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

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## ABSTRACT

The purpose of this study was to investigate effects of PM<sub>1</sub> (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 PM<sub>1</sub> to achieve maximal work accumulation (kJ). Low PM<sub>1</sub> 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 PM<sub>1</sub> 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 PM<sub>1</sub> exercise. PM<sub>1</sub> number counts were not different between high PM<sub>1</sub> trials (336,730 ± 149,206 and 396,200 ± 82,564 for trial 3 and 4, respectively) and were different from low PM<sub>1</sub> trial number counts (2,260 ± 500) ( $P < 0.0001$ ). Mean heart rate was not different between trials (189 ± 6.0, 188 ± 7.6, 188 ± 7.6, 187 ± 7.4, for low and high PM<sub>1</sub> trials; respectively). Work accumulated was not different between low PM<sub>1</sub> trials (96.1 ± 9.38 versus 96.6 ± 10.83 kJ) and the first high PM<sub>1</sub> trial (trial 3, 96.8 ± 10.65 kJ). Work accumulated in the second high PM<sub>1</sub> trial 4, 91.3 ± 10.04 kJ) was less than in low PM<sub>1</sub> trials 1 and 2, and high PM<sub>1</sub> trial 3 ( $P = 0.004$ ,  $P = 0.003$ ,  $P = 0.0008$ ; respectively). Acute inhalation of high (PM<sub>1</sub>) typical of many urban environments could impair exercise performance.

**KEY WORDS** air pollution, cycle ergometry, time, work

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## INTRODUCTION

**I**ncreased 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 PM<sub>1</sub> 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 PM<sub>1</sub> inhalation during exercise on maximal performance. Four 6-min maximal cycle

ergometer exercise trials were performed, 2 trials under low (PM<sub>1</sub>) were followed by 2 trials under high (PM<sub>1</sub>) 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 PM<sub>1</sub> 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 PM<sub>1</sub> trials separated by 3 days (trials 1 and 2) followed by a 7-day recovery period and then 2 sequential high PM<sub>1</sub> 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 (PM<sub>1</sub>) 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 PM<sub>1</sub> ergometer challenges. This procedure provided high (PM<sub>1</sub>) while keeping (CO) below 10 ppm. PM<sub>1</sub> was measured during each trial as previously done (21). Measurements were made using a calibrated condensation particle counter (CPC, P-Trak™ Ultrafine Particle Counter, Model 8525, TSI inc., St. Paul, MN) at a sampling frequency of 1 Hz and recorded as 10 s means of PM<sub>1</sub>·cm<sup>-3</sup>. 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 PM<sub>1</sub> in this study. PM<sub>1</sub> has been shown to account for >90% of total particle count and >95% of particle surface area (μm<sup>2</sup>/cm<sup>3</sup>) for unit density mass concentration of combustion derived air samples (11, 12). Figure 1 depicts PM<sub>1</sub> levels during low PM<sub>1</sub> and high PM<sub>1</sub> trials. Values for High PM<sub>1</sub> 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 PM<sub>1</sub> trial levels (2,260 ± 500) (P < 0.0001). Although these high PM<sub>1</sub> 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 (PM<sub>1</sub>) trials and 6.3 ± 3.4 ppm for high (PM<sub>1</sub>) 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.

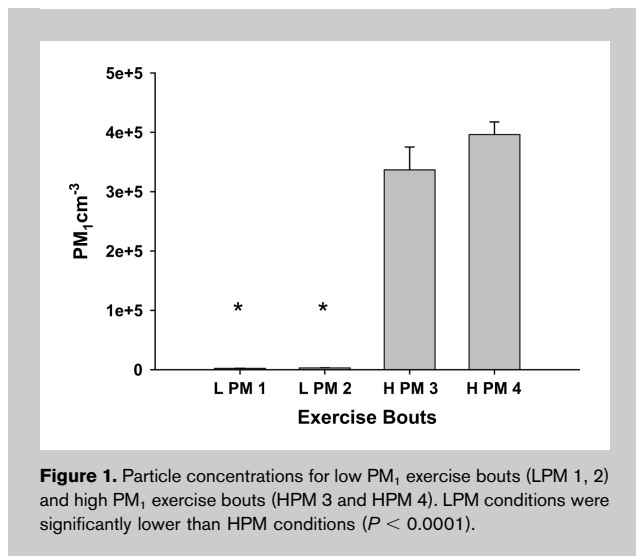
**RESULTS**

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 PM<sub>1</sub> trials; respectively). Figure 2 shows accumulated work for sequential trials. Work accumulated was not different between low PM<sub>1</sub> (trials 1 and 2, 96.1 ± 9.38 versus 96.6 ± 10.83 kJ) and not different from high PM<sub>1</sub> trial 3 (96.8 ± 10.65 kJ). Interestingly, work accumulated in the second high PM<sub>1</sub> (trial 4, 91.3 ± 10.04 kJ), 3 days following the first high PM<sub>1</sub> trial (trial 3), was significantly less than work accumulated in low PM<sub>1</sub> trials and the first high PM<sub>1</sub> trial (P = 0.004, P = 0.003, P = 0.0008; respectively).

Normalizing to kJ·kg<sup>-1</sup> bodyweight gave similar results. No significant correlations were identified between PM<sub>1</sub> concentrations and accumulated work either as absolute values for all trials or as differences in PM<sub>1</sub> values and work between high PM<sub>1</sub> trials.

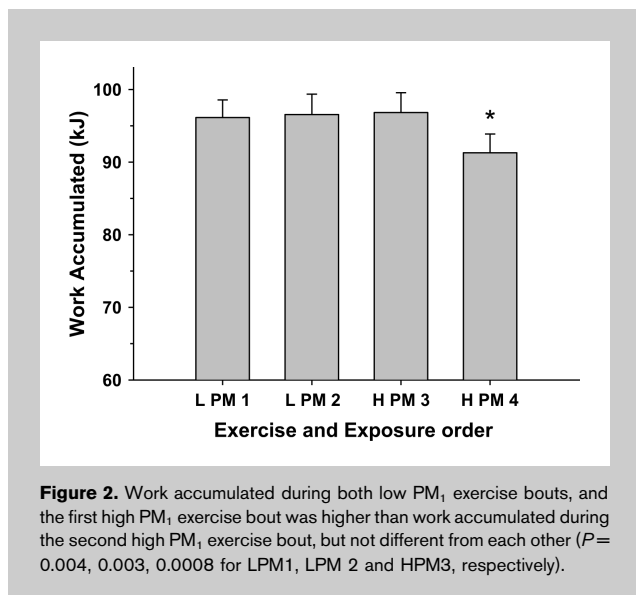
**TABLE 1.** Lung function before and after trial 1, a 6 min low PM<sub>1</sub> inhalation high intensity cycle ergometer challenge. All subjects demonstrated normal resting lung function and were negative for EIB. N = 15.

	FVC (L) baseline	Percent predicted	FVC % fall	FEV <sub>1</sub> (L) baseline	Percent predicted	FEV <sub>1</sub> % fall	FEF <sub>25-75</sub> (L·s <sup>-1</sup> ) baseline	Percent predicted	FEF <sub>25-75</sub> % fall
MN	5.62	108.4	-4.55	4.77	109.5	-2.14	5.12	103.4	-2.49
SD	0.793	10.55	3.902	0.855	15.94	3.556	1.707	32.97	6.444



## DISCUSSION

This study was designed to determine whether PM<sub>1</sub> inhalation affected exercise performance; a mechanism for our observation of decreased performance after the second of 2 (PM<sub>1</sub>) 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 (PM<sub>1</sub>) trials were performed one week prior to the high (PM<sub>1</sub>) trials and work accumulated was not different between the first high (PM<sub>1</sub>) trial and the two low (PM<sub>1</sub>) 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



(PM<sub>1</sub>) exposure trial that affected muscle perfusion in the second high (PM<sub>1</sub>) trial 3 days later.

We previously reported diminished flow-mediated vasodilation (FMD) after 30 min of running while breathing high (PM<sub>1</sub>), suggesting a PM<sub>1</sub>-induced disruption of normal endothelial-mediated vasodilation (19). The near abolishment 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 PM<sub>1</sub> inhalation in the microvasculature that was independent of the observed brachial artery vasoconstriction. Calculation of the return slope of reoxygenation ( $\mu\text{M}\cdot\text{s}^{-1}$ ) to baseline after cuff ischemia (19) provided a rate of decreased reperfusion of the muscle tissue (after 30 minute exercise breathing high PM<sub>1</sub>) that should be proportional to blood flow. We also identified a 4% vasoconstriction of the brachial artery after exercise while breathing high (PM<sub>1</sub>) 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 PM<sub>1</sub> 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 vaso-responsiveness 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 PM<sub>1</sub> exercise bout resulted solely from the first high PM<sub>1</sub> exposure exercise; however, the data suggest that performance for a short duration exercise bout while inhaling PM<sub>1</sub> 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 PM<sub>1</sub> exposure exercise. Future studies should explore effects of PM<sub>1</sub> inhalation during exercise on muscle microcirculation 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 PM<sub>1</sub> in the range of the high PM<sub>1</sub> 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 PM<sub>1</sub> emitted from fossil-fueled zambonis (16). Reduced lung function and increase airway hyperreactivity in ice rink athletes has been associated with high rink PM<sub>1</sub> levels (17, 18); however, use of electric powered zambonis can create a pollution-free healthy rink environment.

### REFERENCES

- Batalha, JR, Saldiva, PH, Clarke, RW, Coull, BA, Stearns, RC, Lawrence, J, Murthy, GG, Koutrakis, P, Godleski, JJ. Concentrated ambient air particles induce vasoconstriction of small pulmonary arteries in rats. *Environ Health Perspect* 110: 1191-1197, 2002.
- Brook, RD, Brook, JR, Urch, B, Vincent, R, Rajagopalan, S, Silverman, F. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation* 105: 1534-1536, 2002.
- Cheng, YW, Kang, JJ. Inhibition of agonist-induced vasoconstriction and impairment of endothelium-dependent vasorelaxation by extract of motorcycle exhaust particles in vitro. *J Toxicol Environ Health A* 57: 75-87, 1999.
- Dockery, DW. Epidemiologic evidence of cardiovascular effects of particulate air pollution. *Environ Health Perspect* 109(Suppl 4): 483-486, 2001.
- Grunig, E, Mereles, D, Hildebrandt, W, Swenson, ER, Kubler, W, Kuecherer, H, Bartsch, P. Stress Doppler echocardiography for identification of susceptibility to high altitude pulmonary edema. *J Am Coll Cardiol* 35: 980-987, 2000.
- Hoek, G, Brunekreef, B, Fischer, P, van Wijnen, J. The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other cardiovascular causes of death in a time series study. *Epidemiology* 12: 355-357, 2001.
- Magari, SR, Hauser, R, Schwartz, J, Williams, PL, Smith, TJ, Christiani, DC. Association of heart rate variability with occupational and environmental exposure to particulate air pollution. *Circulation* 104: 986-991, 2001.
- Muto, E, Hayashi, T, Yamada, K, Eski, T, Sagai, M, Lguchi, A. Endothelial-constitutive nitric oxide synthase exists in airways and diesel exhaust particles inhibit the effect of nitric oxide. *Life Sci* 59: 1563-1570, 1996.
- Nurkiewicz, TR, Porter, DW, Barger, M, Castranova, V, Boegehold, MA. Particulate matter exposure impairs systemic microvascular endothelium-dependent dilation. *Environ Health Perspect* 112: 1299-1306, 2004.
- Nurkiewicz, TR, Porter, DW, Barger, M, Millecchia, L, Rao, KM, Marvar, PJ, Hubbs, AF, Castranova, V, Boegehold, MA. Systemic microvascular dysfunction and inflammation after pulmonary particulate matter exposure. *Environ Health Perspect* 114: 412-419, 2006.
- Oberdorster, G. Pulmonary deposition, clearance and effects of inhaled soluble and insoluble cadmium compounds. *LARC Sci Publ* 118: 189-204, Review. 1992.
- Oberdorster, G, Gelein, RM, Ferin, J, Weiss, B. Association of particulate air pollution and acute morbidity: Involvement of ultrafine particles? *Inhal Toxicol* 7: 111-124, 1995.
- Pekkanen, J, Peters, A, Hoek, G, Tiittanen, P, Brunekreef, B, de Hartog, J, Heinrich, J, Ibaldo-Mulli, A, Kreyling, WG, Lanki, T, Timonen, KL, Vanninen, E. Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the exposure and risk assessment for fine and ultrafine particles in ambient air (ULTRA) study. *Circulation* 106: 890-892, 2002.
- Peters, A, Dockery, DW, Muller, JE, Mittleman, MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation* 103: 2810-2815, 2001.
- Pope, CA, 3rd, Verrier, RL, Lovett, EG, Larson, AC, Raizenne, ME, Kanner, RE, Schwartz, J, Villegas, GM, Gold, DR, Dockery, DW. Heart rate variability associated with particulate air pollution. *Am Heart J* 138: 804-807, 1999.
- Rundell, KW. High levels of airborne ultrafine and fine particulate matter in indoor ice arenas. *Inhal Toxicol* 15: 237-50, 2003.
- Rundell, KW. Pulmonary function decay in women ice hockey players: is there a relationship to ice rink air quality? *Inhal Toxicol* 16: 117-123, 2004.
- Rundell, KW, Caviston, R, Hollenbach, AM, Murphy, K. Vehicular air pollution, playgrounds, and youth athletic fields. *Inhal Toxicol* 18: 541-547, 2006.
- Rundell, KW, Hoffman, JR, Caviston, R, Bulbulian, R, Hollenbach, AM. Inhalation of ultrafine and fine particulate matter disrupts systemic vascular function. *Inhal Toxicol* 19: 133-140, 2007.
- Rundell, KW, Spiering, BA, Evans, TM, Baumann, JM. Baseline lung function, exercise-induced bronchoconstriction, and asthma-like symptoms in elite women ice hockey players. *Med Sci Sports Exerc* 36: 405-410, 2004.
- Rundell, KW, Spiering, BA, Baumann, JM, Evans, TM. Bronchoconstriction provoked by exercise in a high-particulate-matter environment is attenuated by montelukast. *Inhal Toxicol* 17: 99-105, 2005.
- Rundell, KW, Evans, TM, Baumann, JM, Kertesz, MF. Lung function measured by impulse oscillometry and spirometry following eucapnic voluntary hyperventilation. *Can Respir J* 12: 257-263, 2005.
- Ulrich, MM, Alink, GM, Kumarathasan, P, Vincent, R, Boere, AJ, Cassee, FR. Health effects and time course of particulate matter on the cardiopulmonary system in rats with lung inflammation. *J Toxicol Environ Health A* 65: 1571-1595, 2002.