ULTRAFINE AND FINE PARTICULATE MATTER INHALATION DECREASES EXERCISE PERFORMANCE IN HEALTHY SUBJECTS

KENNETH W. RUNDELL AND RENEE CAVISTON
Human Performance Laboratory, Marywood University, Scranton, Pennsylvania

ABSTRACT
The purpose of this study was to investigate effects of PM1 (particulate matter with aerodynamic diameter 0.02-1 μm) inhalation on exercise performance in healthy subjects. Inhalation of internal combustion-derived PM is associated with adverse effects to the pulmonary and muscle microcirculation. No data are available concerning air pollution and exercise performance. Fifteen healthy college-aged males performed 4 maximal effort 6-min cycle ergometer trials while breathing low or high PM1 to achieve maximal work accumulation (kJ). Low PM1 inhalation trials 1 and 2 were separated by 3 days; then after a 7 day washout, trials 3 and 4 (separated by 3 days) were done while breathing high PM1 generated from a gasoline engine; CO was kept below 10 ppm. Lung function was done after trial 1 to verify nonasthmatic status. Lung function was normal before and after low PM1 exercise. PM1 number counts were not different between high PM1 trials (336,730 ± 149,206 and 396,200 ± 82,564 for trial 3 and 4, respectively) and were different from low PM1 trial number counts (2,260 ± 500) (P < 0.0001). Mean heart rate was not different between trials (189 ± 6.0, 188 ± 7.6, 187 ± 7.4, for low and high PM1 trials; respectively). Work accumulated was not different between low PM1 trials (96.1 ± 9.38 versus 96.8 ± 10.83 kJ) and the first high PM1 trial (trial 3, 96.8 ± 10.85 kJ). Work accumulated in the second high PM1 trial (91.3 ± 10.04 kJ) was less than in low PM1 trials 1 and 2, and high PM1 trial 3 (P = 0.004, P = 0.003, P = 0.0008; respectively). Acute inhalation of high (PM1) typical of many urban environments could impair exercise performance.

KEY WORDS air pollution, cycle ergometry, time, work

INTRODUCTION
Increased hospital admissions for heart failure, ischemic heart disease, and ischemic stroke are associated with acute airborne particulate matter (PM) exposure (4,6,14). Inhalation of PM during exercise has been shown to cause decreased heart rate variability (7,15) and ST-segment depression in patients with coronary heart disease (13). However, no information concerning effects of PM inhalation on exercise performance in healthy individuals exists.

Inhalation of PM causes a systemic inflammatory response; Ulrich et al. (23) reported a 4-fold increase in tumor necrosis factor alpha (TNF-α) in bronchial lavage fluid and a 2-fold increase in plasma endothelin-1 (ET-1) in PM exposed rats. Since ET-1 is a vasoconstrictor peptide, increased plasma ET-1 could affect muscle performance by compromising oxygen delivery through muscle arterioles.

Diesel exhaust PM suppresses endothelium-dependent vasodilation via inhibition of nitric oxide (NO) release (8), and Cheng and Kang (3) found impaired vasorelaxation induced by acetylcholine from motorcycle gas engine exhaust particles. In a study using healthy human subjects, short-term inhalation of concentrated ambient particles plus ozone caused acute conduit artery vasoconstriction but no change in flow-mediated vasodilation (2). We (21) demonstrated reduced flow-mediated vasodilation in the brachial artery and impaired reoxygenation after cuff ischemia in the forearm microcirculation after high PM1 inhalation during exercise. Nurkiewicz et al. (9,10) found that PM exposure impaired endothelium-dependent dilation in systemic microcirculation was coincident with PMNL adhesion, myeloperoxidase (MPO) deposition, and oxidative stress. Likewise, inhalation of concentrated ambient particles has been shown causal to vasoconstriction of small pulmonary arteries in rats (1). Local arteriole endothelial dysfunction may compromise muscle reperfusion and vasoconstriction of pulmonary arterioles could reduce oxygen delivery to the working muscles and affect muscle performance.

METHODS

Experimental Approach to the Problem
In this study, we evaluated effects of PM1 inhalation during exercise on maximal performance. Four 6-min maximal cycle...
Ergometer exercise trials were performed, 2 trials under low (PM1) were followed by 2 trials under high (PM1) conditions. Trials within each condition were separated by 3 days with a 7-day washout observed between conditions. This design provided the opportunity to evaluate acute effects and residual effects of PM1 inhalation during exercise.

Subjects
Fifteen healthy nonasthmatic, nonsmoking male ice hockey players, free of cardiovascular disease (age, 19.5 ± 1.13 yrs; weight, 80.9 ± 9.10.66 kg; height, 178 ± 7.6 cm; mean ± SD) served as subjects. All subjects signed a written informed consent and completed a medical history questionnaire. This study was approved by the Marywood University Institutional Review Board. Subjects were instructed to abstain from caffeine ingestion for at least 24 hours prior to testing and were required to report to the Human Performance Laboratory for testing mid-morning 3 hours after a light, low-fat breakfast.

Procedures
Each subject performed 4 maximal effort 6-min cycle ergometer trials to achieve maximal work accumulation (kJ). 2 sequential low PM1 trials separated by 3 days (trials 1 and 2) followed by a 7-day recovery period and then 2 sequential high PM1 trials separated by 3 days (trials 3 and 4). Exercise intensity was verified using portable heart rate monitors (Polar Vantage XL, Polar Electro, Kempele, Finland). Pre and 5, 10, and 15 min postexercise lung function was measured during and after the first ergometer challenge to verify nonasthmatic status and the absence of exercise-induced bronchoconstriction, as previously done (Table 1; 21,22).

Fresh Particulate matter (PM1) was generated using a 2.5 hp gasoline fueled engine that was run for 10 s every min beginning 1 min prior to and during the high PM1 ergometer challenges. This procedure provided high (PM1) while keeping (CO) below 10 ppm. PM1 was measured during each trial as previously done (21). Measurements were made using a calibrated condensation particle counter (CPC, P-TrakTM Ultrafine Particle Counter, Model 8525, TSI inc., St. Paul, MN) at a sampling frequency of 1 Hz and recorded as 10 s means of PM1 cm⁻³. Six 10 second readings were taken throughout each exercise trial and averaged to provide the most representative particle count. The P-Trak CPC sensitivity size range is 0.02–1.0 μm diameter; this range includes ultrafine and fine PM, defined as PM1 in this study. PM1 has been shown to account for >90% of total particle count and >95% of particle surface area (μm²/cm³) for unit density mass concentration of combustion derived air samples (11, 12). Figure 1 depicts PM1 levels during low PM1 and high PM1 trials. Values for High PM1 were not different from each other (336,730 ± 149,206 and 396,200 ± 82,564 for trial 3 and 4, respectively) and were different than low PM1 trial levels (2,260 ± 500) (P < 0.0001). Although these high PM1 levels exceed most outdoor athletic facilities, we have measured levels exceeding these values at college athletic fields, elementary school playgrounds, and in ice hockey rinks (16, 18). CO was below 1.0 ppm for low (PM1) trials and 6.3 ± 3.4 ppm for high (PM1) trials (GrayWolf Direct Sense TOX, Trumbull, CT).

Statistical Analyses
Statistical analysis was done using repeated measures followed by paired t-tests (SPSS 14.0). Significance was set at P ≤ 0.05.

Results
Mean heart rates were not different between trials, verifying intensity of each ride (189 ± 6.0, 188 ± 7.6, 188 ± 7.6, 187 ± 7.4, for low and high PM1 trials; respectively). Figure 2 shows accumulated work for sequential trials. Work accumulated was not different between low PM1 (trials 1 and 2, 96.1 ± 9.38 versus 96.6 ± 10.83 kJ) and not different from high PM1 trial 3 (96.8 ± 10.65 kJ). Interestingly, work accumulated in the second high PM1 (trial 4, 91.3 ± 10.04 kJ), 3 days following the first high PM1 trial (trial 3), was significantly less than work accumulated in low PM1 trials and the first high PM1 trial (P = 0.004, P = 0.003, P = 0.0008; respectively).

Normalizing to kJ·kg⁻¹ bodyweight gave similar results. No significant correlations were identified between PM1 concentrations and accumulated work either as absolute values for all trials or as differences in PM1 values and work between high PM1 trials.

Table 1. Lung function before and after trial 1, a 6 min low PM1 inhalation high intensity cycle ergometer challenge. All subjects demonstrated normal resting lung function and were negative for EIB. N = 15.

<table>
<thead>
<tr>
<th></th>
<th>FVC (L)</th>
<th>Percent</th>
<th>FVC % fall</th>
<th>FEV1 (L)</th>
<th>Percent</th>
<th>FEV1 % fall</th>
<th>FEF25–75 (L·s⁻¹)</th>
<th>Percent</th>
<th>FEF25–75 % fall</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline</td>
<td>MN 5.82</td>
<td>104.4</td>
<td>−4.55</td>
<td>4.77</td>
<td>109.5</td>
<td>−2.14</td>
<td>5.12</td>
<td>103.4</td>
<td>−2.49</td>
</tr>
<tr>
<td></td>
<td>SD 0.793</td>
<td>10.55</td>
<td>3.902</td>
<td>0.855</td>
<td>15.94</td>
<td>3.556</td>
<td>1.707</td>
<td>32.97</td>
<td>6.444</td>
</tr>
</tbody>
</table>
DISCUSSION

This study was designed to determine whether PM₁ inhalation affected exercise performance; a mechanism for our observation of decreased performance after the second of 2 (PM₁) high intensity exercise bouts (but not after the first) can only be speculative from this data. The altered performance was not likely the result of an order effect, since two low (PM₁) trials were performed one week prior to the high (PM₁) trials and work accumulated was not different between the first high (PM₁) trial and the two low (PM₁) trials. And it is not likely that the decreased performance on the last trial was because of a uniform lack of motivation in our subjects; the subjects were highly-motivated competitive athletes and were verbally encouraged throughout each trial. Instead, these results support a response initiated from the first high (PM₁) exposure trial that affected muscle perfusion in the second high (PM₁) trial 3 days later.

We previously reported diminished flow-mediated vasodilation (FMD) after 30 min of running while breathing high (PM₁), suggesting a PM₁-induced disruption of normal endothelial-mediated vasodilation (19). The near abolition of FMD was accompanied by a 55% decrease in muscle reoxygenation slope-to-baseline measured by near-infrared spectroscopy (NIRS) after release of artery occlusion and is consistent with compromised reperfusion. This study (19) supported a constrictive response from PM₁ inhalation in the microvasculature that was independent of the observed brachial artery vasoconstriction. Calculation of the return slope of reoxygenation (µM s⁻¹) to baseline after cuff ischemia (19) provided a rate of decreased reperfusion of the muscle tissue (after 30 minute exercise breathing high PM₁) that should be proportional to blood flow. We also identified a 4% vasoconstriction of the brachial artery after exercise while breathing high (PM₁) ambient air that was consistent with the ~2.6% vasoconstriction reported by Brook et al. (2). Although it is unlikely that constriction of the large vessels could impact delivery to the exercising muscles, the decreased reperfusion in the microcirculation supports a physiologically significant decrease in blood flow that could affect exercise performance.

Whether vasoconstriction of the pulmonary arterioles or vasoconstriction of local arterioles of the exercising muscle, or both, was causal to the observed decrease in performance can only be speculated. Vasoconstriction of small pulmonary arteries after short-term inhalation exposure to concentrated ambient particles has been shown in normal rats (1). If even mild pulmonary arteriole vasoconstriction occurs in humans from high PM₁ exposure exercise, a subsequent bout of exercise could exaggerate the mild pulmonary hypertension and cause decreased oxygen delivery to the working muscle; exercise has been shown to amplify moderate pulmonary hypertension (5). Alternatively, vasoconstriction of the microcirculation in the exercising muscle could impact exercise performance. Nurkiewicz et al. (9) found that residual oil fly ash (ROFA) abolished both NO-dependent and NO-independent systemic arteriolar dilation in the spinotrapezius muscle of rats; approximately 50% of the arteriole vasoresponsiveness was due to NO-dependent factors. More recently, Nurkiewicz et al. (10) identified increased myeloperoxidase and oxidative stress in the microvasculature of the spinotrapezius muscle of ROFA exposed rats, supporting the notion that local arteriole vasoconstriction could compromise oxygen delivery to the working muscle.

We cannot definitively say whether the observed effects on performance during the second high PM₁ exercise bout resulted solely from the first high PM₁ exposure exercise; however, the data suggest that performance for a short duration exercise bout while inhaling PM₁ typical of high traffic areas or ice arenas does not immediately affect performance. We found that performance was affected 3 days...
after the initial exposure, suggesting that either the pulmonary system and/or the microvascular system were affected from the first high PM₁₀ exposure exercise. Future studies should explore effects of PM₁₀ inhalation during exercise on muscle microcircular and pulmonary circulation and define the time course of post-inhalation events leading to and following performance decrements.

**Practical Applications**

The results from this preliminary observation would suggest that one should train in a low pollution environment, especially when leading into competition. Although multiple low-dose exposures of polytetrafluoroethylene particles have been shown to provide a tolerance that protected against a subsequent high dose exposure in the rat model, an inflammatory response was initiated (12). No tolerance from fossil fuel particle inhalation has yet been described in humans, and thus may not be germane to performance-related issues described in this paper.

The results in this paper support the notion that proximity to busy highways should be considered in the location and design of new outdoor athletic facilities. Although particle levels at existing athletic fields close to high traffic roadways could be reduced by screening with coniferous trees. We have previously shown levels of PM₁₀ in the range of the high PM₁₀ used in this study at college soccer fields and elementary athletic fields in close proximity to high traffic roadways (20). In that study, we also showed an exponential decay in particle number count such that 200 m from the roadway source counts were reduced 4-fold. Finally, the indoor ice arena can potentially have hazardous levels of PM₁₀ emitted from fossil fuel particle inhalation has yet been described in humans, and thus may not be germane to performance-related issues described in this paper.

**References**


