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AIR POLLUTION AND CARDIOVASCULAR DISEASE

SUMMARY

The development of cardiovascular disease is a result of a chronic and complex interplay between genetic and environmental factors. Interest in the association between air pollution and health status has been increasingly focused on the relationship between air pollution and cardiopulmonary disease including hypertension, myocardial infarction and other circulatory disorders. There are a small number of publications on the health effects of long-term exposure. Observations related to the adverse health effects of short-term exposure are more numerous. A major challenge to systematic study in this field is the complexity of the particulate components of air pollution. Ambient air particulate matter consist of a mixture of combustive by-products and resuspended crustal materials, as well as biological materials such as pollen, endotoxins, bacteria and viruses. Inhaled fine particles can be detected within minutes of exposure in the systemic circulation, where they can persist for hours, providing a route of entry into all organ systems. Although epidemiological studies have linked air pollution with cardiopulmonary mortality, underlying biological mechanisms remain largely unknown. Current biologic knowledge suggests direct effects of pollutants on the cardiovascular system, blood, and lung receptors and indirect effects mediated through pulmonary oxidative stress and inflammatory responses. We need new studies focused only to those issue because the quality of the air in our environment has a most essential and direct impact on the health and quality of life.

Key words: **air pollution, cardiovascular disease, long-term exposure, short-term exposure**

INTRODUCTION

Cardiovascular disease (CVD) rapidly becoming a major cause of death worldwide. Although intensively studied, the reasons underlying the high incidence of CVD remain unclear because the development of CVD is a result of a chronic and composite interaction among genetic and environmental factors. Large changes in the incidence of CVD over the last century point out that environmental influences are important (1,2).

Numerous recent studies conducted all over the world shown that acute exposures to current ambient levels of air pollution are linked with adverse health status (mortality, elevated hospital admissions, severity of preexisting chronic illnesses, etc).

Interest in the association between air pollution and health status has been increasingly focused on the relationship between air pollution and cardiopulmonary disease. Most previous epidemiologic research on this issue has indicated a generally consistent pattern of excess risk with respect to mortality and hospital admissions for cardiac diseases.

The raise in relative risk for heart disease due to air pollution for an individual is small compared with the impact of the established cardiovascular risk factors such as high blood pressure or high cholesterol. On the other hand, this is a serious public health problem due to the enormous number of people affected and because exposure to air pollution occurs over an entire lifetime. Despite improvements in air quality over the past few decades (3), associations between current ambient pollution levels and excess morbidity and mortality have been over and over again detected (4–9). The amount of evidence and number of studies linking air pollution to cardiovascular diseases has grown extensively (9–11).

Over the last decade of special interest are quite a few enviromental air pollutants that include carbon monoxide, oxides of nitrogen, sulfur dioxide, ozone, lead, and particulate matter. These pollutants are associated with increased hospitalization (12) and mortality due to cardiovascular disease (13,14), particulary in persons with congestive heart failure, frequent arrhythmias or both (15).

In broad terms, studies can be separated into those that have investigated the health effects of acute or chronic air pollution exposure.

LONG-TERM HEALTH EFFECTS

There are a small number of publications on the health effects of long-term exposure. These studies have involved analysis of data (total mortality and in some circumstances cardiovascular events) from a few large cohorts from multiple geographic locations that differ in the average chronic ambient concentrations and mixtures of air pollutants.

Harvard Six Cities study by Dockery et al. (16) was the first great, prospective cohort study that recognized an adverse health impact of long-term air pollution exposure. This study, in a cohort of 8111 adults, confirmed that chronic exposure to air pollutants is independently related to cardiovascular mortality. In study with 14 to 16 years of follow-up, the adjusted generally mortality rate for the most-polluted city in opposite to the least-polluted city was 1.26.

Between air pollutants, elevations of $PM_{2,5}$ and sulfated showed the strongest associations with disease. These result were complemented by similar observations from the first analysis of air pollution in relation to mortality in the ACS Cancer Prevention II study population (17). Lately, a follow-up of the original ACS cohort by Pope et al. (18) based on additional subject mortality and ambient pollutant data, has provided the largest study of the long-term health effects of air pollution. In around 500 000 adults who resided in all 50 states in USA, chronic exposure to multiple air pollutants was linked to mortality statistics for a 16-year period. The key results showed that each 10 $\mu g/m^3$ increase in annual $PM_{2.5}$ mean concentration, was associated with increases in all-cause, cardiopulmonary, and lung cancer mortality of 4%, 6% and 8%, respectively. There appeared to be an association between cardiopulmonary mortality and O_3.

The specific causes of the increased cardiovascular mortality due to long-term air pollution exposure have remained uncertain. In an analysis of the ACS study published last year, the investigators reported PM-mortality associations with the specific cause of death (19). A statistically robust association between $PM_{2.5}$ and overall cardiovascular mortality was confirmed for a 10 $\mu g/m^3$ increase in long-term exposure. The single largest increase in risk was for ischemic heart disease (RR=1.18), which also accounted for the largest proportion of deaths. In addition, the risk for arrhythmia, heart failure, or cardiac arrest mortality was also increased (RR=1.13). There was no evidence for excess mortality in the entire cohort due to other reasons (stroke, hypertensive disease, aortic aneurysms, diabetes or any respiratory illness). These findings imply that air pollution promotes both ischemic and nonishemic cardiovascular events.

SHORT-TERM HEALTH EFFECTS

Observations related to the adverse health effects of short-term exposure are more numerous. In these studies, population-wide changes in acute outcomes (mortality, symptomatology, hospitalizations and healthcare visits) are linked to short-term variations in ambient pollutant concentrations, most frequently through the use of population-based time-series analysis. More recently, case crossover designs have been added to the analytical list.

The Six Cities (17) and ACS studies (18, 19) offer strong evidence for the occurrence of adverse cardiovascular effects as a result of long-term air pollution exposure.

A lot of other studies have focused on short-term associations between pollution exposure and adverse outcomes. The acute effects of air pollution are generally investigated by time-series analyses of changes in health outcomes (mortality) in relation to day-to-day variations in ambient air pollu-

tion concentrations. The two main studies to date are the NMMAPS in the United States (20, 21) and the Air Pollution and Health: a European Approach (APHEA) project (22). These studies produced extraordinarily consistent results. The NMMAPS observed outcomes in 50 milion people in the 20 largest cities in the United States. Average mortality rates were independently associated with particle concentrations the day before death. Each 10 $\mu g/m^3$ elevation in PM_{10} was associated with an increase of 0.21% and 0.31% for daily all-cause and cardiopulmonary mortality, respectively (21).

Apheis is a public health surveillance system and presents the health impact assessment done in 29 cities of Western and Eastern European countries (22). The population covered in this health impact assessment includes nearly 43 milion inhabitants. It shows that current levels of air pollution in urban Europe (PM_{10} concentrations were ranged from 14 to 73 $\mu g/m^3$). The estimated increase in daily mortality was 0.6% for each 10 $\mu g/m^3$ increase in PM_{10}. Cardiovascular deaths were increased by 0.69%. Additional analyses of the APHEA mortality data, based on lag periods up to 40 days, establish that the risk of adverse health effects associated with air pollution more than doubled (1.97%) for each 10 $\mu g/m^3$ elevation in PM_{10} (24).

Over the last few decades numerous short-term studies have been published on the effects of acute air pollution exposure (23, 24). In time-series studies variations in air pollution levels have been shown to increase – mortality rates (25, 26), hospital admissions (27, 28), emergency room visits (29, 30) and symptom exacerebations (31, 32). Observations from across North America (33, 34) and Europe (35, 36) have demonstrated higher rates of hospitalizations for all cardiovascular causes. Direct associations have also been identified with respect to incidence of ischemic heart disease, arrhythmias, and heart failure. A pooled analysis of hospital admissions studies showed significant increases in admission rates of 0.8% and 0.7% for heart failure and ischemic heart disease, respectively, for each 10 $\mu g/m^3$ elevation in PM_{10} (37).

More focused investigations have demonstrated elevated risks for AMI (39), implantable cardioverter defibrillator discharges (40) and myocardial ischemia during stress testing (41).

Excessive elevations in air pollution have also been associated with increased blood pressure during a prolonged air stagnation episode in Europe (40). Finally, recent studies from South Korea (41) and Taiwan (42) have reported higher incidences of ischemic strokes in direct relation to changes in ambient particle concentrations. In summary, these findings imply that short-term elevations in ambient particle levels are capable of evoking cardiac arrhythmias, worsening heart failure, and triggering acute atherosclerotic-ischemic cardiovascular complications.

A a small number of observations have suggested that the elderly (17-19,43) may be mostly susceptible populations. In addition, the presence of preexisting chronic lung disease, coronary heart disease and heart failure may also raise short-term cardiovascular mortality risk (43). There is no convincing evidence that gender, race, and other preexisting coronary risk factors (obesity, dyslipidemia, hypertension) increase the risk of cardiovascular events due to air pollution.

POTENTIAL BIOLOGICAL MECHANISMS

Although epidemiological studies have linked air pollution with cardiopulmonary mortality, underlying biological mechanisms remain largely unknown. Studies about air pollution effects on animal models are sporadic and difficult to interpret (44). Current biologic knowledge suggests direct effects of pollutants on the cardiovascular system, blood, and lung receptors and indirect effects mediated through pulmonary oxidative stress and inflammatory responses. Seaton et al. (45) postulated that inflammation in the peripheral airways, caused by air pollutants, might increase the coagulability of blood, leading to an increased number of cardiac events. Peteres et al. (46) reported that during an acute air pollution episode in Germany increased blood viscosity was observed and that suggested the presence of a pathological mechanism linking air pollution to cardiac events.

Few studies suggests and association between elevated air pollution and increased blood viscosity (47), transient reductions in heart rate variability (48) and reduced blood oxygenation (49) – all of which may trigger cardiovascular disease. Prospective studies have reported that the risk of ischemic cardiovascular events is increased in individuals with coagulation activation or impaired fibrinolytic function associated with several plasma markers (50–52).

A number of studies in humans demonstrate that chronic inhalation of pollutants can induce changes in the electrocardiogram (53–58), but the sensory receptors and neural pathways involved in these changes have not been recognized. By comparison, several studies in experimental animals have permitted identification of sensory receptors and afferent and motor pathways (59–64).

Widespread literature has documented that irritants inhalted into the upper respiratory tract cause cardiovascular reflex changes that are predominantly bradycardiac and include either hyper or hypotension (65).

Human inhalation of pollutants can also cause changes in the heart, based on evidence from complex statistical analysis of the pattern of the ECGs. More modern studies support this possibility. Laryngeal irritation in humans can cause cardiac dysrhythmias with depression of the ST complex of the ECG (66). The role of the vagus nerves in dysrhythmias caused by inhalation of pollutants has been much discussed (55).

Plentiful evidence exists about reflexes, including those to the cardiovascular system, activated by inhalation of pollutants in experimental animals. Nearly all the studies have been acute or short term. Similar experiments suggest that humans have the same reflexes, but they have not been widely analyzed, especially with consider to the cardiovascular system. The applicability of this large body of research to the pathophysiologic results of long-term exposure to athmospheric pollutants is at present very fragile.

Numerous evidence (64) point out that $PM_{2.5}$ inhalation induce systemic inflammation and cytokine production (65) probably related to free radical activity of components in particulate matter (66). In turn, these have the capacity to enhance vascular ET expression by direct mechanisms or via activation of oxidative stress pathways.

Alterations in arterial tone and reactivity in response to $PM_{2.5}$ and O_3 exposure is a new field for future research into the biological mechanisms linking air pollution with acute and potentially chronic cardiovascular events (67). Further investigations are needed to confirm and extend conclustion to the coronary circulation and to subjects with existing heart disease. Overall, today is accepted that the health of many people is affected by exposure to air pollution. According to WHO is was anticipated that 800 000 deaths happen per year due to pollutant exposure (68). In the past decade, a number of epidemiological studies have shown that ambient air pollution adversely affects human health even at levels lower than WHO standards (3). Despite the generally consistent epidemiologic findings on the effects of air pollution on cardiovascular disease-related mortality or morbidity, in only a few studies investigators have focused on particular subtypes of cardiovascular disease. Therefore, we need new studies focused only to those issue because the quality of the air in our environment has a most essential and direct impact on the health and quality of life.

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AEROZAGAĐENJE I KARDIOVASKULARNE BOLESTI

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SAŽETAK

Razvoj kardiovaskularnih bolesti nastaje kao rezultat hroničnih i kompleksnih uticaja genetskih i spoljašnjih faktora. Interesovanje za ispitivanje uticaja aerozagađenja na zdravlje trenutno se fokusira na utvrđivanju eventualne povezanosti između izloženosti aerozagađenju i nastanka kardiovaskularnih bolesti kao što su hipertenzija, infarkt miokarda i poremećaji cirkulacije. Manji je broj studija koji je istraživao uticaj dugotrajne izloženosti aerozagađenju u odnosu na broj studija koji je pratio uticaj kratkotrajne ekspozicije na nastanak kardiovaskularnih oboljenja. Od svih polutanata vazduha, smatra se da čestice imaju najvažniju ulogu u nastanku pomenutih oboljenja. Češće predstavljaju kompleksnu mešavinu organskih i neorganskih materija i mogu imati različit hemijski sastav. Posle inhalacije, u roku od nekoliko minuta mogu se detektovati u cirkulaciji, gde perzistiraju satima i ulaze u unutrašnje organe. Mada su epidemiološke studije dokazale vezu između izloženosti aerozagađenju i kardiovaskularnih bolesti, mehanizam delovanja nije još uvek razjašnjen. Istraživanja u tom pravcu su pokazala da aerozagađenje može direktno delovati putem krvi na kardiovaskularni sistem ili indirektno, izazivajući pulmonarni oksidativni stres i inflamaciju. Potrebno je još mnogo istraživanja u ovoj oblasti jer aerozagađenje u našoj životnoj sredini ima veoma važan uticaj na zdravlje i kvalitet života.

Ključne reči: **aerozagađenje, kardiovaskularne bolesti, dugotrajna izloženost, kratkotrajna izloženost**