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Multifaceted health impacts of Particulate Matter (PM) and its management: An overview

Prabhat Kumar Rai

Department of Environmental Science, Mizoram University, Tanhril, Aizawl-796004, Mizoram, India

E-mail: prabhatrai24@gmail.com

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Abstract

Urban air quality is becoming a serious public health concern at global scale. Particulate matter (PM) pollution is intimately linked with human health. Present review describes the different human health implications associated with PM pollution. PM may derive its origin from natural and anthropogenic sources. Vehicle derived pollutants as well as industrial emissions simultaneously release deleterious fine-grained PM into the atmosphere. Fine PM especially PM_{2.5} and PM₁₀ are particularly deleterious to human health. Air pollution PM is an important environmental health risk factor for several respiratory and cardiovascular morbidity and mortality. Further, PM is inextricably linked with genotoxicity and mutations. Literature review of the cellular and molecular basis of adverse effects associated with PM is presented in this paper. Finally, management, existing technologies and policy options to reduce or mitigate the adverse health impacts of PM pollution is discussed as an eco-sustainable approach.

Keywords dust; cardiovascular disease; DNA adduct; mortality; epidemiology; heavy metals.

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1 Introduction

Particulate matter (PM) is closely associated with increases in morbidity and mortality (Maier et al., 2008; Abbas et al., 2009). The London smog of December 1952 is estimated to have caused some 4000 excess deaths (Harrison and Yin, 2000). In current Anthropocene, the problem of PM pollution is serious health concern for both developing and developed countries.

Solid matter, which is composed of soil, anthropogenic metallic constituents, and natural biogenic materials, is called dust (Ferreira-Baptista and DeMiguel, 2005; Rai, 2013). PM refers to a suspension of solid, liquid or a combination of solid and liquid particles in the air (Hinds, 1999; Wilson et al., 2005). Air pollution originating from PM is generally characterized by its highly complex nature (Alfaro-Moreno et al., 2002; Abbas et al., 2009). PMs are a mixture of particles and droplets in the air, consisting of a variety of

components such as organic compounds, metals, acids, soil, and dust (U.S. Environmental Protection Agency, 2006; Ciencewicki and Jaspers, 2007; Rai et al., 2014; Rai and Panda, 2014).

PM is one of six 'criteria pollutants' designated by the US Clean Air Act of 1971 (Wilson et al., 2005). The PMs belong to the class of poorly soluble particles that also encompasses carbon black, coal mine dust, and titanium dioxide (Borm et al., 2005; Moller et al., 2008). Measurements of the PM in ambient air are usually reported as the mass of particles with an aerodynamic diameter that is less than 2.5 μm ($\text{PM}_{2.5}$) or 10 μm (PM_{10}) (Zhu et al., 2006). Aforesaid particle sizes are emphasized in view of their pertinent health impacts (Jahn et al., 2011; Rohr and Wyzga, 2012; Taner et al., 2013; Rai, 2013; Hicken et al., 2014; Pascal et al., 2014; Yadava et al., 2014; Rai and Pana, 2014; Rai et al., 2014; Kim et al., 2015; Yang et al., 2015).

The urban air quality is continually affected by emission from stationery and mobile combustion sources. Mobile sources contribute to the emission of major urban air pollutants including carbon dioxide (CO_2), nitrogen oxides (NO_x), sulphur oxides (SO_x), particulate matter (PM), Lead (Pb), photo chemical oxidant, such as ozone (O_3) and ozone precursors like hydrocarbons and volatile organic compounds (Costa, 2004). Among these pollutants, the concentration of PM_{10} and $\text{PM}_{2.5}$ airborne aerosols showed a good agreement with traffic released pollutant and other combustion process (Prajapati and Tripathi, 2007). PM has been widely studied in recent years and the United Nation estimated that over 600 million people in urban areas worldwide were exposed to dangerous levels of traffic generated air pollutants (Cacciola et al., 2002).

2 Sources of PM Pollution

Sources of PM pollution may be natural as well as anthropogenic. Natural processes that emit PM into the atmosphere include volcanic eruption, geochemical sources, wind-blown dust, soil and spray from marine sources. Natural sources of PM e.g. volcanic eruptions may contain sulphurous particles and may have an adverse effect on cardio-respiratory health in adults (Longo et al., 2008). Anthropogenic sources include power plant, traffic, agriculture and various industrial activities such as mining and the metallurgical industries. Diesel exhaust emissions are the major source of $\text{PM}_{2.5}$ in urban environment. Vehicular emissions consist of PMs and gaseous emissions, with biologically active carbonaceous products present in both phases. Black carbon, mainly from diesels, is found in ultrafine and fine size fractions, mainly less than 1 μm in size and predominantly below 0.18 μm (Mauderly and Chow, 2008). Such vehicular particulates are often coated with condensed organic and inorganic compounds (Mauderly, 2001; Health Effects Institute, 1995). Ambient concentrations of the platinum group elements (PGE) platinum (Pt), palladium (Pd) and rhodium (Rh) have been on the rise, largely due to the use of automobile catalytic converters which employ these metals as exhaust catalysts and these PGE may impose a considerable human health risk (Wiseman and Zereini, 2009). Approximately 75% of diesel $\text{PM}_{2.5}$ emissions consist of such carbon (Health Effects Institute, 2003). Results of a case study of Restaurants in Turkey indicated that cooking is a significant source of indoor particulate matter that can cause adverse health effects and the lifetime cancer risk associated with As and Cr (VI) exposure was significant at selected restaurants, which might of concern for restaurant workers (Taner et al., 2013).

Anthropogenic emissions in conjunction with the topographical and meteorological conditions can result in high air pollution within the city which may inhibit the performance level as predicted before the Athens Olympics (Geraint et al., 2008). In Udaipur region, Rajasthan, India, PM increase may be associated with high morbidity particularly during winter season (Yadav et al., 2014)

Theophanides et al. (2007) evaluated the atmospheric pollution created by industry and traffic areas near to the Greek cities and the corresponding mortality of citizens in the region and found that adverse environmental impact of air pollutants is a major concern in the industrial centers.

3 Size Fractionation of PM

Particulate matters (PMs) can be classified as coarse, fine and ultrafine depending upon their particle size. PMs measured in urban air used in health effects studies and for regulation are:

- Nuclei mode (smaller than 0.1 μm), often referred to as ultrafine particles (UFPs); they do not last long in the air since they deposit or rapidly form fine particles by coagulation.
- Accumulation mode (between 0.1 and approximately 1.0–2.5 μm) account for the majority of the mass of suspended particles and deposit slowly leading to a long atmospheric life time of 5 to 10 days and the build-up of visible haze. These particles may readily penetrate indoor spaces and are most strongly linked to adverse health effects.
- Coarse mode (larger than 1 μm), which extends upto 100 μm ; they deposit relatively quickly with a lifetime of less than 2 days. (Robert et al., 2003)

Atmospheric PM with aerodynamic diameter $<10\mu\text{m}$ (PM_{10}) or $<2.5\mu\text{m}$ ($\text{PM}_{2.5}$) are of considerable concern for public health (NEPC, 1998; Schwartz et al., 1996; Beckett et al., 1998; Borja-Aburto et al., 1998; Prajapati and Tripathi, 2008a-d; Maher and Matzkaa, 1999; Pope III, 2000; Rai, 2013). The ultrafine particles with typical dimension of nanometre-length scale are most hazardous (Wahlin et al., 2006) as it causes several life threatening diseases of varying dimension (Wahlin et al., 2006; Brook et al., 2004; Veranth et al., 2003; Samet et al., 2000a). Ultrafine particles are responsible for the bulk of adverse health effects associated with particles in ambient air (Penttinen et al., 2001; Rai, 2013). Ultrafine PM is more potent than fine or coarse PM towards inducing cellular damage (Le et al., 2002) and also passes rapidly into the circulatory system (Nemmar et al., 2001).

Dust pollution in the atmosphere, particularly of pollutant particles below 10 μm (PM_{10}), is of current concern worldwide due to adverse health effects associated with their inhalation (Calderon-Garciduenas et al., 2004; Morris et al., 1995; Oberdorster, 2000; Pope et al., 2004; Faiz et al., 2009; Rai, 2011a; Rai, 2011b; Rai, 2013). Moreover, PM in dust is thought to be the most harmful pollution component widely present in the environment (Bealey et al., 2007; Rai, 2013). In addition, air-borne particle pollution also causes a decrease in air visibility and abnormal changes in vegetation (Jiqun et al., 1995).

4 Health Impacts of PM Pollution

PM is associated with many adverse human health impacts (Jahn et al., 2011; Rohr and Wyzga, 2012; Taner et al., 2013; Hicken et al., 2014; Pascal et al., 2014; Yadava et al., 2014; Rai and Pana, 2014; Rai et al., 2014; Kim et al., 2015; Yang et al., 2015). PM vehicular emissions, notably in the ultrafine fraction, have been specifically associated with endpoints such as oxidative stress and mitochondrial damage (Li et al., 2003), lipid peroxidation (Pereira et al., 2007), up regulation of genes relevant to vascular inflammation (Gong et al., 2007), and early atherosclerosis and oxidative stress (Araujo et al., 2008). Progression of atherosclerosis has also been reported due to exposure of PM pollution (Suwa et al., 2002). Table 1 lists the global researches on adverse health impacts of PM pollution.

Table 1 List of adverse impacts of PM demonstrated through global researches.

Particulate Matter	Health Impacts	References
Effect of Particulate Pollutants on Human Health		
<p>Ultrafine Particles and Fine Particle Suspended Particulate Matter (Spm)</p>	<p>Ultrafine particles induce vascular and systemic inflammation, oxidative stress, cellular damage, mitochondrial damage, lipid per oxidation and early atherosclerosis. Fine particle may provoke alveolar inflammation, resulting in the release of harmful cytokines and increased blood coagulability. Its effect attributed to mild eye irritation, mortality and respiratory disorder such as nose block, sneezing, cough and hyperacidity. It also affects infant birth weight and mortality causing sudden infant death syndrome. Ambient Particulate matter exposure can be associated with specific physiologic endpoints including reduced lung function causing lung inflammation and injury increased blood plasma viscosity affecting vascular tone and endothelial function reduced heart rate variability increased circulating markers of inflammation mild hypoxemia or decline in blood oxygen saturation.</p> <p>Increased symptoms of obstructive airway disease, such as chronic cough, bronchitis, and chest illness PM is also associated with decreases in DNA methylation in NOS2A, a gene directly responsible for production of nitric oxide, an important player in both respiratory and cardiovascular diseases.</p> <p>Elevated concentration of PM_{2.5} exposure is strongly associated with myocardial infarction (MI), Ischemic heart disease, dysrhythmias, heart failure, cardiac arrest, increased carotid intima media thickness (CIMT) a measure of subclinical atherosclerosis. Short term exposure to diesel exhaust mainly consisting of PM_{2.5} has an acute inflammatory effect resulting in marked neutrophilia, activation of mast cells and neutrophils and the production of cytokines and chemokine associated with neutrophil accumulation and activation.</p> <p>PM₁₀ exposure is associated with increased ischemic heart diseases among the elderly population and with higher risk of myocardial infarction. Increased concentration of dust particulates in the air contribute human health hazards involving acute respiratory disorders such as sinusitis, bronchitis, asthma and allergy and damage to the defensive functions of alveolar macrophages leading to increase respiratory infections.</p>	<p>Seaton et al., 1995; Driscoll et al., 1997b; Donaldson et al., 2000; Stone et al., 2000; Penttinen et al., 2001; Le et al., 2002; Utell et al., 2002; Li et al., 2003; Grahame and Schlesinger., 2007; Gong et al., 2007; Pereira et al., 2007; Araujo et al., 2008; Ayres et al., 2008.</p> <p>Lave and sekin., 1977; Lipfert., 1978; Wang et al., 1997; Woodruff et al., 1997; David., 2003; Sirajuddin et al., 2010. Dockery et al., 1989; Portney et al., 1990; Schwartz et al., 1993; Dockery et al., 1996; Peters et al., 1997; Hoek et al., 1998; Peters et al., 1999; Pope et al., 1999; Salvi et al., 1999, 2000; Gold et al., 2000; Tan et al., 2000; Peden., 2001; Peters et al., 2001; Vincent et al., 2001; Brook et al., 2002; Kodavanti et al., 2002; Ghio et al., 2003; Demeo et al., 2004; Liao et al., 2004; Pope et al., 2004; Schins et al., 2004; Ghio et al., 2000, 2004; Gong et al., 2005; Park et al., 2005; Schwartz et al., 2005; Rucker et al., 2006; Ulrich et al., 2012. Tarantini et al., 2009; Breton et al., 2011a. Liao et al., 1999; Pope et al., 1999; Peter et al., 2001; Pope et al., 2003; Kunzli et al., 2005; Pope et al., 2006; Samet and Krewski., 2007; Karr et al., 2007. Salvi et al., 2000; Frampton., 2001; Stenfors et al., 2004; Schwartz and Dockery, 1992 a,b; Schwartz and Morris 1994, 1995; Schwartz and Zanobetti 2005; Jahn et al., 2011; Rohr and Wyzga, 2012; Taner et al., 2013; Hickenet al., 2014; Pascal et al., 2014; Yadava et al., 2014; Rai and Pana, 2014; Rai et al., 2014; Kim et al., 2015; Yang et al., 2015</p>
<p>Particulate Matter (PM_{2.5}, PM₁₀)</p>		
<p>Heavy Metals In Dust Particulates</p>	<p>Presence of heavy metals in air borne particulates causes protein denaturation resulting in malfunction or death of cell. It also causes a number of health effects such as cancer, neurotoxicity, immunotoxicity and cardio-toxicity, leading to increased morbidity or mortality in community</p>	<p>Beg., 1999; Al-Hurban and Al-Ostad 2009 Dockery et al., 1993; Pope et al., 1995; USEPA, 1996; Silbergeld., 1995, 1996</p>

Inhalation exposure studies have shown that short term exposure to diesel exhaust has an acute inflammatory effect on normal human air ways resulting in marked neutrophilia, activation of mast cells and neutrophils and the production of cytokines and chemokine associated with neutrophil accumulation and activation (Salvi et al., 2000; Stenfors et al., 2004; Frampton, 2001). Epidemiologic studies conducted in different parts of the world have demonstrated an important association between ambient levels of motor vehicle traffic emissions and increased symptoms of asthma and rhinitis (Rai, 2013). Additionally, recent human and animal laboratory-based studies have shown that particulate toxic pollutants, and in particular diesel exhaust particles (DEP), can enhance allergic inflammation and induce the development of allergic immune responses (Salvi et al., 2000; Stenfors et al., 2004; Frampton, 2001).

Diesel exhaust-exposed workers have been shown to have an increased risk of lung cancer (Nielsen et al., 1996a; Nielsen et al., 1996b; Scheepers et al., 2002). Methods for the assessment of exposures to diesel exhaust were evaluated by comparing underground workers (drivers of diesel-powered excavators) at an oil shale mine in Estonia with surface workers and it was observed that underground miners were also occupationally exposed to benzene and polycyclic aromatic hydrocarbons, as indicated by excretion of urinary metabolites of benzene and pyrene and increased O⁶-alkylguanine DNA adducts were detected in the white blood cells of underground workers, suggesting higher exposure to nitroso-compounds (Scheepers et al., 2002). Diesel exhaust consists of a complex mixture of particulates which contain known genotoxicants, one of which is benzene. Muzyka et al., (1998) indicated significant differences in 5-aminolevulinic acid (ALA) synthesis and heme formation between the exposed workers to PM containing benzene when compared to the non-exposed individuals.

Several air pollutants, comprising PM e.g. benzo[*a*]pyrene among other carcinogenic polycyclic aromatic hydrocarbons (c-PAHs) and diesel engine exhaust emissions, are classified as class-1 carcinogen whereas gasoline engine exhaust emissions, carbon black, and a number of PAHs are classified as group 2B for their carcinogenicity by the International Agency for Research on Cancer (IARC) (IARC, 1983, 1987, 1989; Pedersen et al., 2006).

Chen et al. (2004) reported that ambient air pollution had acute and chronic effects on mortality, morbidity, hospital admissions, clinical symptoms, lung function changes, etc. in China. Schoket (1999), in his exhaustive study found that in Silesia, Poland, and Northern Bohemia, Czech Republic, where coal-based industry and domestic heating are the major sources of PAHs, significant differences have been observed in white blood cell DNA adducts and cytogenetic biomarkers between environmentally exposed and rural control populations, and significant seasonal variations of DNA damage have been detected. Further, Schoket (1999) found that in Copenhagen, Athens, Genoa and Cairo, Bus drivers, traffic policemen and local residents have been involved in biomarker studies and differences have been measured in the level of DNA damage of urban and rural populations.

Traffic originating from increased number of vehicles may cause multiple adverse health effects including asthma and allergic diseases, cardiac effects, respiratory symptoms, reduced lung function growth, adverse reproductive outcomes, premature mortality, and lung cancer (White et al., 2005; Samet, 2007). The occurrence of dramatic Micro Satellite alterations in 3p chromosome multiple critical regions could be a crucial underlying mechanism, which exacerbated the lung toxicity in air pollution PM-exposed target L132 cells (Saint-Georges et al., 2009).

Traffic policemen are heavily exposed to vehicle exhausts during traffic control and other outdoor activities (Carere et al., 2002) which may lead to increased incidences of sister chromatid exchanges (Anwar, 1994; Chandrasekaran et al., 1996; Zhao et al., 1998; Carere et al., 2002) and micronuclei and chromosomal aberrations (Anwar, 1994; Chandrasekaran et al., 1996; Zhao et al., 1998). Carere et al. (2002) while

investigating blood cells and DNA of Rome traffic policemen and office workers indicated that exposure to moderate air pollution levels does not result in a detectable increase of genetic damage in blood cells.

Muzyka et al. (1998) demonstrated the data on determination of 5-aminolevulinic acid (ALA) synthesis and heme formation in lymphocytes from a group of 45 bus-garage workers and an analogous data from a group of 25 unexposed subjects and the outcome indicated significant differences in ALA synthesis and heme formation between the exposed workers when compared to the non-exposed individuals. Also, concentration of porphyrin associated with DNA was significantly increased (Muzyka et al., 1998). Aforesaid findings of Muzyka et al. (1998) reflected that metabolites of heme synthesis in lymphocytes could be a useful biomonitoring index for the determination of a sensitive subgroup of workers who undergo the higher risk of cancer development.

Various studies showed PM exposure, associated with elevated levels of c-reactive protein, a marker of systemic inflammation that may be important and independent predictor of cardiovascular diseases. For example, a recent study reported associations between CRP and interleukin (IL)-6 with PM in subjects with coronary artery disease (Delfino et al., 2008). Inflammatory lung injury, bone marrow and blood cell responses, enhanced human alveolar macrophage production of proinflammatory cytokines, elevated blood plasma viscosity (Ghio et al., 2003), endothelial dysfunction and brachial artery vasoconstriction and triggering of myocardial infarction. Polichetti et al. (2009) extensively reviewed the impact of PM on cardiovascular system. Particulate matter is also linked with psychosocial stress and high blood pressure (Hicken et al., 2014).

Suspended particulate matter (SPM) is of the greatest concern as it contributes 50% to total air pollution (Fuller, 1974) and causes respiratory disorders in human beings on prolonged exposure (Freer-Smith et al., 2004) as it includes all airborne particles in the size range of 0.5 μ to 100 μ . Its effects attributed to mild eye irritation, mortality (David, 1995). Sirajuddin and Ravichandran (2010) also studied SPM related respiratory disorders such as nose block, sneezing, cough and hyperacidity in Tiruchirappalli, India. Bhattacharjee et al. (2010) noted that PM_S<4.6 μ m and PM_S<1.1 μ m are hazardous to human health due to its capacity to be inhaled into the bronchial region and getting deposited in the alveolar region. Air quality monitoring at Kolkata, India (for SPM, RPM, NO_x, SO₂, CO and Pb levels) indicated that they are currently at levels dangerous to human health (Ghose et al., 2005).

In literatures, it is well documented that particulate pollution causes adverse health impact particularly in the size range of less than 10 μ m (Curtis et al., 2006; Lipmann, 2007; Zeger et al., 2008; Mitchell et al., 2010). PM pollutants are associated with adverse effects on respiratory system (Schwartz, 1996; Pope et al., 2002; Knutsen et al., 2004; Knox, 2006; Maher et al., 2008; Hansard et al., 2011). If these particulates of size lower than 10 μ m causes inflammation and diminished pulmonary function can be unavoidable (Knutsen et al., 2004; Seaton et al., 1995; Maher et al., 2008). Further, PM with aerodynamic diameter smaller than 2.5 μ m (PM_{2.5}) have even more deleterious health impacts because when inhaled they penetrate deeper than PM₁₀ and can reach lungs alveola (Rizzio et al., 1999; Harrison and Yin, 2000; Wichmann and Peters, 2000; Saragnese et al., 2011). Links with lung cancer (Pope et al., 2002; Beeson et al., 1998) and increased cardiovascular mortality rates (Schwartz, 1996) have also been established. Lung diseases due to PM may be attributed to presence of inflammatory cells in the airways including neutrophils (PMN), eosinophils and monocytes (Mo), and increased numbers of alveolar macrophages (AM) (Becker et al., 2002).

Children are particularly sensitive to air pollution as their lungs as well as immune systems are not completely developed when compared to adults one (Bateson and Schwartz, 2007). Further, children are considered more vulnerable to the adverse effects of air pollution than adults due to physiological differences related to their body size, growth, development and immaturity of organs and body functions (Pedersen et al., 2006). The air intake of a resting infant of an age less than 1 year is twice that for an adult (Pedersen et al.,

2006). Children also exhibit a higher intake of food and water per kilogram body weights (Pedersen et al., 2006). Bener et al. (2007) determined the impact of asthma and air pollution on school attendance of primary school children 6 to 12 years of age in Qatar and their finding showed that air pollution has an impact on asthma, which results in significant school absenteeism. Wilhelm et al. (2005) assessed the impact of Pb and Cs child-mother pairs in North Rhine Westphalia, Germany and through regression analysis showed that Pb levels in ambient air were associated with Pb in the blood of children and mothers.

Urinary 1-hydroxypyrene (1-OHP), a major metabolite of pyrene may act as a biomarker of exposure to PAHs (Bouchard and Viau, 1999; Jonganeelen et al., 1990; Cavanagh et al., 2007). Cavanagh et al. (2007) found that 1-hydroxypyrene (1-OHP) was found in elevated concentrations in school children immediately after heavy exposure of particulate pollution particularly PAH.

Global records showed PM below size 2.5 μm causes 3% of mortality from cardio-pulmonary disease; 5% of mortality from cancer of the trachea, bronchus and lung; and 10% of mortality from acute respiratory infections in children under five (Cohen et al., 2005; Maher, 2009). It is well established through literatures that air pollution with PM in children results in detectable effects indicated by a number of biomarkers of exposure and early effects (Pedersen et al., 2006). Aforesaid hypothesis was tested through a family pilot study which was conducted in the Czech Republic through fluorescence in situ hybridization (FISH) and it was concluded that micronuclei (MN) is a valuable and sensitive biomarker for early biological effect in children and adults living in two different areas characterised with significant exposure differences in c-PAHs concentrations during winter (Pedersen et al., 2006).

PM pollution is intimately linked with cardiovascular morbidity and mortality (Brook et al., 2004; Adar and Kaufman, 2007). Several researches demonstrated that approximately 50% of the adult mortality from air pollution or 22,000 deaths per year to traffic sources in Austria, France, and Switzerland (Kunzli et al., 2000; Adar and Kaufman, 2007). Adar and Kaufman (2007) reviewed the epidemiological evidence regarding the impact of traffic-related pollution on cardiovascular diseases and future directions being used in ongoing epidemiological studies to identify the cardiovascular health impacts of traffic.

Further, several studies have shown an association between exposure to ambient PM and hospital admissions for cardiac and respiratory causes (Analitis et al., 2006; Atkinson et al., 2001; LeTertre et al., 2002; Migliaretti et al., 2005; Sun et al., 2006; Ciencewicky and Jaspers, 2007). Moreover, it has been shown that hospital visits for asthma associated with exposure to PM are more prevalent in children and the elderly (Migliaretti et al., 2005; Sun et al., 2006; Ciencewicky and Jaspers, 2007) and that exposure to PM is associated with decreased lung function in children (Moshhammer et al., 2006; Ciencewicky and Jaspers, 2007).

In general, PM comprises polycyclic aromatic hydrocarbons (PAH) and volatile organic compounds (VOCs), which may have deleterious impact on human health. Air pollutants particularly VOCs have been reported from waste treatment and disposal facilities and may be of concern to public health (Hamoda, 2006). Associations between urban pollutants and respiratory and cardiovascular problems, and still a greater incidence of certain cancer types have already been mentioned in literatures (Lester and Seskin, 1970; Saldiva et al., 2002; Lin et al., 2003).

Ciencewicky and Jaspers (2007) reviewed the association between and effect of air pollutants and respiratory viral infections, as well as potential mechanisms associated with these effects. PM, especially traffic-related airborne particles, contains a large number of genotoxic/mutagenic chemical substances, which can cause DNA damage and promote malignant neoplasms (Valavanidis et al., 2008). The genotoxicity of PM was extensively studied with the *Salmonella typhimurium* assay (Ames test) by various research groups and reviewed by Claxton et al. (2004) and summarized elsewhere (Valavanidis et al., 2008). Combustion emissions account for over half of the fine particle ($\text{PM}_{2.5}$) air pollution and most of the primary particulate organic matter

(Lewtas, 2007). Lewtas (2007) in his extensive review on the impacts of air pollution on human beings mentioned that both short- and long-term exposures to combustion emissions and ambient fine particulate air pollution have been associated with measures of genetic damage. Moreover, long-term epidemiologic studies have reported an increased risk of all causes of mortality, cardiopulmonary mortality, and lung cancer mortality associated with increasing exposures to air pollution. Adverse reproductive effects (e.g., risk for low birth weight) have also recently been reported in Eastern Europe and North America (Lewtas, 2007). The use of a non-smoking female population in Silesia with a limited age range and a homogeneous occupation indicated that environmental exposure to air pollution may be responsible for genetic damage (Michalska et al., 1999).

Constituents of PM pollutants emanating from vehicular emissions have been demonstrated to cause genotoxicological impact on plants as well as humans. For instance, although PAHs are relatively chemically inert compounds, however, through metabolic activation to electrophilic derivatives (e.g. diolepoxides, quinones, conjugated hydroxyalkyl derivatives) these are capable of covalent interaction with nucleophilic centres of DNA (Schoket, 1999). These adducts of PAH to DNA cause base pair substitutions, frameshift mutations, deletions, S-phase arrest, strand breakage and a variety of chromosomal alterations (Schoket, 1999). Two main cell types are likely to interact with inhaled particles i.e. Alveolar macrophages (AM) and airway epithelial cells in response to PM pollution (Becker et al., 2005). Dagher et al. (2006) demonstrated that in vitro short-term exposure to PM_{2.5} induced oxidative stress and inflammation in human lung epithelial cells (L132) and emphasized the need of such researches to reveal the mechanism of adverse health impact imposed by PM. PM induces the activity of NF-κB manifold which is a transcription factor that can induce gene transcription in a variety of pro inflammatory cytokines, enzymes that generate mediators of inflammation and immune receptors (Yang and Omaye, 2009). Researches investigated the biological effects of PM_{2.5} on human lung epithelial cell line A549 (Calcabrini et al., 2004). Billet et al. (2008) assessed the genotoxic potential of PAH containing PM on human lung epithelial A549 cells. Fine PM are also reported to induce sister chromatid exchange in human tracheal epithelial cells (Hornberg et al., 1998).

Statistically significant increase was established in the frequency of chromosomal aberrations in peripheral blood lymphocytes from the exposed population towards heavy metals and dioxins/furans, hence, chromosomal aberrations in human peripheral blood lymphocytes may be generally used as a biomarker (Huttner et al., 1999). Hellman et al. (1999) demonstrated that increased levels of radon in indoor air (>200 Bq/m³) were found to be associated with an increased level of DNA damage in peripheral lymphocytes.

Reports indicated that constituents of inhaled PM may trigger a proinflammatory response in nervous tissue that could contribute to the pathophysiology of neurodegenerative diseases (Campbell et al., 2006). PM affects sensory and neural pathways through activation of capsaicin-sensitive vanilloid (VRI) irritant receptors (Veronesi and Oortgiesen, 2001).

Claxton and Woodall Jr. (2007) for the first time extensively reviewed the mutagenicity and carcinogenicity of air pollutants. Coronas et al. (2009) investigated genotoxic effects on people exposed to oil refinery in southern Brazil and the mutagenic activity of airborne PM. Individuals who environmentally exposed to heavy metals (mercury and lead), organic (styrene, formaldehyde, phenol and benzo[*a*]pyrene) and inorganic (sulfur and nitrogen oxides, hydrogen and ammonium fluorides) volatile substances may have high rate of chromosomal aberrations and sister-chromatid exchanges (Lazutka et al., 1999).

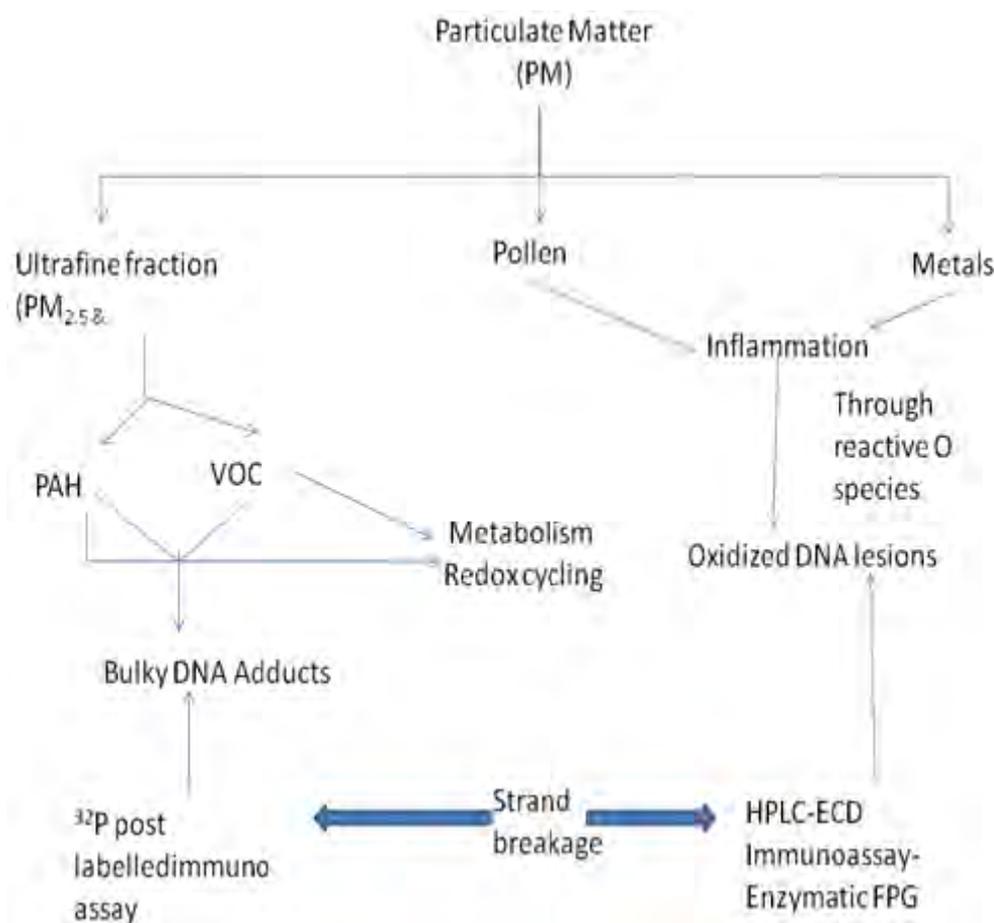


Fig. 1 Interaction of PM and its constituents with DNA leading to DNA adduct formation.

In an integrated way, PAH and VOCs lead to the formation of bulky DNA adducts (Moller et al., 2008). Fig. 1 represents the mechanism through which PM leads to formation of DNA adducts. The genotoxic effect of pollutants on the ecosystem, including the build-up of resistant species, is also of considerable concern (Ma et al., 1994; Grant, 1998). The potential genotoxic effects on human health by such vapour phase chemicals include malignant cell formation, the accumulation of heritable abnormal genes within the population, heart disease, aging and cataracts (Grant, 1998). The effects of toxic compounds, and the subsequent genotoxic effects on plants, are of particular importance as plants comprise a large portion of our biosphere and constitute a vital link in the food chain (Grant, 1998; Rajput and Agrawal, 2005). Estimating air genotoxicity is therefore crucial to evaluating risk to the environment and public health. PAH which are deleterious components of particulates results in risk of breast cancer (Gammon and Santella, 2008). Further, PAH causes DNA damage T- and B-lymphocytes and granulocytes (specifically single strand DNA breakage) in individuals exposed to exhaust fumes (Sul et al., 2003).

As stated earlier, PMs also consist of heavy metals, carbon core and pollen, which further interacts with gaseous pollutant resulting in the formation of DNA lesions. PM (specifically PM_{10} and $PM_{2.5}$) contain several deleterious metals. The problem of metals is very much prevalent in atmosphere particularly those in Asian countries which was extensively reviewed by Fang et al. (2005). Pb may have several genetical impacts in human beings e.g. fidelity of DNA synthesis, mutation, chromosome aberrations, cancer and birth defects

(Johnson, 1998). It reacts or complexes with many bio molecules and adversely affects the reproductive, nervous, gastrointestinal, immune, renal, cardiovascular, skeletal, muscular and hematopoietic systems as well as developmental processes (Johnson, 1998).

Several researches have shown that the size of the airborne particles and their surface area determine the potential to elicit inflammatory injury, oxidative damage, and other biological effects (Valavanidis et al., 2008).

There has been considerable concern on the pulmonary effects of particulates less than 2.5 μm ($\text{PM}_{2.5}$) or 10 μm (PM_{10}), as they can reach the alveoli and translocate to the circulation, whereas particles of larger size deposit mainly in the upper airways and can be cleared by the mucociliary system (Oberdorster et al., 2005; Moller et al., 2008). In the recent past, many studies highlighted the role of ambient airborne PM as an important environmental pollutant for many different cardiopulmonary diseases and lung cancer (Valavanidis et al., 2008). Further, it has increasingly being realized that generation of reactive oxygen species (ROS) and oxidative stress is an important toxicological mechanism of particle induced lung cancer (Knaapen et al., 2004; Risom et al., 2005). The fraction of PM contains a number of constituents that may increase the generation of ROS by a variety of reactions such as transition metal catalyses, metabolism, redoxcycling of quinones, and inflammation. PM, thus, can generate oxidative damage to DNA, including guanine oxidation, which is mutagenic (Kasai, 1997; Moller et al., 2008). The oxidative stress mediated by PM and resulting DNA damage may originate from generation of ROS from the surface of particles, soluble compounds such as transition metals or organic compounds, altered function of mitochondria or NADPH-oxidase, and activation of inflammatory cells capable of generating ROS and reactive nitrogen species (Risom et al., 2005). Production of reactive oxygen species (ROS) and the secretion of inflammatory cytokines could interact by inducing cell death by apoptosis (Shukla et al., 2000; Haddad, 2004; Hetland et al., 2004; Dagher et al., 2006) (Fig. 2). Interaction between oxidative stress by products and certain genes within our population may modulate the expression of specific chronic diseases (Yang and Omaye, 2009).

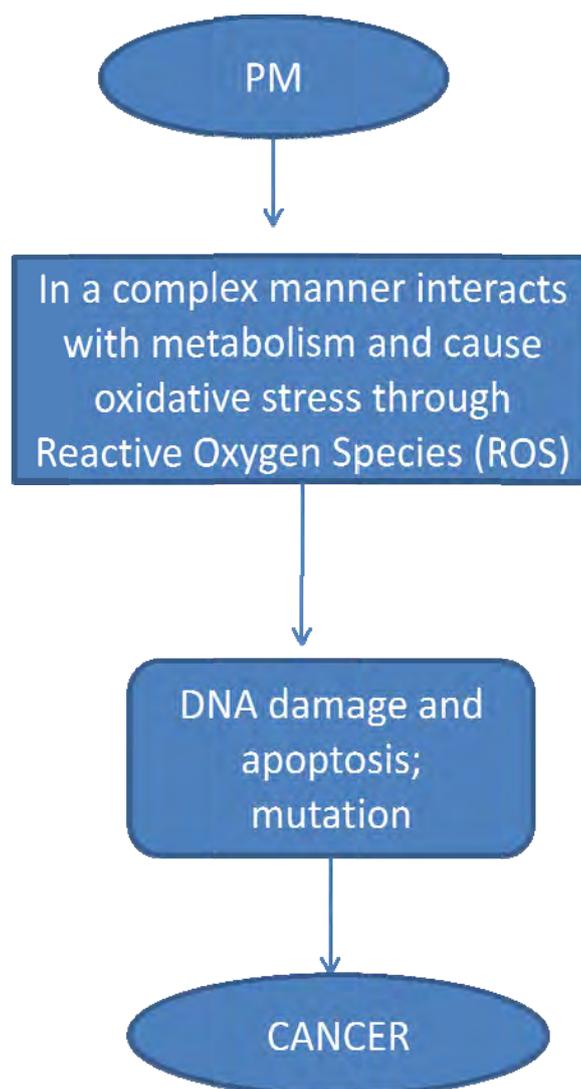


Fig. 2 Impact of PM on cellular machinery leading to apoptosis.

Epidemiological studies have observed positive associations between levels of PM and the incidence of mortality, including those involving cardiovascular and respiratory conditions (Dominici et al., 2003; Katsouyanni et al., 2001; Pope et al., 2002; Samet et al., 2000a; Ciencewicki and Jaspers, 2007). Likewise, numerous epidemiologic studies highlighted the health implication of fine particles with aerodynamic diameter smaller than 10 μm (Kunzli et al., 2000; Katsouyanni et al., 2001; Pandey et al., 2005; Pandey et al., 2006; Pope et al., 2002; Peng et al., 2005; Prajapati et al., 2006). Epidemiologic findings suggest that short term PM exposure can trigger acute or terminal health events whereas long term PM exposure however could promote life shortening chronic illness. PM was also associated with decreases in DNA methylation in NOS2A, a gene directly responsible for production of nitric oxide, an important player in both respiratory and cardiovascular diseases (Tarantini et al., 2009; Breton et al., 2011a). A study in china reported a significant adverse relationship between birth weight and maternal exposure to total SPM (Wang et al., 1997) suggesting that both pre and post natal exposure to ambient PM affects infant birth weight and mortality. Other studies have also suggested a significant link between PM exposure and excess infant mortality (Lave and Sekin, 1977; Lipfert, 1978; Woodruff et al., 1997). Woodruff et al. (1997) also studied the link between PM₁₀ exposures to an

increased incidence of sudden infant death syndrome. Additional evidence suggested that PM exposure over time can alter lung function, lung tissue and structure, air way responsiveness and respiratory defence mechanisms and can increase susceptibility to respiratory infection and damage respiratory cells (EPA, 1996; EPA, 1997). The association of ambient PM with mortality and cardiovascular outcome has been well established in studies of short term exposure (Levy et al., 2000; Samet et al., 2000a, 2000b; Zanobetti et al., 2000). PM_{2.5} exposure increases ischemic cardiovascular events and promotes atherosclerosis (Barajas et al., 2008; Samet and Krewski, 2007; Pope et al., 2006; Pope et al., 2003). Karr et al. (2007) also suggested the infant bronchiolitis as an adverse effect of PM_{2.5} exposure. Long term PM exposure has been associated with sub clinical chronic inflammatory lung injury and sub clinical atherosclerosis and also associated with the incidence of cardiovascular diseases and death among post-menopausal women (Miller et al., 2007).

Christophe et al. (1997) have reported some metal in air borne dust particulates, having toxic effect on human health. Heavy metals associated with respirable dust particles having size below 10 µm (PM₁₀) in urban air can penetrate deep into the lungs and retained their causing many health problems. Heavy metals are toxic even at low concentration in air. After inhaled, they form complexes or legends with vital protein molecules, denaturing them and resulting in malfunction or death of cell (USEPA, 1996). Heavy metal are now correlated with a number of health effects such as cancer, neurotoxicity, immune-toxicity and cardio-toxicity, leading to increased morbidity or mortality in community (Dockery et al., 1993; Pope et al., 1995; Silbergeld, 1995,1996).

Beg (1999) performed a study on the toxic response of urban dust leading to the dispersion of PM in the atmosphere at a shocking high concentration. Increased concentration of this inhalable PM in the air due to dust storms contribute human health hazards involving acute respiratory disorders such as sinusitis, bronchitis, asthma and allergy and damage to the defensive functions of alveolar macrophages leading to increase respiratory infections. Research has also found that exposure to ambient PM concentration results in increased blood level of endothelins which can affect vascular tone and endothelial function (Vincent et al., 2001; Brook et al., 2002).

In the early and mid-1990's studies by Schwartz and his colleagues (Schwartz and Dockery, 1992a, 1992b; Schwartz and Morris, 1994, 1995) found increase in ischemic heart diseases among the elderly population associated with PM₁₀ exposure. Schwartz and Zanobetti (2005) also observed that PM_{2.5} and PM₁₀ were strongly associated with higher risk of myocardial inflammation. Al-Hurban and Al-Ostad (2009) conducted a study on the textural and mineralogical characteristics of the dust fall out and their effect on human health in Kuwait city. It was found that the dust comprises mostly high concentration of calcite and quartz with grain size ranging from 1-25 µm. Inhalation of calcite particles may cause coughing, sneezing and nasal irritation and in cases of chronic exposure to excessive oral doses of calcite may produce alkalosis and hypercalcemia. Quartz can have potentially serious respiratory effects following long term exposure as it is declared as carcinogenic.

Experimental data shows that redox active PM components (that are especially enriched in ultrafine PM<0.1 µm) lead to the production of reactive oxygen species (ROS) in various cells in the lungs, blood and vascular tissues. This is followed by oxidative stress, which can then lead to increased airway and systemic inflammation, and adverse cardiovascular responses when antioxidant defences are overwhelmed (Ayres et al., 2008; Utell et al., 2002). Oxidative stress is a biochemical imbalance in which production of ROS exceeds the natural antioxidant capacity. This imbalance can occur in the body following exposure to pro-oxidant air pollutants. Oxidative stress may play a central role in the respiratory and cardiovascular effects of air pollution through its immune modulating effects and its ability to initiate the inflammatory process and thrombogenic activity.

5 Management or Reduction of Health Impacts Due To PM

Present part of this review discusses briefly the management options associated with PM pollution. The persistence of air pollutants and the rapid rise in vehicle travel in recent decades have raised concerns within the planning sector (Stone et al., 2007). Harrison and Yin (2000) in their review supported that single major or trace component of the PM is responsible for the adverse health effects. Costa (2004) described the interaction among PM constituents in the light of scientific and regulatory agendas. Ugwuanyi and Obi (2002) used the environmental impact matrices of the patients versus diseases and found that pollution is already affecting the quality of life and productivity of the people (particularly farmers) in Benue state of Nigeria. Due to extreme health impacts of PM, proper caution should be taken in setting its air quality standards (McClellan, 2002).

Further, comprehensive understanding about the health consequences of traffic exposures is necessary for formulating mitigation policies (Samet, 2007). Wilson et al. (2005) emphasized the need of intra-urban assessment of exposure to PM pollution. Epidemiological meta studies for the health impacts of fine PM may be used to predict the number of premature deaths and some morbidity impacts from prevailing ambient concentrations (Pearce and Crowards, 1996).

Air quality modelling may be a useful approach for the management of air quality. Tchepel et al. (2007) attempted modelling approach to estimate population exposure to benzene through inhalation and suggested that aforesaid model may be used in combination with human biomonitoring in order to select who and where should monitoring be done, as well as for interpretation and extrapolation of biomonitoring results. Stocker (2000) suggested a methodology for evaluating air quality impacts on meteorological model to simulate the meteorological conditions which correlate with prescribed fire and wildfire activity in Colorado. According to Stocker (2000) meteorological fields are input into an air quality model which simulates transport and secondary aerosol formation for certain pollutants. Also, air quality indices may be helpful to assess the human health implications (Dimitriou et al., 2013).

Specific model systems may be developed in order to estimate the concentration of air pollutants to which humans are exposed (Borrego et al., 2009). The operational air quality forecasting modelling system is composed by the chemistry-transport model CHIMERE, forced by the MM5 meteorological fields (Monteiro et al., 2005; Borrego et al., 2009). Oxidative and immune effects of PM pollution have been demonstrated in several in vitro and animal models with in European projects (Sandstrom et al., 2005). Abbas et al. (2009) extensively studied PM induced gene expression of volatile organic carbon and polyaromatic hydrocarbons metabolizing enzymes in an in vitro coculture lung model which may be powerful tool to identify the mechanisms by which air pollution PM induced adverse health effects.

Vegetation used to filter the PM concentration and consequently provide health benefits (Tiwary et al., 2007). Biomonitoring with screening of potent plants may be emphasized in the regulation of air quality. Wolterbeek and Verburg (2004) observed correlations between moss metal concentrations and epidemiological data on health and mortality rates in The Netherlands and their data suggested that correlation studies between biomonitoring data on metal air pollution and (epidemiological) health data may prove valuable in turning attention to specific metal-health issues and in directing further study into possible dose-response mechanisms in air-associated metal epidemiology. Okona-Mensah et al. (2005) described in his review regarding novel biomonitoring approach to evaluate exposure, uptake and the role of high potency PAHs in air pollution related lung cancer.

Education and awareness of people towards the adverse health impacts of PM pollution may be very useful in its management. Dorevitch et al. (2008) assessed the efficacy of an outdoor air pollution programme in a community at risk for Asthma and their findings after exhaustive survey recommended that air quality education efforts should be further developed, evaluated, and promoted for the general public, for people with

underlying cardiopulmonary disease, and given the documented health disparities within the general population, for low-income and minority communities.

El-Fadel and Massoud (2000) also emphasized the need of health based economic assessment originating due to PM pollution in urban areas. In relation to socio-economic and related components, the benefits and costs associated with Canada-Wide Standards for PM is highly uncertain and controversial (Adamowicz et al., 2004). Likewise, similar recommendation of costs and benefit analysis of remedial measures including an assessment of the impact of urban air quality on human health was submitted by Mitchell et al. (2000). Epidemiology-based exposure-response functions were used in order to assess economy of health impacts in Shanghai, China and it was estimated that the total economic cost of health impacts due to PM pollution in urban areas of Shanghai in 2001 was approximately 625.40 million US dollars (Kan and Chen, 2004). The health benefits of pollution reduction are compared with the investment costs for the new strategies in Shanghai, China and the result showed benefit-to-cost ratio is in the range of 1–5 for the power-sector initiative and 2–15 for the industrial-sector initiative (Li et al., 2004). This study by Li et al. (2004) provides economic grounds for supporting investments in air pollution control in developing cities of Asia.

Airborne PMs are associated with increased mortality and estimates have been used to forecast the impact on life expectancy (Coyle et al., 2003). Sultan (2007) emphasized on the role buildings, their ventilation and filtration to evaluate the health risk from outdoor PM pollution. Modern tools like satellite remote sensing may be applied in monitoring of PM (Gupta et al., 2006).

Therefore, in the light of abovementioned discussion, a well defined particulate pollution control policy structure is the need of the hour in view of their adverse impacts on flora and fauna, including human beings (Rai, 2013). Craig et al. (2008) made a guidance document in order to reflect critical science and policy aspects of air quality risk management including i) health effects, ii) air quality emissions, measurement and modelling, iii) air quality management interventions, and iv) clean air policy challenges and opportunities. It was based on findings of five annual meetings of the NERAM (Network for Environmental Risk Assessment and Management) International Colloquium Series on Air Quality Management (2001–2006) as well as researches of international repute (Rai, 2013).

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