ exposure during the entire pregnancy is significantly associated with a decrease of 13.80 g (95% CI = − 21.10 to − 6.05) in birth weight. The OR for a premature birth is also reported as 1.06 (95% CI = 1.01–1.13) for each 10 μg/m^3 increase of $PM_{2.5}$ exposure during the entire pregnancy period (Kloog et al. 2012). Greater exposure to $PM_{2.5}$ during the second trimester is associated with gestational diabetes mellitus (Fleisch et al. 2016), and those living close to a major roadway are associated with higher odds (1.09, 95% CI 1.00–1.19) of gestational diabetes mellitus (Choe et al. 2018).

With regard to obesity, the toxicity mechanisms are as follows: pro-inflammatory cytokine release, systemic inflammation, and recruitment of inflammatory cells to adipose and other tissues (Wei et al. 2016). Rao et al. (2015) reported that the toxicity effect of high levels of air pollution (dominated $PM_{2.5}$) may be equivalent to high-fat diet having toxicity mechanisms of lipogenesis, lipolysis, hypothalamic inflammation, etc.

Associations between PM exposure and cancer

Lipid peroxidation has been implicated as a major mechanism in the generation of diseases and disorders such as cancer, cardiovascular, and neurodegenerative diseases that function

Table 6 Association of PM exposure and all-cause mortality

Study design	Country	Main finding (mean at 95% CI)	Reference
Quasi-experimental variation	China	A 10-μg/m^3 increase in PM_{10} reduces life expectancy by 0.64 years.	Ebenstein et al. (2017)
Adult cohort study	England	An increase in overall mortality associated with each 10 μg/m^3 increase in $PM_{2.5}$ (RR = 1.16)	Laden et al. (2006)
Adult cohort study	England	Improved overall mortality is associated with decreased mean $PM_{2.5}$ (10 g/m^3) (RR = 0.73).	
Cohort study	USA	A decrease of 10 μg/m^3 in $PM_{2.5}$ is associated with increase in life expectancy of 0.61.	Pope et al. (2009)
Cohort study	California	A 10 μg/m^3 change in 2-day average $PM_{2.5}$, associated with a 0.6% increase in all-cause mortality	Ostro et al. (2006)
Time series analysis	USA	A 1.1% increase and 2.3% increase in total mortality for a 10 μg/m^3 increase in the same day and the 2-day average of $PM_{2.5}$, respectively	Zanobetti et al. (2014)
Cohort study	China	A 0.13% increase in all-cause mortality with a 10 μg/m^3 increase in $PM_{2.5}$ on the same day	Chen et al. (2018a, b)
Time-stratified case-crossover analysis	China	A 10 μg/m^3 increment in the 2-day moving average $PM_{2.5}$ corresponded to 0.49% increase of total mortality	Ma et al. (2011)
Cohort study	China	A 10 μg/m^3 increase of $PM_{2.5}$ could lead to 5.37% in all-cause mortality.	Liu et al. (2018)
Cohort study (> = 65)	China	A 10 μg/m^3 increase in $PM_{2.5}$ is associated with increase in all-cause mortality (HR = 1·08).	Li et al. (2018)
A time-stratified case-crossover study	USA	A 1.21% increase in all-cause mortality with a 10 mg/m^3 increase in previous day's $PM_{2.5}$	Franklin et al. (2007)
Cross-sectional study on 51 cities	USA	A 10 μg/m^3 increase in the annual $PM_{2.5}$ is associated with 4% increases in all-cause mortality.	Pope III et al. (2009)
Cohort study	USA	A 10 μg/m^3 increase in the annual $PM_{2.5}$ is associated with 7.3% increases in all-cause mortality.	Di et al. (2017)
Cohort study	USA	A 10 μg/m^3 increase in the annual $PM_{2.5}$ is associated with 20.8% increases in all-cause mortality.	Eftim et al. (2008)
Case crossover and meta-analysis	USA	A 1.21% increase in all-cause mortality with a 10 mg/m^3 increase in previous day's $PM_{2.5}$	Franklin et al. (2007)
Time-series analysis	China	Daily mortality of total causes is estimated to increase by 0.44% per 10 μg/m^3 of $PM_{10.}$	Yin et al. (2017a, b)
Cohort study of aged society (> = 65 years)	China	For each 10 μg/m^3 increase in $PM_{2·5}$ concentration, estimated HR of 1·08 is reported.	Ji (2018)

through alteration of the integrity, fluidity, and permeability of the membranes (Jan et al. 2015). Moreover, PM could make the cells produce pro-inflammatory mediators such as cytokines that promote a local response to exacerbate COPD and asthma and promote lung infection (van Eeden 2005). These cells also induce a systemic inflammatory response that includes stimulation of the marrow to release leukocytes and platelets, activation of proteins that may increase coagulability, and activation of the vascular endothelium (the layer of cells line in the blood vessels directly in contact with the blood) (Pinkerton et al. 2008; Chen et al. 2016). The association between PM exposure and cancer is shown in Table 5.

Vehicular emission is also reported as the main source of PM for lung cancer mortality. An increase in road traffic of 4000 vehicle/Km per day within 100 m of the residence was associated with HR for lung cancer of mean 1.09 (Raaschou-nielsen et al. 2016). Such anthropogenic sources of $PM_{2.5}$ are associated with 220,000 ± 80,000 lung cancer mortalities annually (Anenberg et al. 2010). For particular mortality cases, each 10 μg/m^3 elevation in $PM_{2.5}$ is associated with 8% (Pope

et al. 2009) and 8.93% (Liu et al. 2018) increased risk of lung cancer mortality.

The burden of disease for long-term exposure and at higher doses of $PM_{2.5}$ is attributed to lung cancer, and a nearly linear exposure–response relationship for $PM_{2.5}$ and lung cancer mortality is reported (Pope et al. 2009). Prenatal exposure to PM that contains PAH can also cause genetic alterations that linked to increased cancer risk (Moorthy et al. 2015). Globally, 5% of lung cancer deaths are attributable to PM (WHO 2013).

All-cause mortality

The effects of $PM_{2.5}$ on daily mortality vary with source, season, and locality (Zhou et al. 2011). About 32% of reported deaths, with a mortality rate of 1.9%, are associated with $PM_{2.5}$, in which deaths from cardiovascular, respiratory, and lung-cancer causes accounted for 20%, with a mortality rate of 1.2% (Fang et al. 2016). A total of 169,862 additional deaths from short-term $PM_{2.5}$ exposure throughout China is also reported (Li et al. 2019). A higher percentage increase in mortality associated with long-term exposure to $PM_{2.5}$ is reported for lower home value, or lower median income (Wang et al. 2016) (Table 6).

According to Song et al. (2017), $PM_{2.5}$ contributed as much as 40.3% to total stroke deaths, 33.1% to acute lower respiratory infection deaths, 26.8% to ischemic heart disease deaths, 23.9% to lung cancer deaths, 18.7% to chronic obstructive pulmonary disease deaths, and 30.2% to total deaths.

Mortality due to PM exposure is more pronounced in premature cases. For example, in China, it accounted to 1.27 million in total, and 119 (16.8%), 167 (9.5%), 83,976 (20%), and 390 (53.2%) for adult chronic obstructive pulmonary disease, lung cancer, ischemic heart disease, and stroke, respectively (Li et al. 2018; Wang et al. 2018). Annual national mortality rates for China indicated 2.19 million (2013), 1.94 million (2014), and 1.65 million (2015) premature deaths attributed to $PM_{2.5}$ long-term exposure (Li et al. 2018) which is higher than 1.27 million. Based on this situation, total premature mortality attributable to $PM_{2.5}$ could reach 574 thousands across only 161 cities by the end of year 2030 (Maji et al. 2018), requires scientific and legal actions for controlling emission to the level conducive to public health.

Due to air pollution control laws and practices, $PM_{2.5}$-induced mortalities in developed regions of Europe and North America have decreased substantially by 67% and 58%, respectively. In contrast, associated mortalities in East Asia and South Asia increased by 21% and 85%, respectively (Wang et al. 2017b). The increment rate may be higher for Africa due to increasing industrialization and older vehicle importation, and little air pollution control practices as indicated in state of the environment reports. Therefore, there should be scientific control strategies and legal enforcements intended to decrease the total number and old age vehicles used in Asian and African countries.

Conclusion

The aim of this review was to assess the associations between PM exposure and human health risks and mortality. There is a strong positive association between long-term exposure (mainly) to PM and cardiovascular disease, hypertension, cancer, obesity and diabetes, and all-cause mortality due to tremendous constituents like inorganic compounds, organic compounds, metals (heavy metals), and elemental carbon core that individually have adverse effects to human health. The exposure effects are more pronounced in gestational blood pressure and diabetes, and higher risks for those living road sides (up to 100 m) than living at a distance as the main cause for deadly particles are vehicles. The increase in hospital admissions of respiratory problems (like COPD), kidney failure, heart attacks, lung problems, high blood pressure, obesity, and diabetes in urban areas, the cause of which are left unknown for many of the patients, could be attributed to PM exposure. There should be scientific control strategies and legal enforcements intended to decrease the total number and old age vehicles used as vehicular emission is the main cause for urban mortality.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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