

Air pollution effects on exercise performance

Araz Nazari^{1*}, Yazgaldi Nazari²

¹: Department of Physical Education and Sport Sciences, Higher Educational Complex of Saravan, Saravan, Iran

²: PhD Student of Shahid Chamran University of Ahwaz, Ahwaz, Iran

y.nazari53@gmail.com

Abstract: Much of the research investigating exercise and air pollution focuses on exposure to air pollution during exercise. Most studies assessing the effects of carbon monoxide exposure on exercise performance suggest that performance is impaired. Acute ozone exposure reduces maximal exercise time, workload, oxygen consumption, and performance on a maximal exercise test. These performance decrements are likely due to increased respiratory discomfort. Any PM exposure experienced en route to an exercise location might cause physiological changes, which could also affect exercise performance. While gender and environmental conditions (PM count or particle size) may influence the effects of PM inhalation, the mechanism by which performance may be impacted is even more unclear. There are few evidences regarding the effects of pre-exercise exposure to diesel exhaust (DE) on exercise performance.

[Nazari A, Nazari Y. **Air pollution effects on exercise performance.** *N Y Sci J* 2016;9(7):82-88]. ISSN 1554-0200 (print); ISSN 2375-723X (online). <http://www.sciencepub.net/newyork>. 13. doi:[10.7537/marsnys090716.13](https://doi.org/10.7537/marsnys090716.13).

Keywords: Carbon monoxide, exercise performance, ozone, Particulate matter

1. Introduction

As athletes and exercisers may be exposed to air pollutions during the journey to their exercise location, it is important to understand both the physiological and performance implications of exposure to air pollution prior to exercise. Maximal oxygen consumption is a key indicator of aerobic exercise performance.

Most studies assessing the effects of carbon monoxide exposure on exercise performance suggest that performance is impaired (Koike et al., 1991; Aronow et al., 1984; Adams et al., 1988; Adir et al., 1999; Aronow and Cassidy., 1975; Aronow et al., 1977; Calverley et al., 1981; Drinkwater et al., 1974). However, a few studies do not (Keramidas et al., 2012; Turner and McNicol., 1993) and this difference is likely explained by the low levels of carbon monoxide administered, or the submaximal nature of exercise protocols (Keramidas et al., 2012; Turner and McNicol., 1993).

For each 17-ppb increase in ozone there is an associated reduction in aerobic fitness score by 1.52 % (Cakmak et al., 2011). Ambient ozone levels are associated with impaired marathon performance (El Helou et al., 2012); however, it is possible that the elevated temperature occurring in conjunction with ozone causes this response. Acute ozone exposure reduces maximal exercise time, workload, oxygen consumption, and performance on a maximal exercise test (Foxcroft and Adams, 1986; Gong et al., 1986; Linder et al., 1987). A Performance impairments can occur at relevant environmental concentrations (120 ppb) (Linder et al., 1987), and at 200 ppb a 30 % reduction in maximal exercise time (Gong et al.,

1986). These performance decrements are likely due to increased respiratory discomfort (Adams and Schelegle., 1983). In contrast to the above studies, two studies found that exposure ozone (100–300 ppb) does not affect exercise performance (Gomes et al., 2010), or maximal work (Savin and Adams., 1979). However, there is no clear reason for the discrepancy between studies.

Given that method of travel (e.g. walking, cycling, driving), distance from the source, and fuel type all play a role in individual PM exposure (Kingham et al., 1998; McNabola et al., 2008; Kaur and Nieuwenhuijsen, 2009; Zuurbier et al., 2010), any PM exposure prior to exercise may adversely affect the cardio-respiratory system, and impair performance, even when exercise is performed in a climate-controlled environment.

Carbon monoxide effects on exercise performance

Carbon monoxide is the leading cause of death by poisoning. It is colourless, odourless, tasteless, and non-irritating, which makes its presence difficult to detect. The signs and symptoms of CO poisoning are non-specific, and usually appear when COHb concentrations are above 10%.

In a study on athletes English, It was found that exposure to urban air CO (such as when people are exposed to urban traffic) decreased exercise performance) Carlisle and sharp, 2001).

In a cross - sectional study, Cakmak et al (2011) found a significant associations between exposures to elevated levels of air pollutants and increased blood pressure and heart rate, and decreased pulmonary function and physical exercise capacity. Carbon

monoxide exposure was associated with a reduction in treadmill exercise performance among fifteen healthy men but no ischemic cardiac changes could be detected. Blood carboxy hemoglobin concentrations were 0.6% and 5% in the air and air plus carbon monoxide exposures.

Morton et al's (1984) data indicate that nicotine may act as a direct coronary vasoconstrictor and produce myocardial ischemia. Thus, active or passive smoking sufficient to produce the same level of carboxyhemoglobin attained in this study may be associated with greater impairment of exercise performance.

Aronow (1981) reported an adverse effect of 2% carboxyhemoglobin on exercise performance in 15 patients with angina. In healthy individuals, COHb levels less than 4 % cause a significant reduction in maximal exercise time between 30 and 90 s, in tests lasting between 11 and 17.5 min (Adir et al., 1999; Aronow and Cassidy., 1975). In those with COPD and anemia, carbon monoxide exposure causes a 33 % reduction in time until marked dyspnea (Aronow et al., 1977) and a 16 % reduction in exercise time to fatigue (Aronow et al., 1984).

Exercise performance (the time taken to complete a walking task) may be more impaired in nonsmokers than smokers (Drinkwater et al., 1974), and is likely because of smokers adapting to high levels of COHb. Similar COHb levels to those measured in the above studies (4–5%) can occur during exercise in an urban environment (Nicholson and Case., 1983) therefore, oxygen consumption and exercise performance could be affected in urban environments on a regular basis. This suggests that the location of exercise events may play a key role in determining exercise performance.

Carbon monoxide exposure was associated with a reduction in treadmill exercise performance among fifteen healthy men but no ischemic cardiac changes could be detected. Blood carboxy hemoglobin concentrations were 0.6% and 5% in the air and air plus carbon monoxide exposures. The mean adjusted difference of work output, measured as metabolic equivalents between exposure to air and CO was 2.04 (95% CI 1.33–2.76) (Adir et al., 1999). However, exposure to 9 ppm has resulted in a gradual increase in COHb which peaked at only 0.7% after 5 h of continuous exposure (Horvath & Bedi, 1989), therefore, it does not appear as though elevated COHb levels contributed to the decreased performance observed in the present study.

In a study from Adams et al (1988) nonsmoking patients with coronary artery disease and evidence of exercise-induced ischemia underwent supine bicycle exercise after double blind exposure to air or carbon monoxide in a crossover design. Comparative exercise

performance after these exposures was assessed by clinical, electrocardiographic (ECG) and radionuclide variable: Subjective and objective indexes of cardiovascular function derived from these studies indicated a deleterious effect of 6% carboxyhemoglobin on patient performance. This work suggests that the threshold for an adverse effect of blood carboxyhemoglobin on exercise performance in general populations of patients with ischemic heart disease is between 4 and 6%. The most likely explanation for these deleterious effects of carbon monoxide is a worsening of myocardial ischemia due to impaired oxygen delivery to working myocardium. Exercise duration was significantly shorter after carbon monoxide than after air exposure.

Ozone Effects on Exercise Performance

Foxcroft and Adams (1986) showed that maximal exercise performance time was significantly affected by pre-exposure to ozone (700 $\mu\text{g}\cdot\text{m}^{-3}$). The participants, eight aerobically trained males, cycled on a cycle ergometer for 50 min at an intensity that required a minute ventilation of about 60 L $\cdot\text{min}^{-1}$. Then within 10 min of exposure they completed a $\dot{V}\text{O}_{2\text{max}}$ test on the same cycle ergometer. Performance time on the $\dot{V}\text{O}_{2\text{max}}$ test decreased from 253 to 211 s and $\dot{V}\text{O}_{2\text{max}}$ itself decreased from 3.85 to 3.62 L $\cdot\text{min}^{-1}$. The participants in this study were then exposed to the same concentration of ozone on four consecutive days. Significant improvements in $\dot{V}\text{O}_{2\text{max}}$ and performance time were observed when compared with initial exposure. The authors concluded that habituation to high ambient concentrations of ozone may benefit the performer in competition, but repeated long-term exposure to higher concentrations of ozone may be detrimental and should be avoided. Authors have shown clear discord in the relationship of pulmonary impairment and subjective symptoms response to maximum exercise performance.

Gong et al. (1986) also observed substantially elevated subjective symptoms following 1 hour's exposure to 0.20 ppm ozone with very heavy exercise ($\text{VE} = 90 \text{ L}/\text{min}$), which was associated with significant impairment in maximal exercise performance (-29.7%), $\dot{V}\text{O}_{2\text{max}}$ (-16.4%) and VE_{max} (-18.5%).

Significant reductions in maximum exercise performance have been observed following 2-hour intermittent exercise exposures to ozone at 0.60 ppm (Folinsbee et al. 1977). Conversely, Folinsbee et al. (1984) observed no reduction in their highly trained competitive cyclists' ability to complete 1 hour of continuous exercise at 90 L/min VE (75% of $\dot{V}\text{O}_{2\text{max}}$) while exposed in moderate ambient temperature (20°C) to 0.21 ppm ozone, even in one

subject who experienced a FEV_{1.0} decrement of 35%. Finally, Folinsbee et al (1986) found that highly trained runners' treadmill run performance at ~ 80% V_O2 max was reduced from 71.7 minutes in filtered air to 66.2 minutes upon exposure to 0.18 ppm. Tidal volume was increased 10% and f decreased 6% with ozone, but no effect on arterial O₂ pressure was observed. These authors concluded that limitation to exercise performance associated with ozone results mainly from respiratory discomfort and not from impairment of O₂ delivery.

Avol and colleagues (1985) have suggested that reduced FEV_{1.0} and increased subjective symptoms, first observed at 0.16 ppm ozone, precede the occurrence of exercise performance reduction (assessed as the necessity to reduce workload near the end of 1 hour of continuous heavy exercise) in exposures to 0.24 and 0.32 ppm.

Adams & Schelegle (1983) observed no significant increase in VD or VA in 1 hour continuous training and competitive simulation protocols in highly trained endurance athletes exposed to 0.20 and 0.35 ppm ozone. Even though 4 of 10 subjects were unable to complete the full hour's exposure to 0.35 ppm, no significant effect of ozone was observed for VE, V_O2, HR, nor for Oz pulse (V_O2/HR), an indirect index of O₂ diffusion and delivery. Schelegle & Adams (1986) observed significantly reduced exercise performance during the last 30 minutes of competitive simulations (mean VE =120 L/min) in highly trained cyclists exposed for hour to 0.18 and 0.24 ppm ozone. However, no significant effects of ozone were observed for HR, VE, or V_O2.

Evidence previously presented clearly documents that, if sufficient ozone inhalation occurs prior to even short (i.e. several minutes) maximum exercise, performance is significantly impaired, along with V_O2_{max} (Folinsbee et al. 1977; Foxcroft & Adams 1986; Gong et al. 1986). Further, continuous exercise of 1-hour duration is compromised upon exposure to ambient ozone alert levels between 0.20 and 0.30 ppm when exercise intensity exceeds about 75% at moderate ambient temperatures (DeLucia & Adams 1977; Folinsbee et al. 1986; Schelegle & Adams 1986).

The apparent incongruity of all of the highly trained subjects studied by Adams and Schelegle (1983) and Folinsbee et al. (1984) completing 1 hour of heavy continuous exercise at 0.20 and 0.21 ppm ozone, respectively, whereas about one-third of the subjects in the study of Gong et al. (1986) exposed to 0.20 ppm ozone did not, may be due to the addition of ambient heat (31°C) in the latter study. Some subjects in the study of Avol et al. (1984) also exposed to ambient heat (32°C), were unable to complete 1 hour of moderately heavy continuous exercise at lower V

without reduction in workloads at 0.24 and 0.32 ppm. This contention is substantiated by the observations of Gibbons and Adams (1984), who found that 3 of 10 well-trained female subjects attempting 1 hour of continuous exercise at 66% of V_O2 max, while exposed to 0.30 ppm ozone, were able to complete only 44 minutes of exercise in 35°C, while only 1 (51 minutes) was unable to complete the protocol in the 24°C condition. These authors concluded that the effects of ambient heat coinciding with ozone inhalation are likely to result in more severe impairment of exercise performance, although the mechanisms remain unclear.

Mechanisms accounting for reduced exercise performance are at present not well defined. In general, reduced exercise performance consequent to ozone exposure has been accompanied by pulmonary function impairment, enhanced subjective symptoms of respiratory discomfort, and usually enhanced rapid, shallow breathing. Decreased FVC upon exposure to ozone at ambient photochemical smog episode levels is primarily due to reduced maximal inspiration, either involuntarily or due to discomfort (Folinsbee and Raven., 1984; Adams & Schelegle 1983; Folinsbee et al. 1978; McDonnell et al. 1983). Reduced FEV_{1.0} and FEF₂₅₋₇₅ are primarily associated with reductions in maximal expiratory position and reduced inspiratory capacity, although also affected less so by increased bronchoconstriction and Raw (Beckett et al. 1985; Folinsbee et al. 1978; McDonnell et al. 1983). Rapid, shallow breathing induced via ozone inhalation during moderate to heavy exercise, and the occurrence of subjective symptoms of respiratory discomfort (cough and pain on deep inspiration), also appear to be more closely associated with reduced inspiratory capacity and FVC than increased Raw (Beckett et al. 1985; Schelegle & Adams 1987).

Particulate matter effects on exercise performance

In researches of Cutrufello et al (2011), the correlation between the change in performance from low particulate matter to high particulate matter and the corresponding change in flow-mediated vasodilation (FMD) suggest that the change in FMD accounted for 24.4% of the change in exercise performance in response to inhaled exhaust emissions. This novel finding suggests a mechanism by which exercise performance may be negatively affected through HPM inhalation and while multiple physiological responses are likely responsible for a change in exercise performance, these data support an acute change in FMD and a related change in exercise performance.

Marr and Ely (2010) found that PM₁₀ was associated with impaired marathon performance for women but not men. The authors suggested that

increased airway hyper responsiveness and a vagal response among women may trigger a decrease in the depth of inhalation and result in a slower running pace. Giles et al (2012) reasoned that any PM exposure experienced en route to an exercise location might cause physiological changes, which could also affect exercise performance. During the experiment by Rundell et al. (2008) participants were exposed to 2 low PM₁ days separated by 3 days, then a 7-day washout period followed by 2 high PM₁ days separated by 3 days. It is important to note that this study only found a significant impairment in exercise performance on the second day of high PM exposure, but not on the first day of high PM exposure. Despite reasonable washout periods it is possible that the decrement in performance could be because of the cumulative effect of exposure, which may explain why we did not find an effect on exercise performance.

While gender and environmental conditions (PM count or particle size) may influence the effects of PM inhalation, the mechanism by which performance may be impacted is even more unclear. Oxidative stress (Vinzents et al., 2005; LeBlanc et al., 2010), an inflammatory response (Donaldson & Tran, 2002; Cho et al., 2007; Larsson et al., 2007; Barraza-Villarreal et al., 2008), decreased lung function (Pietropaoli et al., 2004; Rundell, 2004; Barraza-Villarreal et al., 2008; Rundell et al., 2008), pulmonary vascular dysfunction (Pietropaoli et al., 2004; Frampton, 2007), and systemic vascular dysfunction (Brook et al., 2002; Nurkiewicz et al., 2004; Nurkiewicz et al., 2006; Rundell et al., 2007) have each been identified as a potential mechanism which may negatively affect exercise performance, however, neither of the aforementioned studies (Rundell & Caviston, 2008; Marr & Ely, 2010) examining exercise performance assessed any potential mechanism.

Exhaust emissions effects on exercise performance

There are few evidences regarding the effects of pre-exercise exposure to diesel exhaust (DE) on the cardio-respiratory system prior to, during and following exercise, and on exercise performance.

The result of study of Cutrufello et al (2011) identified a link between exercise performance and the inhalation of freshly generated whole exhaust among male collegiate athletes. While a 2.87% decline in exercise performance may seem insignificant, this may significantly affect the outcome of competition. For instance, the 1st and 11th place finishers in the 3000 m steeplechase at the 2008 Olympics, an event similar in duration to the maximal exercise bouts used in the present study, were separated by less than 2.87%. Endothelial dysfunction

has been observed 24 h after a single 1 h exposure to diesel exhaust among humans (Törnqvist et al., 2007) and may suggest that a single short-term exposure to elevated PM inhalation may result in exercise performance deficits up to 24 h. Initial studies assessing the effects of air pollution exposure on exercise performance date back to the 1960s and involved exposure to car exhaust (Holland et al., 1968).

Giles et al (2012) determined the effect of pre-exercise exposure to DE containing 300 $\mu\text{g}/\text{m}^3$ of particulate matter 2.5 microns or less (PM_{2.5}) on 20 km cycling time trial performance, pulmonary function, and cardio-respiratory responses during exercise. they hypothesized that pre-exercise exposure to DE (300 $\mu\text{g}/\text{m}^3$ of PM_{2.5}) would significantly impair performance of a 20 km cycling time trial, and pulmonary function and alter cardio-respiratory responses during exercise. In healthy endurance-trained males they found that pre-exercise exposure to DE did not impair exercise performance but attenuated exercise-induced bronchodilation, and increased exercise heart rate.

Discussions

In air pollution the higher VE and breathing frequency, and lower tidal volumes for the same amount of work may increase dead space ventilation, perceived exertion, and competition for blood flow between the exercising and respiratory muscles, all of which may impair exercise performance. Elevated ambient temperature can also increase perceived exertion and air pollution-induced lung inflammation, and impair exercise performance; therefore, exposure to high levels of air pollution in a warm climate may be the least suitable environment for exercise performance. Individuals should take into account the method of transport to an exercise location. In particular, competing athletes and those with heart disease could consider avoiding traffic prior to exercise to minimize the health risk and potential effects on exercise performance. Because high levels of ozone and carbon monoxide increase symptoms during exercise, this could also discourage an individual from exercising, thus reducing physical activity levels and health status. Therefore, to avoid exposure to air pollution, individuals should be encouraged to exercise away from traffic.

Corresponding Author:

Araz nazari

Department of Physical Education and Sport Sciences, Higher Educational Complex of Saravan, Saravan, Iran

E-mail: Y.nazari53@gmail.com

References

1. Adams WC, Schelegle ES. Ozone and high ventilation effects on pulmonary function and endurance performance. *J Appl Physiol*. 1983;55(3):805–12.
2. Adams KF, Koch G, Chatterjee B, Goldstein GM, O'neil JJ, Bromberg PA, Sheps DS (1988). Acute elevation of blood carboxyhemoglobin to 6% impairs exercise performance and aggravates symptoms in patients with ischemic heart disease. *Iacc voi* 12 900- 909.
3. Adir, Y., Merdler, A., BenHaim, S., Front, A., Harduf, R., Bitterman, H., 1999. Effects of exposure to low concentrations of carbonmonoxide on exercise performance and myocardial perfusion in young healthy men. *Occup. Environ. Med.* 56 (8), 535– 538.
4. Aronow WS. Aggruvmiun of angina pectoris by Iwo percent curhoxyhemoglebin. *Am Heart J* 1981 :101 :154 7.
5. Aronow WS, Ferlinz J, Glauser F. Effect of carbon monoxide on exercise performance in chronic obstructive pulmonary disease. *Am J Med.* 1977;63(6):904–8.
6. Aronow WS, Cassidy J. Effect of carbon monoxide on maximal treadmill exercise: a study in normal persons. *Ann Int Med.* 1975;83(4):496–9.
7. Aronow WS, Schlueter WJ, Williams MA, et al. Aggravation of exercise performance in patients with anemia by 3% carboxyhemoglobin. *Environ Res.* 1984;35(2):394–8.
8. Avol EL, Linn WS, Shamoo DA, Valencia LM, Anzar UT, et al. Respiratory effects of photochemical oxidant air pollution in exercising adolescents. *American Review of Respiratory Diseases* 132: 619-622, 1985.
9. Avol EL, Linn WS, Venet TG, Shamoo DA, Hackney JD. Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. *Journal of the Air Pollution Control Association* 34: 804-809, 1984.
10. Barraza-Villarreal A, Sunyer J, Hernandez-Cadena L, Escamilla-Nuñez MC, Sienna-Monge JJ, Ramirez-Aguilar M, Cortez-Lugo M, Holguin F, Diaz-Sánchez D, Olin AC, Romieu I. 2008. Air pollution, airway inflammation, and lung function in a cohort study of Mexico City schoolchildren. *Environ Health Perspect* 116:832–838.
11. Beckett WS, McDonnell WF, Horstman DH, House DE. Role of the parasympathetic nervous system in acute lung response to ozone. *Journal of Applied Physiology* 59: 1879-1885, 1985.
12. Brook RD, Brook JR, Urch B, Vincent R, Rajagopalan S, Silverman F. 2002. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation* 105:1534–1536.
13. Cakmak S, Dales R, Leech J, et al. The influence of air pollution on cardiovascular and pulmonary function and exercise capacity: Canadian Health Measures Survey (CHMS). *Environ Res.* 2011;111(8):1309–12. doi:10.1016/j.envres.2011.09.016.
14. Carlisle AJ, Sharp NC. "Exercise and outdoor ambient air pollution. *British journal of Sports Medicint*". Vol 35. pp. 214-222, 2001.
15. Calverley PM, Leggett RJ, Flenley DC. Carbon monoxide and exercise tolerance in chronic bronchitis and emphysema. *Br Med J Clin Res Ed.* 1981;283(6296):878–80.
16. Cho WS, Choi M, Han BS, Cho M, Oh J, Park K, Kim SJ, Kim SH, Jeong J. 2007. Inflammatory mediators induced by intratracheal instillation of ultrafine amorphous silica particles. *Toxicol Lett* 175:24–33.
17. Cutrufello P T., Rundell K W., Smoliga J M., Stylianides G A. Inhaled whole exhaust and its effect on exercise performance and vascular function. *Inhalation Toxicology*, 2011; 23(11): 658–667.
18. DeLucia AJ, Adams We. Effects of O₂ inhalation during exercise on pulmonary function and blood biochemistry. *Journal of Applied Physiology* 43: 75-81, 1977.
19. Donaldson K, Tran CL. 2002. Inflammation caused by particles and fibers. *Inhal Toxicol* 14:5–27.
20. Drinkwater BL, Raven PB, Horvath SM, et al. Air pollution, exercise, and heat stress. *Arch Environ Health.* 1974;28(4): 177–81.
21. El Helou N, Tafflet M, Berthelot G, et al. Impact of environmental parameters on marathon running performance. *PLoS ONE.* 2012;7(5):e37407. doi:10.1371/journal.pone.0037407.
22. Folinsbee U, Bedi JF, Horvath SM. Pulmonary function changes after 1h continuous heavy exercise in 0.21 ppm ozone. *Journal of Applied Physiology* 57: 984-988, 1984
23. Folinsbee U, Drinkwater BL, Bedi JF, Horvath SM. The influence of exercise on the pulmonary function changes due to low concentrations of ozone. In Folinsbee et al. (Eds) *Environmental stress*, pp. 125-145, Academic Press, New York, 1978.
24. Folinsbee U, Horstman DH, Vorona RD, Prince JM, Berry M. Determinants of endurance performance during ozone inhalation. 30th

- Congress of the International Union of Physiological Sciences, Vancouver, July 13-18, 1986. Abstract No. P217.04, p. 176, 1986.
25. Folinsbee LJ., Raven PB. Exercise and air pollution. *Journal of Sports Sciences* 2: 57-75, 1984.
 26. Folinsbee LJ., Silverman F, Shephard RJ. Decrease of maximum work performance following ozone exposure. *Journal of Applied Physiology* 42: 531-536, 1977.
 27. Foxcroft, W. Adams, W. (1986). Effects of ozone exposure on four consecutive days on work performance and $\dot{V}O_2\text{max}$. *Journal of Applied Physiology*, 61, 960 – 966.
 28. Frampton MW. 2007. Does inhalation of ultrafine particles cause pulmonary vascular effects in humans? *Inhal Toxicol* 19 Suppl 1:75–79.
 29. Gibbons SI, Adams We. Combined effects of ozone exposure and ambient heat on exercising females. *Journal of Applied Physiology* 57: 450-456, 1984.
 30. Giles LV, Carlsten C, Koehle MS. The effect of pre-exercise diesel exhaust exposure on cycling performance and cardio-respiratory variables. *Inhalation Toxicology*, 2012; 24(12): 783–789.
 31. Gomes EC, Stone V, Florida-James G. Investigating performance and lung function in a hot, humid and ozone-polluted environment. *Eur J Appl Physiol*. 2010;110(1):199–205. doi:10.1007/s00421-010-1485-8.
 32. Gong H Jr, Bradley PW, Simmons MS, et al. Impaired exercise performance and pulmonary function in elite cyclists during low-level ozone exposure in a hot environment. *Am Rev Resp Dis*. 1986;134(4):726–33.
 33. Holland GJ, Benson D, Bush A, et al. Air pollution simulation and human performance. *Am J Public Health Nations Health*. 1968;58(9):1684–91.
 34. Horvath SM, Bedi JF. 1989. Alteration in carboxyhemoglobin concentrations during exposure to 9 ppm carbon monoxide for 8 hours at sea level and 2134 m altitude in a hypobaric chamber. *JAPCA* 39:1323–1327.
 35. Kaur S, Nieuwenhuijsen MJ. 2009. Determinants of personal exposure to PM_{2.5}, ultrafine particle counts, and CO in a transport microenvironment. *Environ Sci Technol* 43:4737–4743.
 36. Keramidis ME, Kounalakis SN, Eiken O, et al. Carbon monoxide exposure during exercise performance: muscle and cerebral oxygenation. *Acta Physiol (Oxf)*. 2012; 204 (4):544–54. doi:10.1111/j.1748-1716.2011.02363.x.
 37. Kingham SM, Andrew Sheard J, Lawrenson O. 1998. Assessment of exposure to traffic-related fumes during the journey to work. *Transport Res* 3:271–274.
 38. Koike A, Wasserman K, Armon Y, et al. The work-ratedependent effect of carbon monoxide on ventilatory control during exercise. *Respir Physiol*. 1991;85(2):169–83.
 39. Larsson BM, Sehlstedt M, Grunewald J, Skld CM, Lundin A, Blomberg A, Sandstrm T, Eklund A, Svartengren M. 2007. Road tunnel air pollution induces bronchoalveolar inflammation in healthy subjects. *Eur Respir J* 29:699–705.
 40. LeBlanc AJ, Moseley AM, Chen BT, Frazer D, Castranova V, Nurkiewicz TR. 2010. Nanoparticle inhalation impairs coronary microvascular reactivity via a local reactive oxygen species-dependent mechanism. *Cardiovasc Toxicol* 10:27–36.
 41. Linder J, Herren D, Monn C, et al. Effect of ozone on physical performance capacity. *Soz Praventivmed*. 1987;32(4–5):251–2.
 42. Marr LC, Ely MR. 2010. Effect of air pollution on marathon running performance. *Med Sci Sports Exerc* 42:585–591.
 43. McDonnell WF, Horstman DH, Hazucha MJ, Seal Jr E, Haak ED, et al. Pulmonary effects of ozone exposure during exercise: Dose-response characteristics. *Journal of Applied Physiology* 34: 1345-1352, 1983.
 44. McNabola A, Broderick BM, Gill LW. 2008. Relative exposure to fine particulate matter and VOCs between transport microenvironments in Dublin: Personal exposure and uptake. *Atmos Environ* 42:6496–6512.
 45. Nicholson JP, Case DB. Carboxyhemoglobin in New York City runners. *Physician Sports Med*. 1983;11(3):135–8.
 46. Nurkiewicz TR, Porter DW, Barger M, Castranova V, Boegehold MA. 2004. Particulate matter exposure impairs systemic microvascular endothelium-dependent dilation. *Environ Health Perspect* 112:1299–1306.
 47. Nurkiewicz TR, Porter DW, Barger M, Millecchia L, Rao KM, Marvar PJ, Hubbs AF, Castranova V, Boegehold MA. 2006. Systemic microvascular dysfunction and inflammation after pulmonary particulate matter exposure. *Environ Health Perspect* 114:412–419.
 48. Pietropaoli AP, Frampton MW, Hyde RW, Morrow PE, Oberdrster G, Cox C, Speers DM, Frasier LM, Chalupa DC, Huang LS, Utell MJ. 2004. Pulmonary function, diffusing capacity, and inflammation in healthy and asthmatic subjects exposed to ultrafine particles. *Inhal Toxicol* 16 Suppl 1:59–72.

49. Rundell KW. 2004. Pulmonary function decay in women ice hockey players: is there a relationship to ice rink air quality? *Inhal Toxicol* 16:117–123.
50. Rundell KW, Caviston R. 2008. Ultrafine and fine particulate matter inhalation decreases exercise performance in healthy subjects. *J Strength Cond Res* 22:2–5.
51. Rundell KW, Hoffman JR, Caviston R, Bulbulian R, Hollenbach AM. 2007. Inhalation of ultrafine and fine particulate matter disrupts systemic vascular function. *Inhal Toxicol* 19:133–140.
52. Rundell KW, Slee JB, Caviston R, Hollenbach AM. 2008. Decreased lung function after inhalation of ultrafine and fine particulate matter during exercise is related to decreased total nitrate in exhaled breath condensate. *Inhal Toxicol* 20:1–9.
53. Savin WM, Adams WC. Effects of ozone inhalation on work performance and VO₂ max. *J Appl Physiol*. 1979;46(2):309–14.
54. Schelegle ES, Adams We. Indomethacin pretreatment reduces ozone induced pulmonary function decrements in human subjects. *American Review of Respiratory Diseases*, in press, 1987.
55. Schelegle ES, Adams We. Reduced exercise time in competitive simulations consequent to low level ozone exposure. *Medicine and Science in Sports and Exercise* 18: 408-414, 1986.
56. Trnqvist H, Mills NL, Gonzalez M, Miller MR, Robinson SD, Megson IL, Macnee W, Donaldson K, Sderberg S, Newby DE, Sandstrom T, Blomberg A. 2007. Persistent endothelial dysfunction in humans after diesel exhaust inhalation. *Am J Respir Crit Care Med* 176:395–400.
57. Turner JA, McNicol MW. The effect of nicotine and carbon monoxide on exercise performance in normal subjects. *Respir Med*. 1993;87(6):427–31.
58. Vinzents PS, Miller P, Srensen M, Knudsen LE, Hertel O, Jensen FP, Schibye B, Loft S. 2005. Personal exposure to ultrafine particles and oxidative DNA damage. *Environ Health Perspect* 113:1485–1490.
59. Zuurbier M, Hoek G, Oldenwening M, Lenters V, Meliefste K, van den Hazel P, Brunekreef B. 2010. Commuters' exposure to particulate matter air pollution is affected by mode of transport, fuel type, and route. *Environ Health Perspect* 118:783–789.

7/20/2016