Abstract

Particulate matter (PM) is a complex mixture of solid and liquid particles from various sources. Fine PM or PM\(_{2.5}\) is defined as a mass with a size of less than 2.5 μm in aerodynamic diameter and has a large contribution to the world increasing annually. Iran is a developing country set in the Middle East that is not secured from this pollutant mainly due to its industries, desert dust and the travel of dust from the neighboring countries. Poor air quality caused by PM\(_{2.5}\) can induce multiorgan dysfunction including cardiovascular disease, respiratory impairment, and other adverse effects that lead to morbidity and even death. Since PM\(_{2.5}\) is a risk factor for health problems, the comprehension of the detailed molecular mechanisms of PM\(_{2.5}\) including oxidative stress and inflammation would be beneficial. The aim of this review is gathering information from epidemiological studies about the health effects of this pollutant in Iran for the sake of a healthy environment and proposing solutions which can be applied to every country that is concerned about the air quality.

Keywords: Particulate Matters; Air Pollution; Health Effects

Introduction

Nowadays, air quality and air pollution are big challenges around the world. The classification of atmospheric particulate matters (PMs) is as follows PM\(_{10}\) (dp<10 μm), fine PM or PM\(_{2.5}\) (dp<2.5 μm), and ultrafine PM (dp<0.1 μm) in aerodynamic diameter [1]. Among the air pollutants, PM\(_{2.5}\) is the most important pollutant deteriorating air quality [2], and it is putative due to its wide range of origins including combustion engines, power plants, industry, home energy consumption, cultivation, smokes from biomass burning, and natural sources like desert dust. Also, PM\(_{2.5}\) is associated with a variety of health disorders (cardiovascular, respiratory, endocrine, cancers, etc.) as mentioned in epidemiological cohort studies [3-5]. The PM\(_{2.5}\) can cause death and have health impaction at very low concentration levels [6-9]. People living in the Middle East are exposed to PM\(_{2.5}\) [10], and Iran is one of the countries of the Middle East encountering a serious environmental problem because of the rapid growth in urban population, industries, desert dust, and deserts in its neighborhood [11]. Although the air quality index (AQI) in Iran differs from the standards of environmental
protection agency [12], everyone agrees on the disaster of PM$_{2.5}$ for Iran and other countries where the AQI ranges between 50-150 for PM$_{2.5}$, which equals a 24-hour average of 12 to 55.4 µg/m$^3$.

The PM$_{2.5}$ is not in accordance with the standards set by World Health Organization (WHO) which shows that public health is in danger in main provinces of Iran [13-15]. The purpose of this study is to evaluate and overview the hazards of PM$_{2.5}$ in Iran and to propose solutions to make better decisions to solve this problem.

**Epidemiology**

The PM$_{2.5}$ is noticed around the world mainly due to its impact on mortality rate and hospital admission; on the report of WHO it was responsible for 3.7 million deaths in 2012 [16, 17]. Atkinson *et al.* [18] suggested that the association of PM$_{2.5}$ with respiratory mortality (1.51%) was larger than the percent reported for cardiovascular mortality (0.84%) and all-age, all-cause mortality ranged from 0.25% to 2.08% with an overall estimate of 1.04% which declared the public health importance of PM$_{2.5}$. However, a larger part of the population is affected by cardiovascular diseases (CVD) than by respiratory diseases.

Not only cardiovascular and respiratory mortalities are attributable to PM$_{2.5}$ [19-21], but also it is a major risk factor for premature deaths globally [4]. However, PMs is the consequence of urbanization. Previous research in California demonstrated that surprisingly the mortality rate due to chronic exposure to PM$_{2.5}$ was higher in rural areas [22].

According to the study conducted in Mashhad (one of the cities with high PM$_{2.5}$ levels in Iran), total mortality rate related to PM$_{2.5}$ was 1.61% (600 cases in a year due to short-term exposure) and results of the relative risks (RR) for the increase in total mortality and hospitalization caused by PM$_{2.5}$ indicated that with every 10 µg/m$^3$ increase in PM$_{2.5}$ concentration, there was 0.3% increase in RR [23].

Also, dust is a powerful source of PM$_{2.5}$ in Iran that is located in the dust belt, so dust can contaminate the atmosphere of cities and change the cloud characteristics and even soil properties [24-28]. The outdoor concentrations of PM$_{2.5}$ are strongly correlated with indoor concentrations of PM$_{2.5}$ in the schools which are built near the main roads in Iran [29]. The PM$_{2.5}$ is one of the pollutants in dust which attenuates the air quality in metropolises of Iran. Hence, vulnerable groups such as children and older adults would be in danger [13]. Two studies conducted in Tabriz revealed that in February, there is the maximum daily mass concentration of total suspended PM$_{2.5}$ (96.6µg/m$^3$) and annual average concentration is 85.3 µg/m$^3$ that mostly comes from natural sources especially soil and Urmia lake bed [15,30,31]. However, a study in Ahvaz (a city in Iran with many dust events) indicated that the average concentration of PM$_{2.5}$ is 69.5µg/m$^3$ and the peak concentration is in May and early July [32]. By evaporation of the water from semi-arid lakes, dust contains PM$_{2.5}$ are created and transported by winds for thousands of kilometers [33,34]. A study claimed that the dust in Iran is similar to the dust around the world in their characteristics [35]. Unfortunately, there was 12 percent reduction in the air quality of Iran from the year 2002 until 2012 [36].

**Mechanism**

Several mechanistic pathways linking air pollution exposure to adverse health outcomes have been described [5]. The inhalation of air pollution can alter the autonomic balance leading to sympathetic activation which can for example cause changes in heart rate or impaired heart rate variability. Secondly, nanoparticles or their constituents may translocate into the circulation leading to direct harmful effects on the cardiovascular system. Thirdly, PM$_{2.5}$ can induce detrimental effects by the impact of oxidative stress and inflammation (Figure-1).

The PM$_{2.5}$ contains metals which can alter antioxidant enzymes, eg, glutathione (GSH) resulting in increasing reactive oxygen species (ROS), lipid peroxidation and redox imbalance. On the other hand, nuclear factor erythroid-2-related factor-2 (Nrf-2) translocation to the nucleus increases due to the ROS produc-
tion and attenuate the activity of antioxidant enzymes by changing their transcription [37]. Also, ROS overproduction occurs by altering electron transport chain and through Haber-Weiss and Fenton reactions in the cell cytosol [31, 38-40]. The ROS formation leads to the damage of DNA and proteins which can be the cause of cancers or apoptosis [41-43]. Inhaled PMs are capable of production of proinflammatory cytokines and adhesion molecules such as tumor necrosis factor (TNFα), interleukin-6 (IL-6). Inflammation leads to activate the ataxia telangiectasia and rad3-related-tumor protein53 (ATR-TP53) axis and induces autophagy. Also, TP53 triggers autophagy in response to DNA damage [44-48]. However, DNA damage induced by PM$_{2.5}$ causes apoptosis, ROS elevation increases AMP-activated protein kinase (AMPK) and mechanistic target of rapamycin (mTOR) inhibition due to the activation of AMPK cause proliferation and inhibition of autophagy [49]. The PM$_{2.5}$-induced IL-8 expression occurs via activation of Toll-like receptor-2 (TLR2) [50]. The ROS increases transformation of procaspase-1 to caspase-1, and caspase-1 increases IL-18. The ROS activates nuclear factor kappa B (NF-κB) and causes insulin resistance and IL-1β promotion. The IL-18 and IL-1β augmentation, transcription, and cleavage in endothelial progenitor cells (EPCs) accelerate EPC depletion [51]. The PM$_{2.5}$ promote more inflammation and proliferation via NF-κB and ROS-mediated signal transducer and activator of transcription-3 (STAT3) activation [52]. Some studies showed that using extracellular signal-regulated kinase-1/2 (ERK1/2) inhibitor and/or protein kinase B (AKT) inhibitor which block ERK/AKT/ NF-κB pathway can reduce the number of adhesion molecules such

**Figure 1.** Mechanism of PM$_{2.5}$ induced autophagy and apoptosis by various pathways in cell.
as intercellular adhesion molecule-1 (ICAM-1) and vascular adhesion molecule-1 (VCAM-1) production due to the oxidative stress [53]. Cao et al. suggested that ROS elevation caused by PM$_{2.5}$ activates mitogen-activated protein kinase (MAPK) and causes apoptosis in rat cardiac cells [54]. The PM$_{2.5}$ effects on EGFR/PI3K/AKT and NLRP (ie, NACHT, LRR and PYD domains-containing protein) and leads to inflammation [55]. Besides, PM$_{2.5}$ accelerates regulatory T cell (Treg) generation by altering forkhead box P3 (Foxp3) gene transcription [56]. The PM$_{2.5}$ disrupts the metabolism of xenobiotics through Aryl hydrocarbon receptor (AhR) and increases transforming growth factor β (TGFβ) [57].

**Cardiovascular Impairment**

Studies showed that there is a significant correlation between air pollution (PM$_{2.5}$) and CVD [58, 59]. Recent studies indicated that PM$_{2.5}$ could decrease heart rate variability (HRV) a marker for cardiac parasympathetic tone modulation which may correlate with cardiac morbidity and sudden death [60,61]. The PM$_{2.5}$ exacerbates the autonomic nervous system and can lead to cardiac arrhythmia [62]. Furthermore, polymorphisms in glutathione S-transferase enzyme-1(GSTM1) and HFE (hemochromatosis) genes impairs HRV [63-65]. The HRV depressing is associated with aging, and low HRV causes CVD and coronary disease [66, 67]. As mentioned above, proinflammatory cytokine release occurs due to the exposure to PM$_{2.5}$, TNF-α and IL-6, two inflammatory markers associated with increased PM$_{2.5}$ [5], increase the risk of myocardial infarction (MI) and out-of-hospital cardiac arrest [68-70]. Also, IL-6 increases C-reactive protein (CRP). The CRP is an indicator of inflammation in CVD as a rapid response to air pollution. With every 100 μg/m$^3$ in PM$_{2.5}$ concentration, blood CRP level increases to 8.1 mg/L [71]. In this case, Dabass et al. suggested that PM$_{2.5}$ is not significantly correlated with biomarkers of CVD, though can increase these biomarkers in the presence of metabolic disease [72]. Another adverse effect of PM$_{2.5}$ is shortening prothrombin time and increasing fibrinogen, tissue factor, hypercoagulable state, and thrombosis [73, 74]. The PM$_{2.5}$ activates platelets at high shear rates through phosphoinositide 3-kinase/AKT (PI3Kβ/AKT) and glycogen synthase kinase 3 (GSK3) pathway and ultimately causes emboli [75]. Macrophages devour any xenobiotic that is foreign to them including PM$_{2.5}$ and result in increasing adhesion molecules. The ROS leads to inflammation, endothelial damage, and apoptosis; thrombus formation, vascular damage, EPC depletion, high levels of endothelin-1 and imbalance between supply and demand occurs. Hence, all these events explain vascular pathologies, hypertension and coronary artery disease related to the PM$_{2.5}$ [76-79].

In Tehran the capital city of Iran which trapped by air pollution with the largest population there was a significant relationship between the exposure to PM$_{2.5}$ and changes in HRV in 2010. Tehran is one of the most populated cities in the world, and approximately 20% of total population of Iran lives in Tehran; thus, the HRV, cardiac morbidities and mortalities should be more taken into consideration. Davoodi et al. conducted a study on 21 young people who had exposure to the polluted air and compared it to their situation in the clean air. Besides, they applied continuous Holter monitoring of electrocardiogram (ECG) and determined QT interval; they suggested that air pollution may increase nonsustained supraventricular tachycardia [80, 81]. Another study in Tehran demonstrated that tachycardia, cardiac dysfunction, endothelial damage and more atherosclerosis happens under the high PM$_{2.5}$ concentration circumstances [82-84].

**Lung Impairment**

The PM$_{2.5}$ passes the barriers deeply into the alveoli where it can exert respiratory problems [85]. It gives rise to cough hypersensitivity by increasing the expression of a non-selective cation channel [86]. Increasing asthma and asthma-like airway inflammation, reflect the danger of PM$_{2.5}$ for the respiratory system [87, 88].
The PM$_{2.5}$ is a risk factor for lung cancer and increases mortality from lung cancer [89, 90]. Sometimes, chemical components such as polycyclic aromatic hydrocarbons attach to the PM$_{2.5}$ and go deep into the alveoli acting as a carcinogen [91]. The higher rate of phagocytosis, oxidative stress, and inflammation post PM$_{2.5}$ exposure could impair immunity of the respiratory system which can lead to aggregation of microbes and infection. This process adds to decreasing cardiovascular function resulting in exacerbation of chronic obstructive pulmonary disease (COPD) [92]. The ROS and the activation of metabolic enzymes may induce pneumonia [93]. The PM$_{2.5}$ correlates with the onset of asthma and higher asthma morbidity; also decreasing forced expiratory volume in 1 second (FEV1) due to the asthma is more noticeable in comparison with non-polluted air condition [94]. The presence of PM$_{2.5}$ aggravates respiratory symptoms such as chest pain, dyspnea, sore throat, and wheezing [83, 95]. In Iran, a cross-sectional study gathered meteorological and air pollution information from 31 air quality monitoring station in Tehran and compared cardiovascular (68.36%) and respiratory admission (31.64%). This study showed that PM$_{2.5}$ increases respiratory hospital admissions and also it is correlated with more respiratory admissions to emergency [96].

### Neurodegenerative Impairment

A survey carried out on animals suggested that PM progresses to the brain through the nose and absorption from the digestive tract [97]. It revealed that lipid peroxidation and level of CRP had been associated with Parkinson’s disease (PD) [98]. The PM$_{2.5}$ correlates with neuroinflammation in the central nervous system, while some cohort studies showed that fine PM does not have potential effects on PD [99, 100]. Liu et al. observed a higher prevalence of PD among non-smoking women who inhaled PM$_{2.5}$ [101]. Microglia (the resident innate immune cell in the brain) express TLRs on their surface which PM$_{2.5}$ can attach these receptors leading to a chronic activation of microglia. Chronic microglial activation results in more ROS production as a protective mechanism. However, the subsequent inflammation triggers a neuronal loss [102].

In an area covered with air pollutant, solar rays do not reach the earth properly, and vitamin D deficiency is prevalent among the people in such areas. Vitamin D deficiency is a risk factor for cognitive impairments. High concentration of lipids and poor level of vitamin D play a critical role in Alzheimer’s disease (AD) [103]. Cyclooxygenase-1 (COX-1) and COX-2 increase during prolonged inhalation of particulate matter which reflects early changes in the brain in AD [104]. A pilot study in Iran suggested that there is a relationship between hypertension and AD [105]. The PM$_{2.5}$ also is a trigger of stroke and raises the number of the emergency admissions posts cerebrovascular accident. A retrospective cross-sectional study in Iran indicated that increasing concentration of PM$_{2.5}$ over 2 weeks and long-term changes in this pollutant elevates the risk of stroke admission 1.09 times [106].

### Endocrine Disorders

According to the fact that the prevalence of diabetes is considerable in Iran and PM$_{2.5}$ increases the risk of diabetes due to the insulin resistance along with aggravating the metabolic imbalance; it is more important to pay more attention to the air quality in Iran [107, 108]. Another effect of PM$_{2.5}$ on the endocrine system is damaging the blood-testis barrier, oligospermia, and impairing testicular function as shown in animal studies [109]. There is some evidence that states the interaction between PM$_{2.5}$ and AhR [57]. Furthermore, AhR-dependent apoptosis diminishes follicular maturation and has negative effects on the female reproductive system [110]. Having a child is one of the most vital issues in Iranian culture. Therefore, the clear mechanism of endocrine disruption by PM$_{2.5}$ through AhR and other pathways remain to be understood. There is no extensive study conducted on the relationship between PM$_{2.5}$ and
endocrine disorders in Iran; so, future studies should be done to fill our knowledge gaps.

**Other Adverse Effects**

Hypercoagulable state, hemodynamic problems, endothelial dysfunction, oxidative stress, and inflammation are five possible reasons for low birth weight children and infant mortality (add references for this statement). The PM$_{2.5}$ competes with growth factors (add references for this statement); also, impairs the passage of nutrient and oxygen across the placenta which causes preterm birth. Eosinophilic rise and nasal inflammation induce asthmatic allergy during childhood [111, 112].

Treg proliferation is the result of activation of Foxp3 transcription factor via TGFβ induction that happens following PM$_{2.5}$ exposure. Besides, AhR promotes Treg differentiation that acts as an immunosuppressive agent [56, 113]. Genotoxic effects of PM$_{2.5}$ as a fundamental aspect of various cancers and other disorders should be fully determined in Iran [114]. Finally, the detrimental effects of PM$_{2.5}$ on gastrointestinal tract is another dilemma which should be surveyed broadly because of its intestinal absorption and evidence gave for more hospitalization due to the peptic ulcer disease after PM$_{2.5}$ exposure [115].

In Iran, some studies conducted on particulate matter and defined the adverse effect of these matters (Table-1).

**Suggestions**

Since a mild to the moderate rise of PM$_{2.5}$ would be harmful to a vulnerable group including old age people and children, one suggestion is to install air cleaners in the schools for school-aged children and nursing homes for elderly care [88]. Forasmuch as the mechanisms of PM$_{2.5}$ is somewhat known; a possible solution is using the agents which modulate these mechanisms, for example, applying antioxidants like vitamin A, C, and E or β carotene may be beneficial in alleviating inflammation. However, more studies are necessary to prove this claim [116, 117].

Soy oil and fish oil supplements (rich in Omega-3) are the best sources to decrease oxidative stress. These polyunsaturated fatty acids should be more consumed in cities which are established in the deserts of Iran where far from the sea [118].

As discussed above wide range of disorders are related to PM$_{2.5}$ (Figure-2); as a consequence wide range of treatments are enable for attenuating these effects.

Controlling HRV, hypertension, diabetes by the use of β blockers, antihypertensive agents, and managing diabetes is approximately a kind of constitutional treatment [119].

Other environmental interventions including using air purifiers, assessment of PM$_{2.5}$ concentration by calibrator machines, the foundation of industries away from metropolises, seeding more plants, and using new technologies which are less harmful to the atmosphere are programs that should be performed by the government. For example, Tehran was equipped with subway system several years ago. Thus the concentration of pollutants is a key factor for the quality of underground stations [120].

On the other hand, people should follow some advice: avoid going out or exercising at high concentrations of PM$_{2.5}$ (based on the daily reports), more use of public transports, more walking and cycling instead of using private cars and motorcycles, and ultimately cooperating with the government in order to improve air quality and advance healthy air [121, 122].

Characterization of PM$_{2.5}$ and visibility measurement with professional satellites is another way to determine more accurate concentrations of this pollutant which can predict the exposure in Iran and the countries in its neighborhood [123, 124]. So, it could be helpful for civilization, migration, living and programming.

**Conclusion**

Iran is a country in the Middle East which wrestles with the PM$_{2.5}$ pollution. Many studies suggested that PM$_{2.5}$ can be the cause of mortalities and morbidities in Iran and even trigger many diseases due to the multi-organ damage. Despite the discovery of mechanisms of adverse effects worldwide
<table>
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<tr>
<th>Authors</th>
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<tr>
<td>Shahsavani et. al. [28]</td>
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<td>Rashki et. al. [30]</td>
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<td>Urmia lake bed</td>
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<td>Gholampour et. al. [26]</td>
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<td>Dust</td>
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<td>Bonyadi et. al. [117]</td>
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<td>Mashhad</td>
<td>Variable sources</td>
<td>Increased total mortality</td>
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**CVD**: Cardiovascular Disease; **AQI**: Air Quality Index
such as inflammation and oxidative stress, no study demonstrates which mechanism is prominent in Iran to better overcome the detrimental effects.

Increasing PM$_{2.5}$ is a major concern for public health effect. Since the countries are affected by each other in the air pollution, and Iran nearly is in the center of other countries in the Middle East geographically, new strategies should be scheduled to attenuate the amount of PM$_{2.5}$ concentration and mitigate its detrimental effects. The accurate assessment and finding characterization PM$_{2.5}$ in Iran are the priorities that should be tightly performed. There are some solutions to improve air quality and clean environment that WHO, the government of Iran and every individual should attempt to apply. Finally, more and more studies must be done to fill our knowledge gaps.

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Conflict of Interests

The authors declare that they have no conflict of interests.

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