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Impact of Climate Change and Air Pollution on Dyslipidemia and the Components of Metabolic Syndrome

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1. Introduction

Environmental factors, notably climate change and air pollution influence health before conception, and continue during pregnancy, childhood, and adolescence. Experts have suggested that such health hazards may represent the greatest public health challenge humanity has faced. The accumulation of greenhouse gases such as carbon dioxide, primarily from burning fossil fuels results in warming, which has an impact on air pollution, particularly on levels of ozone and particulates. Heat-related health effects include increased rates of pregnancy complications, pre-eclampsia, eclampsia, low birth weight, renal effects, vector-borne diseases as malaria and dengue; increased diarrheal and respiratory disease, food insecurity, decreased quality of foods (notably grains), malnutrition, water scarcity, exposures to toxic chemicals, worsened poverty, natural disasters, and population displacement. Air pollution has many adverse health effects, which would have long-term impact on the components of the metabolic syndrome. In addition to short-term effects as premature labor, intrauterine growth retardation, neonatal and infant mortality rate, malignancies (notably leukemia and Hodgkin lymphoma), respiratory diseases, allergic disorders and anemia, exposure to criteria air pollutants from early life might be associated with dyslipidemia, increase in stress oxidative, inflammation and endothelial dysfunction which in turn might have long-term effects on chronic non-communicable diseases.

2. Environmental factors: Climate change and air pollution

2.1 Air pollutants

Air pollution is a mixture of solid particles and gases in the air. The six common and hazardous air pollutants consist of particulate matter, ground-level ozone, carbon monoxide, sulfur oxides, nitrogen oxides, and lead; of which, particle pollution and ground-level ozone are the most widespread health hazards (Samet & Krewski, 2007; Chen & Kan, 2008).

Particulate matter or PM consists of a diverse mixture of very small particles and liquid droplets suspended in air. The PM size is directly related to their potential for affecting health. Particles with diameter < 10 micrometers are the particles that usually pass through the throat and nose to enter the lungs. Then, they can affect different body organs especially the heart and lungs, and may cause serious health effects. According to the size, the particle pollution is grouped into: "inhalable coarse particles" which have a diameter of 2.5 to 10 micrometers, and are found near roadways and industries, and "fine particles" < 2.5 micrometers in diameter such as those found in smoke and haze; they can form when gases emitted from power plants, industries and automobiles react in the air. Ozone (O3) is a gas composed of three oxygen atoms. In the presence of sunlight, it is created at ground-level by a chemical reaction between oxides of nitrogen and volatile organic compounds. Ozone might have harmful effects when formed in the earth's lower atmosphere, i.e. at ground-level. Hot weather and sunlight cause ground-level ozone to form in harmful concentrations in the air. Carbon monoxide (CO) is an odorless and colorless gas formed by incomplete carbon combustion. It is mainly emitted from the motor vehicle exhaust followed by non-road engines as construction equipment, industrial processes and wood burning. The increasing number of cars has an important role in the increase in CO emission worldwide. Sulfur Dioxide (SO2) is a gas formed when fuel containing sulfur, such as coal and oil, is burned, and when gasoline is extracted from oil or metals are extracted from ore. Nitrogen oxides (Nox) are a group of highly reactive gases containing various levels of nitrogen and oxygen. Lead is usually emitted from motor vehicles and industrial sources (Chen & Kan, 2008; Brook et al., 2004). Other stationary sources are waste incinerators, utilities, and lead-acid battery manufacturers. In addition to exposure to lead in air, other major exposure pathways include ingestion of lead in drinking water and lead-contaminated food as well as incidental ingestion of lead-contaminated soil and dust. Lead-based paint remains a major exposure pathway in older homes. Some toys might contain considerable amounts of lead that would be harmful for children's health (Samet & Krewski, 2007; Han & Naeher, 2006).

2.2 Climate change

Climate change and global warming have various health hazards (Poursafa & Kelishadi 2011).

Climate change has an impact on levels of ozone and particulate matters, which are both associated with various health hazards. The accumulation of greenhouse gases such as carbon dioxide, primarily from burning fossil fuels results in warming. Heat increases ground level ozone production, which in turn augments morbidity and mortality (Bell et al., 2007). Moreover, warming increases water vapor and ground-level ozone formation, and will result in harmful ozone levels (Jacob & Winner, 2009). Warming modify the risk of forest fires, and may generate massive amounts of carcinogens, as formaldehyde and benzene, potent lung irritants, as acrolein and other aldehydes, carbon monoxide, and particulates (Wegesser et al., 2009).

3. Association of environmental factors and dyslipidemia

Environmental factors are associated with many chronic diseases. Air pollution and climate change are associated with risk factors of non-communicable diseases in children and adolescents (Kelishadi et al., 2009; Sheffield & Landrigan, 2011; Kelishadi & Poursafa, 2010; Mansourian et al., 2010; Poursafa et al., 2011; Kargarfard et al., 2011).

The effects of environmental factors on intrauterine growth retardation and preterm labor are well documented. In turn, low birth weight (Sinclair et al., 2007) and prematurity (Evensen et al., 2008) would increase the risk of chronic non-communicable diseases and their risk factors. Exposure to air pollutants is reported to be associated with stress oxidative and markers of insulin resistance (Kelishadi et al., 2010) as well as with diabetes mellitus (Brook et al., 2008). These systemic responses to environmental factors can potentially increase the risk for dyslipidemia, development of the metabolic syndrome, hypertension, and other chronic diseases.Moreover, it is documented that some environmental factors as increased humidity are associated with preeclampsia and eclampsia, and their related consequences (Subramaniam, 2007).

Several reports exist on the association of environmental factors with dyslipidemia and the components of metabolic syndrome. By applying generalized additive models, Secondary analyses of a Taiwanese survey in 2002 demonstrated that increased particulate matter with aerodynamic diameters <10 microm was associated with elevated systolic blood pressure, triglycerides, apolipoprotein B, hemoglobin A1c, and reduced high-density lipoprotein (HDL) cholesterol. Elevated ozone was associated with increased diastolic blood pressure, apolipoprotein B, and hemoglobin A1c (Chuang et al., 2010). Genetic-environment interactions may have a role in this regard (Eisenberg et al., 2010).

The associations of both obesity and air pollution with several age-related diseases remain poorly understood with regard to causality and underlying mechanisms. Exposure to both, excess body fat and particulate matter, is accompanied by systemic low-grade inflammation as well as alterations in insulin/insulin-like growth factor signaling and cell cycle control. Understanding the causality of exposure disease associations and differences in susceptibilities to environment and lifestyle is an important aspect for effective prevention (Probst-Hensch, 2010).

A case-control study evaluated the effects of urban pollution on the lipid balance of members of a municipal police force in comparison with controls. Mean and frequency distributions of HDL-cholesterol and triglycerides had significant difference between the exposed traffic police group and controls. This study suggested that some chemical agents, as carbon dioxide, of the urban pollution could cause dyslipidemia among exposed people (Tomao et al., 2002).

A study among asthmatic patients showed that with a 1-microg/m3 increase in coarse PM, triglycerides increased 4.8% ($p = 0.02$), and very low-density lipoprotein increased 1.15% ($p = 0.01$). This study suggested that small temporal increases in ambient coarse PM are sufficient to affect lipid profile in adults with asthma (Yeatts et al., 2007).

Hypercholesterolemia may potentiate diesel exhaust-related endothelial gene regulation. These regulated transcripts may implicate pathways involved in the acceleration of atherosclerosis by air pollution (Maresh et al., 2011). The systemic pro-inflammatory and pro-thrombotic response to the inhalation of fine and ultrafine particulate matters may be associated with platelet activation (Poursafa & Kelishadi, 2010 platelets).

A study conducted in Greece, explored the relations between ambient environmental factors and arterial stiffness, peripheral and central hemodynamics in a cohort of 1222 participants.It found that the exposure to lower environmental temperatures is related to impaired hemodynamics not only to the periphery but also to the aorta. In men, PM10 levels were associated with intensified amplitude of the reflection wave resulting in significant alterations in central-pulse pressure (Adamopoulos et al., 2010).

A study examined the associations of PM2.5 with heart rate variability, a marker of autonomic function, and whether metabolic syndrome modified these associations. It found significant correlations; which were stronger among individuals with metabolic syndrome than among those without it. This study proposed that autonomic dysfunction may be a mechanism through which PM exposure affects cardiovascular risk, especially among persons with metabolic syndrome (Park et al., 2010).

Exposure to high and low air temperatures are associated with cardiovascular mortality, the underlying mechanisms are still under investigation. In a cohort in the US, 478 men with a mean age of 74.2 years were followed up from 1995 to 2008. Associations of three temperature variables, i.e. ambient, apparent, and dew point temperature with serum lipid profile were studied with linear mixed models by including possible confounders such as air pollution and a random intercept for each individual. HDL decreased -1.76%, and -5.58% for each 5°C increase in mean ambient temperature. For the same increase in mean ambient temperature, LDL increased by 1.74% and 1.87%. Similar results were also found for apparent and dew point temperatures. No changes were found in total cholesterol or triglycerides in relation to temperature increase. This study suggested that changes in HDL and LDL levels, which are associated with an increase in ambient temperature, may be among the underlying mechanisms of temperature-related cardiovascular mortality (Halonen et al., 2011).

A study in Taiwan found that increased 1-year average ozone, PM and nitrogen dioxide were associated with elevated blood pressure, total cholesterol, fasting glucose, and HbA1c. PM2.5 was more significantly associated with end-point variables than other gaseous pollutants (Chuang et al., 2011). A study on the association of blood markers of cardiovascular risk and air pollution in a national sample of the U.S. population found that PM_{10}, but not gaseous air pollutants, is associated with blood markers of cardiovascular risk (Schwartz, 2001). In a study in Italy, the carboxyhaemoglobin concentration had an inverse correlation with HDL-C (Biava et al., 1992). In addition to the outdoor environment, the health hazards of indoor pollution should be considered (Kaplan, 2010).

In recent years, global climate change has affected many biological and environmental factors. Of its most important effects are the increasing levels of atmospheric carbon dioxide, ultraviolet radiation, and ocean temperatures. In turn, they have resulted in decreased marine phytoplankton growth and reduced synthesis of omega-3 polyunsaturated fatty acids. It is suggested that the detrimental effects of climate change on the oceans may reduce the availability of dietary omega-3, which may have detrimental effects on serum lipid profile (Kang, 2011).

4. Conclusion

Climate change may alter concentrations of air pollutants or alterations in mechanisms of pollutant transport and thus influence individual and public health. The potential impacts of climate change and air pollution on lipid disorders is considered as an important area of investigation. This is of special concern for low- and middle-income countries, where the burden of air pollution and climate-related health disorders, and the burden of non-communicable diseases are emerging. Effects of environmental factors on dyslipidemia should be considered in primordial and primary prevention of chronic diseases from early life.

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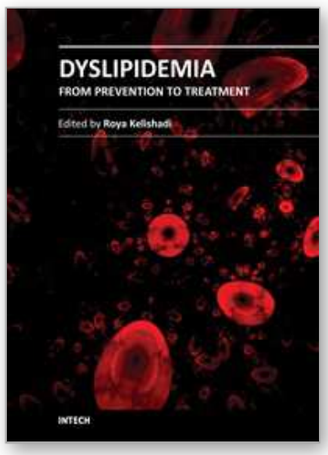

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Dyslipidemia has a complex pathophysiology consisting of various genetic, lifestyle, and environmental factors. It has many adverse health impacts, notably in the development of chronic non-communicable diseases. Significant ethnic differences exist due to the prevalence and types of lipid disorders. While elevated serum total- and LDL-cholesterol are the main concern in Western populations, in other countries hypertriglyceridemia and low HDL-cholesterol are more prevalent. The latter types of lipid disorders are considered as components of the metabolic syndrome. The escalating trend of obesity, as well as changes in lifestyle and environmental factors will make dyslipidemia a global medical and public health threat, not only for adults but for the pediatric age group as well. Several experimental and clinical studies are still being conducted regarding the underlying mechanisms and treatment of dyslipidemia. The current book is providing a general overview of dyslipidemia from diverse aspects of pathophysiology, ethnic differences, prevention, health hazards, and treatment.

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