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EDITORIAL

Particulate Matter and Vascular Endothelial Function: Does Aerobic Exercise Really Benefit Human Cardiovascular Health?

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Air pollution has recently become a global public health and social issue. Mortality rates from cardiovascular and chronic lung disease are steadily increasing [1,2]. PM2.5 (particulate matter with aerodynamic diameter ≤2.5 μm) and PM10 (particulate matter with aerodynamic diameter ≤10 μm) has been attracting attention as major air pollutants, and they are closely related to both prevalence and mortality rate of cardiovascular and pulmonary disease [3].

Vascular endothelial function is a clinical parameter to predict the pathological development of atherosclerosis. Atherosclerosis is established as the principal contributor to cardiovascular disease mortality. Both PM2.5 and PM10 inhaled into the human body through the pulmonary system increase oxidative stress and inflammation level in the tissues of the pulmonary system. Pro-inflammatory cytokines activated in the pulmonary system enter into the cardiovascular system, which subsequently increases the level of oxidative stress and inflammation in the vascular tissues [4]. In particular, PM2.5 smaller than PM10 in size is known to increase the inflammation level in vascular endothelial cells and promotes the production and secretion of endothelin-1, an intrinsic vasoconstrictor, because it is easy to enter into the vascular tissue through the lungs. Increase in oxidative stress and inflammation level and endothelin-1 concentration in the vascular endothelium leads to the dysfunction of endothelial nitric oxide synthase that produces nitric oxide (NO) in vascular endothelial cells. It is established that the reduction in NO production and bioavailability causes vascular endothelial dysfunction.

Regular aerobic exercise leads to structural and functional improvement of the cardiovascular system regardless of gender and age. Increased cardiopulmonary fitness reduces the risk of cardiovascular disease and its mortality. Regular aerobic exercise improves vascular endothelial function by reducing pro-inflammatory cytokines and increasing NO production and bioavailability [5]. The increase in NO production and bioavailability in vascular endothelium induces an increase in endogenous antioxidants and a decrease in nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, which facilitates the production of reactive oxygen species that cause oxidative stress [6]. Additionally, long-term aerobic exercise prevents pathological hypertrophy of vascular smooth muscle and decreases the secretion of endothelin-1 which inhibits eNOS activity.

There is a growing interest if the positive effects of aerobic exercise on vascular endothelial function can be maintained or preserved when aerobic exercise is performed in an elevated PM2.5 environment that causes vascular endothelial dysfunction. Research studies evaluating the effect of aerobic exercise on human vascular endothelial function at high PM environment are still very scarce, and the study results are also controversial. Some studies have shown that just one-bout of aerobic exercise at high PM condition impairs vascular endothelial function even in healthy young adults and the impaired function can last up to 24 hours after the exercise at the environment with PM2.5 or smaller PM particles (aerodynamic diameter ≤1.0 μm) [7-10]. On the other hand, a previous study has presented that there is no effect on vascular endothelial function [11]. Because it is unethical to experimentally expose humans under high PM conditions for long periods of time, no studies have investigated the effects of long-term aerobic exercise in a high PM environment on human vascular endothelial function.

In summary, any types of aerobic exercise, which substantially increases minute ventilation at high PM concentrations, have the potential

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to temporarily impair vascular endothelial function, so it may be advisable for the vulnerable populations such as the elderly and those with chronic diseases to refrain from doing it. More clinical studies in humans are needed to establish systematic guidelines for maintaining and promoting cardiovascular health while exercising safely, even at high PM concentrations.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest.

AUTHOR CONTRIBUTIONS

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