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Editorial

Fine Particulate Matter: a Threat to the Heart Rhythm

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▸ See the article "High Level of Real Urban Air Pollution Promotes Cardiac Arrhythmia in Healthy Mice" in volume 51 on page 157.

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Air pollution has attracted growing interest with regard to public health, since it is predicted to increase in severity as a result of climate change. Among various sources of environmental pollution, the *Lancet* Commission on pollution and health has estimated that air pollution is the single most important factor contributing to greater disease burden and health risk.[1] The morbidity associated with air pollution is considerable, as is the economic cost (1.7 trillion US dollars per year in Organization for Economic Co-operation and Development countries).[2] Also, a recent airborne particle concentration-effect curve estimated that up to 8.9 million excess deaths per year occur specifically from ambient air pollution, depending on exposure duration.[3]

Air pollution is a heterogeneous mixture of particulate matter (PM), semi-volatile liquids such as benzene and formaldehyde, and gases including ozone, nitric dioxide, carbon monoxide, and sulfur dioxide, among others.[4] PM has received the most attention due to increasing levels of traffic-derived emissions across urbanizing societies worldwide, and the robust evidence supporting its role as the principal air pollutant contributing to public health risk. PM is subdivided into three size classifications by aerodynamic diameter: <0.1 μm (ultrafine particles or $PM_{0.1}$), <2.5 μm (fine particles or $PM_{2.5}$), and <10 μm (coarse particles or PM_{10}).[4] Because of their small diameter and large surface area, ultrafine particles are able to penetrate deep into the alveoli and, consequently, reach the bloodstream.[4] Although they are considered especially harmful for health owing to their wide-ranging reactive chemistry, there is no standard with which to measure ultrafine particles based on environmental monitoring system data at present; thus, there is a growing research focus on $PM_{2.5}$.

In this issue of the *Korean Circulation Journal*, Park et al.[5] report a significant correlation between $PM_{2.5}$ and cardiac arrhythmias using a mouse model. The authors artificially produced urban air pollution mostly consisting of $PM_{2.5}$ at different concentrations: control, 200 μg/m^3, 400 μg/m^3, and 800 μg/m^3. After 120 hours (8 hours/day × 5 days/week × 3 weeks) of exposure, electrophysiological changes, fibrosis or inflammation extent, and ventricular arrhythmia occurrence were examined. They report dose-dependent increases in QRS, QTc interval, action potential or intracellular Ca^{2+} duration, and inflammatory markers, as well as profound ventricular remodeling associated with cardiac fibrosis and high expression levels of oxidative stress. In addition, spontaneous ventricular tachyarrhythmia was

observed in 42% of mice exposed to concentrations of 800 μg/m^3. An important strength of the study is the concentration-stratified design used to measure the threshold level to induce ventricular arrhythmia and to investigate the arrhythmogenic mechanism mediated by real urban air pollution.

Epidemiological studies to date have indicated a significant association between $PM_{2.5}$ and the risk of cardiovascular mortality or morbidity including hypertension, ischemic heart disease, cerebrovascular disease, peripheral arterial disease, arrhythmia hospitalization, heart failure, and cardiac arrest.[6][7] In terms of ventricular tachyarrhythmia, the ARIA study reported that in patients with left ventricular dysfunction and implantable cardioverter-defibrillator, short-term exposure to $PM_{2.5}$ significantly increased the risk of ventricular tachycardia or fibrillation (odds ratio 1.59), with a greater susceptibility in patients with previous myocardial infarction.[8] Several hypotheses have been suggested for the mechanism by which inhaled particles affect the cardiovascular system: direct passage into the bloodstream, inflammatory mediators produced by the lungs, alterations in neuroendocrine activity, or increased oxidative stress of plasma proteins and lipids.[6] The current study is noteworthy in that it provided insight into more specific electrophysiological and histological mechanisms related to $PM_{2.5}$-mediated arrhythmia occurrence.

However, some weaknesses should be mentioned before applying these results to humans. First, this is a small animal study under artificially generated conditions. Second, the air quality guidelines from the World Health Organization stipulate a $PM_{2.5}$ level less than 10 μg/m^3 annually or 25 μg/m^3 daily,[2] and the concentrations used in the study greatly exceeded these values. However, these findings remain a concern. Most prior studies presented concentration-dependent increases in cardiovascular risk, with excess risk even at or below the range of current standard values.[9]

Thus, improving air conditions to reduce cardiovascular risk should be a priority. Beyond governmental strategies such as mandatory reductions in traffic emissions from diesel particles, shifting to lower-carbon fuels, reforming urban landscapes, and expanding public transportation,[10] personal steps to reducing exposure to ambient air pollution could include wearing face masks, using indoor air purifiers, staying at home and closing windows in areas with high levels of pollution, reducing time spent on the road, modifying lifestyle including exercise, healthy diet, and medical screening programs. However, the long-term efficacy of these personal behaviors has yet to be established.

From a clinical perspective, recognition of fine PM as an independent risk factor of cardiovascular diseases is important when developing effective policies that will improve public health.

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