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The Effect of Air- Polluting Particles on Cardiovascular Disease: A Review

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Abstract
Air pollution is the result of the emission of chemicals, particulate matter, and biologic material into the atmosphere, which caused adverse effects on the environment and health. Major air pollutants that threaten human health are carbon monoxide, Nitrogen oxide, ozone, Sulfur dioxide, chemical vapors, and particulate matter. Air pollution is an important risk factor for cardiovascular health, therefore in this review article; we discuss air-polluting particles and the adverse effects of them on cardiovascular disease.
Keywords: Air Pollution, Particulate Matter, Cardiovascular Diseases

Introduction
With advances in technology, air pollution has also increased. Air pollution causes mortality and cardiovascular disease in population (1). Studies has shown that increase in air pollution is associated with myocardial infarction (2) and heart failure (3). Air pollutants causes adverse effect on vascular tone, endothelial function, thrombosis, and myocardial ischemia (4). Nanomaterials could enter the blood, lymph, and bone marrow and arrive to lymph nodes, spleen and heart (5) and then affecte coagulation and heart rhythm (6). Brown showed that nanoparticles like titanium dioxide (TiO_2) and Carbone (C) caused air pollution, inflammation, skin injury, and lung accumulation (7). Air pollutants include gaseous pollutants (e.g, carbon monoxide, ozone, nitrogen oxide and sulfur dioxide) and particulate matters (PMs). In this review article, we discuss air-polluted particle (include ambient air particles, diesel exhaust, ozone and carbon monoxide) and adverse effects of them on cardiovascular disease.

Ambient Particulate Matter (PM)
The properties of PM are similar to mineral dusts. Studies show that the existence of PM is associated with premature cardiopulmonary death (8). The gaseous air pollutants (ie, ozone, sulfur and nitric oxides, carbon monoxide) and particulate matter (PM) cause adverse effects on health. There is relationship between exposure to PM10 and venous thrombosis; as for each 10 g/mm^3 rise of PM10 there was a 70% increase of venous thrombosis (9). Another study showed exposure to PM make shorter prothrombin time (10). Living near the traffic roads cause risk increase of venous thrombosis and arterial cardiovascular events (11). There are relationship between concentration of PM and a quickly change in heart rate (12) in healthy subjects. Brook *et al.* (2010) and Goldberg *et al.* (2001) showed that increased in

atmospheric pollution caused chronic obstructive pulmonary disease (13) and heart failure (14), respectively. Heart failure is associated with ultrafine particles regardless of age, sex, cardiovascular risk factors, and drug treatments. PM level is a risk factor for the almost diseases that contributed in cardiovascular disease such as hypercholesterolemia, arterial hypertension, tobacco smoking, diabetes and obesity (13, 14). Gaseous and particulate pollutants induced adverse health effects. Dockery *et al.* showed that PM is a major reason of various types of human disease. PM rarely exists by itself within the ambient environment because gaseous and volatile compounds (ie, aldehydes and polycyclic aromatic hydrocarbons) are continuously changing and interacting. PM2.5 and PM0.1 are inhaled deeply into the lungs, after that deposited in the alveoli, and entering the pulmonary circulation (15). Peters *et al.* (2001 and 2004) showed that the exposure to road traffic pollution (16) or PM2.5 (17) increased risk of acute myocardial infarction. After exposure to a mixture of concentrated ambient particles and ozone reduced brachial artery diameter (18). Nemmar *et al.* showed that PM caused plasma viscosity, endothelial dysfunction and altered autonomic control of the heart. The intravenous administration of ultrafine polystyrene particles, diesel exhaust particles, or PM2.5 changed hemostasis (19). Baccarelli *et al.* (2007) showed the air pollution exposure changed blood homeostasis (20). Baccarelli *et al.* (2008) investigated the effect of exposure of PM10 on the risk of developing deep vein thrombosis in 870 patients and 1210 control subjects. They showed that the long-term exposure of PM10 is associated with altered coagulation function and deep vein thrombosis risk (21). Air pollution may induce atherosclerosis in the peripheral arteries, coronary arteries, and aorta. The short-term exposure of PM is associated with increased acute cardiovascular mortality, although prolonged exposure caused a causative factor for atherosclerosis (22). The exposure of PM2.5 caused a risk factor for cardiovascular disease mortality through pulmonary and systemic inflammation, accelerated of atherosclerosis, and altered cardiac autonomic function (23). A panel study showed a relationship between long-term PM exposure and atherosclerosis in humans (24). Pekkanen *et al.* showed that increased levels of fibrinogen, platelets, and white blood cell counts were also associated with exposure to total suspended particles (25).

Diesel Exhaust

The emissions of diesel exhaust are a significant air pollution source in urban environments (26, 27). Exposure of diesel exhaust particles (DEP) used as particulate air pollution models in experimental studies (26). Experimental studies showed that exposure of air pollution increased leukocyte and erythrocyte numbers, and hematocrit (28). A key mechanism of air pollution relationship to the generation of inflammation and induce oxidative stress (26). Recently, studies showed that exposed to air pollution induced myocardial inflammation characterized by increased of tumor necrosis factor alpha (TNFα) and interleukin 1 beta (IL1β) (29). Nemmara *et al.* showed that emodin adminstraton following DEP decreased cardiac proinflammatory cytokine TNFα and IL1β (30). Exposed to DEP caused a significant decreased of superoxide dismutase (SOD) in heart tissue. Nemmara *et al.* showed that exposure of DEP caused shortening of the thrombotic occlusion time in venules and pial arterioles. Also, emodin has protective effect against DEP-induced thrombotic complications in pial arterioles and venules (30). Clinical studies showed that acute DE inhalation caused systolic blood pressure increasing, vasodilation impairments in humans (31). DE exposure caused moderate changes in HRV and electrocardiography in rats with minimal cardiac hypertrophy (32). Short-term exposure of air pollution is

associated with changes in the general coagulation function that suggested a tendency towards hypercoagulability (27). Ikeda et al showed that prepared aortic ring with diesel exhaust particles prevented acetylcholine-mediated relaxation (33). Mills *et al.* (34) and Peretz *et al.* (35) not observed any HRV changes in humans exposed to diesel exhaust.

Ozone

Ozone (O_3) is one of the most air pollutants that effects on human health. Ozone is a molecule composed of three oxygen atoms linked together in a high-energy compound. Generally, Ozone during the day in the summer months reaches the upper levels and this type of air pollution known as summer and daily changes (36). Sequential exposures of O3 and then PM2.5 reduced HRV, systolic blood pressure and heart rate (HR) in rats (37). Simoultanously exposure to PM2.5 and O_3 caused acute arterial vasoconstriction in healthy men (38). Although, another study found no significant change in mean arterial pressure, and systolic blood pressure (39).

Carbon Monoxide

Carbon monoxide (CO) toxicity has clinical adverse effects on all the organ and tissue (40), and causes high toxicological morbidity and mortality (41). Several studies showed relationship between CO toxicity and cardiac dysfunction like myocardial injury (41). Bernal and Moro showed that there is significant relationship between CO toxicity and increased risk of developing arrhythmia and coronary artery disease (CAD). Following CO toxicity in cohort study, the incidence rate of arrhythmia in men was higher than in women (42). Akdemir *et al.* (2014) showed female patient with CO toxicity following treatment with normobaric oxygen, the rhythm of their heart returned to normal sinus rhythm (43). Studies showed a correlation between CO toxicity and CAD (44).

Discussion

The mechanism of air- polluted particle in cardiovascular diseases

Thrombosis

Studies showe that ambient PM causes increased plasma viscosity and fibrinogen concentrations (45). Also Ambient PM10 levels increase platelet aggregation 24 to 96 hours after exposure among healthy adults (46). Studied showe that PM causes prothrombotic effects, including increased expression of tissue factor on endothelial cells (47), and accumulation of platelets and fibrin on the endothelial surface (48). The results displayed increased risk of venous thromboembolic disease is associated with long- term exposure to air pollution (49). Generally, exposed to PM caused to increase the thrombotic potential, especially under conditions of vascular injury. Also, another study showed that exposed to indoor air pollution from the combustion of biomass fuels caused to increase in platelet activation and platelet–leucocyte aggregation (50) (Table 1).

Atherosclerosis

The studies showed that long-term exposures to ambient air pollution associated with increased rates of atherosclerosis in humans. In a cohort study reported that exposure to long-term PM2.5 caused to abdominal aortic calcium (51). Animal study showed that inhalation of ultrafine particles (UFPs) and PM2.5 increased the atherosclerotic aortic lesions in apolipoprotein E-deficient in human and rabbit (51-53) (Table 2).

Conclusion

Air pollution is an important agent of cardiovascular disease in urban people. Acute exposure is associated with adverse cardiovascular events including hospital admissions with myocardial infarction, angina and heart failure. Long-term exposure increase risk of death from coronary heart disease. The

countries should use air pollution control methods with decreased emission of particulate matter, and biologic material into the atmosphere.

Table 1. The effect of air pollution on thrombosis (54-57)

Study	Exposure	Animal	Main Result
Nemmar et al. 2003	PM2.5	hamster	Exposure of PM2.5 caused occurrence of thrombotic complications
Baccarelli et al. 2009	Traffic road pollution	human	Living near major traffic roads increased risk of deep vein thrombosis (DVT)
Baccarelli et al. 2008	PM10	human	PM10 shortened prothrombin time (PT) in DVT cases
Rich et al. 2012	pollution in Olympic period	human	Air pollution during the Beijing Olympics are associated with acute changes in biomarkers of inflammation and thrombosis

Table 2. The effect of air pollution on atherosclerosis (58-64)

Study	Exposure	Animal	Main Result
Suwa et al. 2002	PM10	rabbits	PM10 progressed atherosclerosis and increased vulnerability to plaque rupture
Künzli et al. 2005	PM2.5	human	There are association between atherosclerosis and ambient air pollution
Chi et al. 2016	PM2.5 and oxides of nitrogen (NOX)	human	There are association between long-term ambient air pollution exposure and site-specific DNA methylation.
Rivera et al. 2013	Nitrogen dioxide (NO2)	human	There are association between Long-term traffic- exposure and subclinical markers of atherosclerosis
Hartiala et al. 2016	PM2.5 and NO2	human	PM2.5 increased coronary artery disease and the risk of incident myocardial infarction
Gan et al. 2014	Traffic air pollution	human	There are no significant relationship between traffic-related air pollution and progression of carotid artery atherosclerosis
Yatera et al. 2008	PM10	human	PM10 promoted the recruitment of circulating monocytes into atherosclerotic plaques

List of abbreviations

CO (Carbon Monoxide); NO_2 (Nitrogen Oxide); SO_2 (Sulfur dioxide); O_3 (Ozone); DEP (Diesel Exhaust Particles); HR (Heart Rate); HRV (Heart Rate Variability); PM (Particulate Matter); ROS (Reactive Oxygen Species); UFPM (Ultrafine Particulate Matter).

Ethical issues

Not applicable.

Authors' contributions

All authors equally contributed to the writing and revision of this paper.

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