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Acute Moderate-Intensity Aerobic Exercise under High $PM_{2.5}$ Levels Does Not Influence the Pulmonary Function and Lung Diffusion Capacity in Healthy Young Men

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Abstract: Exposure to fine particulate matter ($PM_{2.5}$) impairs the respiratory system and increases the morbidity and mortality of respiratory diseases. Even though aerobic exercise is known to improve pulmonary function in diverse populations, it can lead to an increase in the inhalation of $PM_{2.5}$ in polluted environments. We aimed to investigate the effects of aerobic exercise under high $PM_{2.5}$ conditions on the pulmonary function in young adults. Nine healthy young men performed indoor treadmill running in both high and low $PM_{2.5}$ conditions (59.0 $\pm$ 2.1 vs. 7.8 $\pm$ 1.0 $\mu g/m^3$) by a crossover study design. Pulmonary function was assessed by spirometry and diffusing capacity for carbon monoxide (DLCO) at pre- and 1-h post-exercise. There was no difference in the response of pulmonary function and lung diffusion capacity to the acute aerobic exercise in high and low $PM_{2.5}$ conditions ($p \geq 0.09$). These findings indicate that aerobic exercise in high $PM_{2.5}$ conditions may not adversely affect pulmonary function in healthy young adults.

Keywords: fine particulate matter; aerobic exercise; pulmonary function; lung diffusion capacity

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1. Introduction

Exposure to air pollution is widely known to increase all-cause morbidity and mortality. Particulate matter (PM), one of the constituents of air pollution, adversely affects the human cardiorespiratory system, and as a result, the incident rate of cardiopulmonary complications and their mortality can increase with augmented PM levels [1–4]. Due to its harmful influence on human health and wellbeing, the International Agency for Research on Cancer (IARC) categorized outdoor air pollution and PM as a carcinogen to humans [5]. In an outdoor environment, PM is mainly derived from traffic output, industrial activities, and biomass burning [6]. Furthermore, particles from the outdoor environment can rapidly penetrate the indoor environment, making people inevitably suffer from PM exposure in their daily lives [7].

PM is mainly classified as PM_{10}, $PM_{2.5}$, and PM_1 according to the particles' aerodynamic diameter (PM $\leq$ 10 μm, 2.5 μm, and 1 μm in diameter, respectively), and its effects on the cardiorespiratory system are determined by its size. PM_{10} can only penetrate and deposit in the upper respiratory tract [8]. However, $PM_{2.5}$ can seep into the alveolar region, the deepest part of the lung, which can trigger negative influences on the whole respiratory system [8,9]. Short-term exposure to $PM_{2.5}$ impairs the respiratory tract by inducing inflammation and oxidative stress, which may induce bronchoconstriction and temporarily decrease pulmonary function [10]. Furthermore, long-term exposure to $PM_{2.5}$

engenders morphological changes in nasal airways and increases the risk of respiratory diseases [10].

Aerobic exercise is known to improve pulmonary function in asthmatic patients [11]. Even in healthy individuals, pulmonary function was enhanced after participating in regular aerobic exercise [12–14]. However, performing aerobic exercise under high $PM_{2.5}$ conditions might annul the positive effect of the exercise and impair the respiratory system. Substantially increased minute ventilation during aerobic exercise is likely to increase the number of inhaled particles into the lungs [15,16]. In addition, increased velocity of airflow during exercise can cause particles to reach a deeper region of the respiratory system [15]. Previous findings on the effect of aerobic exercise on pulmonary function in high $PM_{2.5}$ conditions are limited, and their results are still controversial [17–21]. Hence, this study aimed to demonstrate the effect of acute moderate-intensity aerobic exercise on pulmonary function under high $PM_{2.5}$ conditions compared with low $PM_{2.5}$ conditions. We hypothesized that exposure to high $PM_{2.5}$ conditions during aerobic exercise would negatively affect pulmonary function due to the elevated $PM_{2.5}$ inhalation caused by the increased minute ventilation during exercise. Moreover, our study controlled indoor $PM_{2.5}$ conditions with penetrated particles from the outdoor environment to emphasize that the influx of outside ambient air has a negative effect on indoor air quality.

2. Materials and Methods

2.1. Participants

Nine healthy young men (24.6 $\pm$ 0.4 years) participated in this study and they were recruited by word of mouth at Incheon National University. All participants were free of clinical diseases and non-smokers with normal lung function - over 80% of predicted forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1) for sex, age, height, and ethnicity [22]. Participants were excluded if they were taking any medications that might affect cardiovascular and pulmonary function, and if they have or had a history of musculoskeletal, cardiovascular, or respiratory diseases. We confirmed via interview that all participants were recreationally active before initiating the study. This study observed the ethical standards of the Declaration of Helsinki. The purpose and risks of the study were fully explained to each participant, and they voluntarily signed an informed consent form before participating in the study. This study was reviewed and approved by the Institutional Review Board of Incheon National University.

2.2. Study Design

This study used a crossover design and all participants visited the laboratory three times. In the initial visit, the participants performed a maximal graded exercise test with a modified Bruce protocol to assess their maximal oxygen consumption (VO_2max) and heart rate (HRmax) [23]. Since not all participants met at least three of the following criteria: (1) a heart rate within 10 bpm of age-predicted max heart rate (220-age); (2) a score of at least 18 on the Borg rating of perceived exertion scale; (3) above 1.15 of maximal respiratory exchange ratio; (4) a plateau of oxygen consumption with increasing exercise intensity; we defined the VO_2max as peak oxygen consumption (VO_2peak) and HRmax as peak heart rate (HRpeak). In the second and third visits, the participants performed acute moderate-intensity aerobic exercise in high and low $PM_{2.5}$ conditions in an indoor training facility. A randomized crossover study design could not be employed because the decision as to whether or not to run the planned experiments on the same day depended on the external weather conditions. But luckily, four of nine participants executed their second visit in low $PM_{2.5}$ conditions and five participants completed the second visit in high $PM_{2.5}$ conditions. We measured clinical pulmonary function parameters before and 1-h after an established aerobic exercise session in each condition. In both second and third visits, we used air purifiers with high-efficiency particulate absorbing (HEPA) filters to minimize the $PM_{2.5}$ exposure in the laboratory during pulmonary function measures at pre- and post-exercise intervention. The aerobic exercise intervention consisted of 30 min of treadmill running at

70% of HRpeak. Heart rate was measured by Polar H7 (Polar Electro Oy, Kempele, Finland) devices and was continuously monitored by using the Polar Team App (version 1.8.8, Polar Electro Oy, Kempele, Finland) during the exercise intervention. Participants were asked to refrain from alcohol consumption and any moderate to vigorous physical activity for at least 24 h and to fast for 12 h prior to the second and third visits. To avoid any confounding effect of aerobic exercise and $PM_{2.5}$ inhalation between the second and third visits, the participants had at least 7 days of wash-out period.

2.3. $PM_{2.5}$ Concentration

Concomitant outdoor and indoor $PM_{2.5}$ concentrations were continuously monitored during the exercise intervention by using a light-scattering laser photometer device (SIDEPAK^{TM} AM520 Personal Aerosol Monitor, TSI Ltd., Shoreview, MN, USA). The light-scattering laser photometers were placed at the same spots throughout the research. $PM_{2.5}$ concentrations were averaged every minute with continuously recorded values during the exercise intervention.

2.4. Height, Body Mass and Body Composition

Height was measured to the nearest 0.1 cm by a conventional stadiometer and body mass was measured with an Inbody 720 Scale (Biospace, Seoul, Korea). Body composition was measured and assessed by dual-energy X-ray absorptiometry (Prodigy, GE Healthcare, Waukesha, WI, USA). Body mass index (BMI) was calculated using the following equation; body mass (kg)/height^2 (m^2). All anthropometric measurements were performed before the exercise intervention in the second and third visits.

2.5. Pulmonary Function

Spirometry and DLCO were measured to assess the basic pulmonary function parameters and the lung's capability to transfer the inhaled gas to the alveolar capillary vessels by using a Quark PFT system connected with its respiratory chamber (COSMED, Rome, Italy). The same research staff continuously monitored the participant's mouthpiece (Pro-guard EX, GMS KOREA, Suwon, Korea) and nose clip position to ensure that there was no gas leakage during the test. The spirometry measurement was performed to assess FVC, FEV_1, FEV_1/FVC, and forced expiratory flow between 25% and 75% of the FVC ($FEF_{25–75}$) in a standing position following the American Thoracic Society (ATS) guidelines [24]. The DLCO was measured using a gas containing 0.3% CO, 0.3% CH_4, 20.85% O_2, and Bal N_2 while sitting on a chair in the respiratory chamber. The participants exhaled fully before maximal gas inhalation and then held their breath for 10 s before exhaling the gas normally during the DLCO procedure. In our laboratory, the day-to-day coefficient of variation for spirometry variables were lower than 0.9% and DLCO variables were less than 3.8%.

2.6. Statistical Analysis

All statistical analyses were performed with SPSS version 27 (IBM SPSS Inc., New York, NY, USA). Data are presented as mean ± standard error. We confirmed the normality of data by using the Shapiro–Wilk test. We used a paired *t*-test to confirm whether there were changes in the body composition and pulmonary function at pre-exercise intervention between the second and third visits. The Mann–Whitney U test was used to compare the outdoor and indoor $PM_{2.5}$ concentrations between high and low $PM_{2.5}$ conditions. We employed the analysis of variance (ANOVA) with repeated measures to demonstrate the $PM_{2.5}$ conditions (high vs. low) × time (pre- vs. post-exercise) interaction effect on pulmonary function. All statistical significance was set as $p < 0.05$.

3. Results

3.1. Participants' Characteristics and $PM_{2.5}$ Concentration

The characteristics of participants are shown in Table 1. They were all healthy young men (n = 9; 24.6 ± 0.4 years) without abnormal lung function.

Table 1. Participant's characteristics.

Group	Participants (n = 9)
Age (years)	24.6 ± 0.4
Height (cm)	177.4 ± 1.5
Body mass (kg)	77.9 ± 1.6
BMI (kg/m^2)	24.8 ± 0.6
VO_2peak (mL/kg/min)	55.0 ± 18.3

Note. Data are mean ± standard error. BMI, body mass index; VO_2peak, peak oxygen consumption.

Outdoor and indoor $PM_{2.5}$ concentrations in high $PM_{2.5}$ conditions were significantly higher than those in low $PM_{2.5}$ conditions during the aerobic exercise session ($p < 0.001$; Table 2). According to the air quality index (AQI) issued by Environmental Protection Agency (EPA), the concentration of indoor $PM_{2.5}$ was categorized as 'good' in low $PM_{2.5}$ conditions and 'unhealthy for sensitive groups or unhealthy' in high $PM_{2.5}$ conditions during exercise. Additionally, indoor $PM_{2.5}$ concentration in high $PM_{2.5}$ conditions was higher than the recommended U.S. National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$ of 35 μg/m^3 (mean: 59 μg/m^3, range: 50–73 μg/m^3). There were no adverse events during or after the exercise interventions in both high and low $PM_{2.5}$ conditions.

Table 2. Outdoor and indoor $PM_{2.5}$ concentrations during exercise intervention.

	HPM	LPM
Outdoor $PM_{2.5}$ concentration (μg/m^3)	150.9 ± 27.1 (71, 280)	17.6 ± 4.7 (5, 40) *
Indoor $PM_{2.5}$ concentration (μg/m^3)	59.0 ± 2.1 (50, 73)	7.8 ± 1.0 (5, 13) *

Note. Data are mean ± standard error (minimum, maximum). HPM, high $PM_{2.5}$ condition; LPM, low $PM_{2.5}$ condition. * $p < 0.001$ vs. HPM.

3.2. Pulmonary Function

Performing acute moderate-intensity aerobic exercise did not have any notable difference in FVC, FEV_1, FEV_1/FVC, and $FEF_{25–75}$ between high and low $PM_{2.5}$ conditions ($p \geq 0.24$; Table 3, Figure 1). Additionally, there was no effect of high $PM_{2.5}$ conditions on DLCO and DLCO/VA in response to acute moderate-intensity aerobic exercise compared with low $PM_{2.5}$ conditions ($p \geq 0.09$; Table 3, Figure 2).

Table 3. Pulmonary function measures at pre- and post-exercise intervention.

	HPM		LPM	
	Pre	Post	Pre	Post
FVC (L)	5.22 ± 0.16	5.23 ± 0.15	5.27 ± 0.19	5.30 ± 0.18
(% predicted)	106.67 ± 2.84	106.78 ± 2.84	107.44 ± 3.22	108.22 ± 3.20
FEV_1 (L)	4.47 ± 0.13	4.43 ± 0.16	4.49 ± 0.13	4.58 ± 0.13
(% predicted)	106.22 ± 3.08	105.33 ± 3.33	106.56 ± 3.12	108.67 ± 3.30
FEV_1/FVC (%)	86.01 ± 2.49	85.16 ± 3.68	85.76 ± 2.44	86.76 ± 2.39
(% predicted)	99.33 ± 2.81	98.44 ± 4.14	98.89 ± 2.61	100.00 ± 2.60
$FEF_{25–75}$ (%)	5.01 ± 0.43	4.94 ± 0.47	4.96 ± 0.38	5.21 ± 0.42
(% predicted)	105.33 ± 9.28	101.56 ± 9.85	104.00 ± 8.30	109.00 ± 9.19
DLCO (mL/min/mmHg)	36.41 ± 1.66	35.05 ± 1.83	35.66 ± 1.64	34.94 ± 1.70
(% predicted)	101.44 ± 4.11	97.56 ± 4.50	99.33 ± 4.53	97.33 ± 4.46
DLCO/VA (mL/min/mmHg/L)	5.40 ± 0.12	5.24 ± 0.13	5.25 ± 0.13	5.02 ± 0.11
(% predicted)	104.56 ± 2.27	101.44 ± 2.56	101.44 ± 2.29	97.33 ± 2.11

Note. Data are mean ± standard error. HPM, high $PM_{2.5}$ condition; LPM, low $PM_{2.5}$ condition; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of the FVC; DLCO, diffusing capacity for carbon monoxide; DLCO/VA, DLCO corrected by alveolar volume.

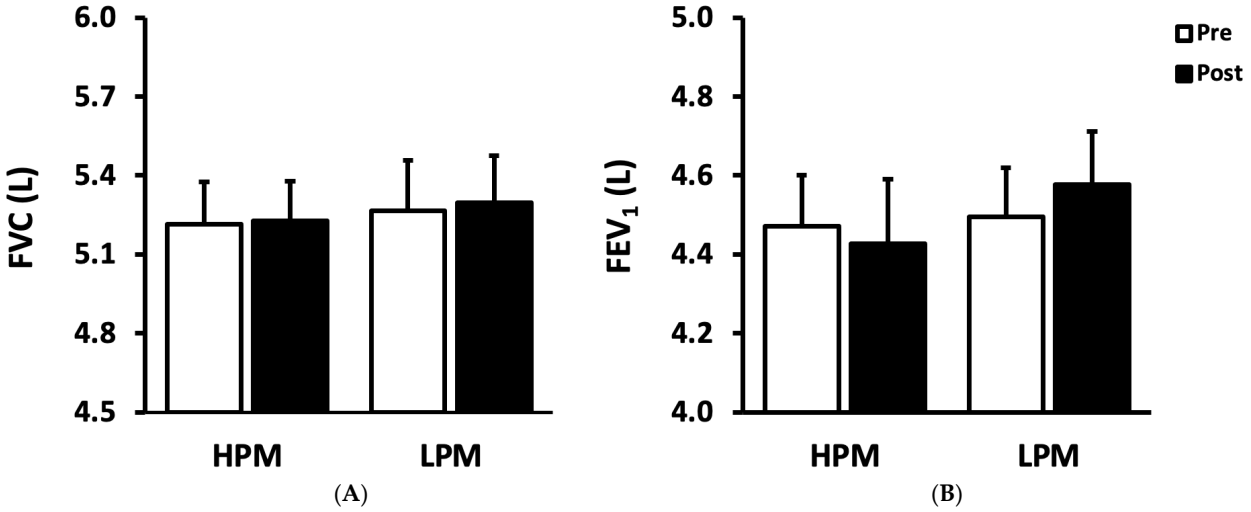

Figure 1. FVC (panel **A**) and FEV_1 (panel **B**) at pre- and post-exercise intervention. Data are mean ± standard error. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; HPM, high $PM_{2.5}$ condition; LPM, low $PM_{2.5}$ condition. $p \geq 0.50$ for time-condition interaction.

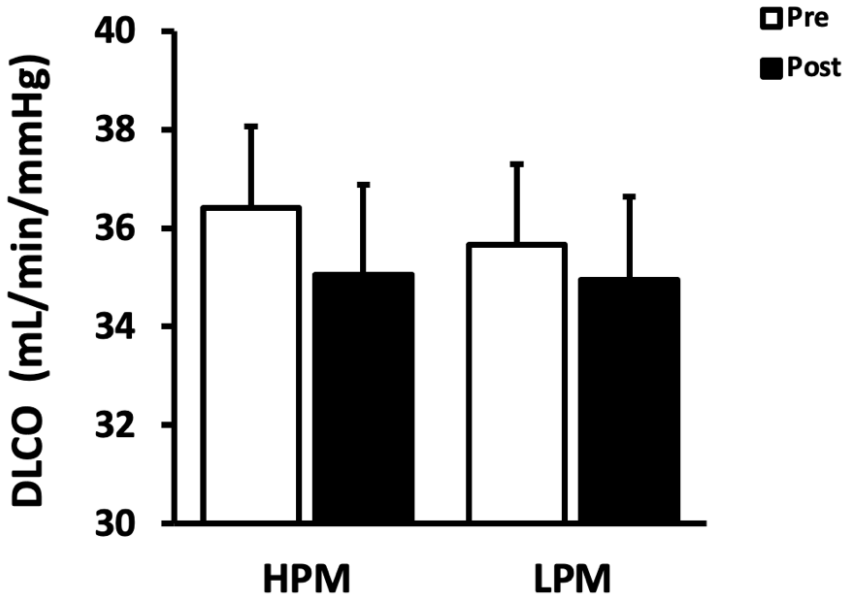

Figure 2. DLCO at pre- and post-exercise intervention. Data are mean ± standard error. DLCO, diffusing capacity of carbon monoxide; HPM, high $PM_{2.5}$ condition; LPM, low $PM_{2.5}$ condition. $p = 0.09$ for time-condition interaction.

4. Discussion

This study examined the effect of acute moderate-intensity aerobic exercise, which might substantially increase in the inhalation of $PM_{2.5}$ particles, on the pulmonary function and lung diffusion capacity under high $PM_{2.5}$ conditions compared with low $PM_{2.5}$ conditions. To our knowledge, this is the first study to assess changes in DLCO with conventional pulmonary function parameters in response to aerobic exercise interventions in high $PM_{2.5}$ conditions. Our results imply that performing moderate-intensity aerobic exercise even in high $PM_{2.5}$ concentrations categorized as 'unhealthy for sensitive groups or unhealthy' level from AQI might not play a negative role on pulmonary function in healthy young men.

Spirometry is a pulmonary function test that assesses the volume and flow rate of air during inhalation and/or exhalation. FVC and FEV_1, the main results from spirometry, are

helpful in distinguishing obstructive and restrictive respiratory diseases and categorizing the severity of asthma and chronic obstructive pulmonary disease (COPD). Inhalation of $PM_{2.5}$ can impair the respiratory system by irritating the whole airway and inducing oxidative stress and inflammation [21,25–29]. Oxidative stress and inflammation may cause mucus hypersecretion and contraction of the airway smooth muscle, increasing airway resistance [29–31]. Moreover, long-term $PM_{2.5}$ exposure is known to increase the risk of respiratory diseases, COPD, pneumonia, and lung cancer mortality [32]. However, even though aerobic exercise induces increased inhalation of $PM_{2.5}$, we could not find any changes in pulmonary function after exercise in high $PM_{2.5}$ conditions. Previous studies have reported inconsistent findings on the effects of exercise on pulmonary function in polluted environments [17–21,33,34]. It is speculated that the inconsistent findings resulted from studies' designs and the methodological differences among them; the source of air pollutants, exposure time, exercise type, exercise intensity, and time point of measuring pulmonary function varied. Moreover, our participants' cardiorespiratory fitness might nullify the negative effect of $PM_{2.5}$ on pulmonary function. They were recreationally active men with high VO_2peak values, which can be defined as an excellent group according to the American College of Sports Medicine (ACSM) fitness classification [35]. VO_2peak is negatively correlated with oxidative stress and inflammation, and positively related to total antioxidant capacity [36,37]. Therefore, an increased cardiorespiratory fitness level might protect the pulmonary function against oxidation stress and inflammation caused by $PM_{2.5}$.

We did not observe any changes in DLCO after acute moderate-intensity aerobic exercise in high $PM_{2.5}$ conditions. In contrast to our findings, a previous study showed a decrease in DLCO after exposure to 50 $\mu g/m^3$ of carbon ultrafine particles for two hours [38]. Pietropaoli et al. suggested that pulmonary gas diffusing capacity can be reduced owing to PM-induced pulmonary vasoconstriction [38]. Pulmonary endothelial dysfunction caused by exposure to high $PM_{2.5}$ levels is considered a probable physiological mechanism for pulmonary vasoconstriction [39,40]. In a rodent model, the lumen to wall ratio in small pulmonary arteries was also reduced after exposure to ambient pollutants [41]. Regarding DLCO results, the reason for the differences between this study and the previous findings is also thought to be because of discrepancies in the experiment settings, major pollutants, pollutant exposure time, and post-exposure measurement time. Thus, the lack of related studies and unclear physiological mechanisms needs to be supplemented through well-designed future studies.

In the present study, we did not find any significant difference in pulmonary function and DLCO response to exercise in high and low $PM_{2.5}$ conditions. This result implies that high levels of $PM_{2.5}$ do not impair lung airways and pulmonary function in recreationally active healthy young individuals. However, these results should not be applied to older adults and patients with acute or chronic respiratory diseases because their lung airways are likely to be obstructed by high levels of $PM_{2.5}$ exposure. This study was conducted in an indoor training facility where the ambient $PM_{2.5}$ level changes every day according to outside ambient $PM_{2.5}$ concentration. Unlike laboratory settings in which only diesel exhaust gas is exposed in a chamber, the experimental design of this study may have the advantage that the local characteristics of real world air pollutants are reflected. Moreover, our study's data presenting the difference between the indoor and outdoor average $PM_{2.5}$ concentrations can be helpful in establishing guidelines that help those who want to exercise safely in an indoor facility by referring to real-time outdoor air quality data provided by the country.

Despite this study's strengths, we admit there were several limitations in this study. First, contrary to our study plan, the difference in the wash-out period between the second and third visits varied among study participants because we scheduled them based on the weather forecast and real-time measurements of $PM_{2.5}$ early in the morning of each visit. However, we asked our participants to keep their normal lifestyle throughout the study participation and confirmed that there was no difference in their body composition and pulmonary function parameters between the second and third visits. Second, even

though we asked participants to wear masks to minimize $PM_{2.5}$ exposure on the way to our laboratory, they might be unintentionally exposed to different $PM_{2.5}$ conditions because of their different residences, distances from the laboratory, and the way in which they reached the laboratory. In addition, our findings cannot be generalized to other groups of people who have different age, gender, place of residence, health status, or physical activity levels because the sample size was small and all participants were recreationally active and healthy young men. Third, even if the exercise intervention in our study followed the ACSM's guideline [42], individuals might have different responses to the exercise and $PM_{2.5}$ exposure depending on their lactate threshold. If the intensity of exercise intervention were above the lactate threshold, the participant would have increased ventilation due to lactate acid buffering and increased metabolism, causing an increase in $PM_{2.5}$ inhalation. Fourth, since participants were exposed to the indoor ambient air penetrated from the outdoor environment, we could not maintain a constant $PM_{2.5}$ concentration during the exercise and control the potential unexpected influence of other ambient gaseous pollutants, such as ozone, nitrogen dioxide, black carbon, and so on. Additionally, we admit that it would be very hard for this study to be replicated by future studies with the same concentrations and constituents of $PM_{2.5}$ due to differences in regional $PM_{2.5}$ conditions. However, future studies with similar study designs with our protocol can be conducted under similar $PM_{2.5}$ concentrations in indoor facilities or environmental chambers using naturally formed $PM_{2.5}$ or diesel exhaust. Lastly, this study had also planned to test the feasibility of whether we could conduct this type of functional study in natural, ambient settings without artificial manipulation of the environmental conditions. Thus, we could not get an insight into the physiological mechanisms involved in our new findings.

5. Conclusions

In conclusion, performing acute moderate-intensity aerobic exercise under high $PM_{2.5}$ levels may not negatively affect pulmonary function and lung diffusion capacity in healthy young adults. However, these findings must be cautiously interpreted due to the small sample size and participants' characteristics. Even though increased $PM_{2.5}$ inhalation during aerobic exercise did not influence pulmonary function and lung diffusion capacity in recreationally active healthy young men, it might cause clinical or sub-clinical consequences in other populations more susceptible to air pollutants.

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