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RESEARCH ARTICLE

The effect of pre-exercise diesel exhaust exposure on cycling performance and cardio-respiratory variables

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Abstract

Purpose: To determine the effect of pre-exercise exposure to diesel exhaust (DE) on 20-km cycling performance, pulmonary function, and cardio-respiratory variables during exercise.

Methods: Eight endurance-trained males participated in the study. Test days consisted of a 60-min exposure to either filtered air (FA) or DE, followed by a 20 km cycling time trial. Exposures to DE were at a concentration of 300 μg/m³ of $PM_{2.5}$. Forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) were measured before and after exposure, and after exercise. Oxygen consumption (VO_2) and carbon dioxide production (VCO_2), minute ventilation (V_E), tidal volume (V_T), breathing frequency (F_B), heart rate and oxyhemoglobin saturation (SpO_2), were collected during the time trials. The effect of condition on time trial duration, an order effect, and mean cardio-respiratory variables were each analysed using paired T-tests. Repeated-measures ANOVA were used to assess the effect of DE exposure on pulmonary function.

Results: There was a main effect of condition (FA vs. DE) on the change in FEV_1 from baseline, and in exercise heart rate. *Post hoc* tests revealed that exercise-induced bronchodilation was significantly attenuated following DE compared to FA. There were no main effects of condition on 20 km cycling performance, or VO_2, VCO_2, V_E, V_T, F_B and SpO_2 during a 20 km time trial.

Conclusion: A 60-min exposure to DE prior to exercise significantly attenuated exercise-induced bronchodilation and significantly increased heart rate during exercise. Pre-exercise exposure to diesel exhaust did not significantly impair 20 km cycling time trial performance.

Keywords: Air pollution, exercise performance, heart rate, particulate matter, pulmonary function

Introduction

In healthy individuals and asthmatics, urban levels of particulate matter (PM) can impair pulmonary function (McCreanor et al., 2007; Rundell et al., 2008) and cause pulmonary inflammation (McCreanor et al., 2007; Graff et al., 2009). However, there is limited knowledge about how air pollution/PM and exercise interactively affect the cardio-respiratory system, and how they affect exercise performance. Ambient studies suggest that exposure to ultrafine particles during exercise is associated with decrements in pulmonary function immediately after 30 min of exercise in a high particulate matter of 1 micron or less (PM_1) environment (Rundell et al., 2008). Additionally, endurance-trained cyclists exposed to high levels of ozone exhibited a 21% reduction in forced expiratory volume in 1 s (FEV_1), and a significant reduction in peak minute ventilation (V_E), tidal volume (V_T), and maximal oxygen consumption (VO_2) (Gong et al., 1986).

The effects of ozone and carbon monoxide on exercise performance have been previously documented (Aronow et al., 1977; Gong et al., 1986; Adir et al., 1999), both of which caused a significant reduction in exercise work (Gong et al., 1986) and exercise time (Aronow and Cassidy, 1975; Gong et al., 1986; Adir et al., 1999).

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However, there are minimal data regarding PM exposure and exercise performance. The only two studies assessing the effects of PM exposure on exercise performance also suggest that in healthy adults exposure to high levels of PM_1 was associated with decreased performance during a 6-min cycling ergometer trial (Rundell and Caviston, 2008; Cutrufello et al., 2011). Additionally, there is a correlation between PM_{10} levels and reduced marathon performance in women (Marr and Ely, 2010).

There is currently no evidence regarding the effects of pre-exercise exposure to diesel exhaust (DE) or PM on the cardio-respiratory system prior to, during and following exercise, and on exercise performance. 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. As athletes and exercisers may be exposed to PM 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. Therefore, the purpose of this study was to determine the effect of pre-exercise exposure to DE containing 300 μg/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. We hypothesized that pre-exercise exposure to DE (300 μg/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.

Methods

Participants

Eight endurance-trained males aged [mean (SD)] 29 (6) yrs; height: 1.79 (0.10) m; weight: 72.6 (5.4) kg, with a mean maximal oxygen consumption 68 (8) mL·kg^{-1}·min^{-1}, volunteered for the study. The sample size was established based on an expected difference in exercise duration of 1.7 min, and standard deviation of 0.5 min (Adir et al., 1999). Each participant was a nonsmoker and had no history of respiratory or cardiovascular disease. This study that was approved by the Clinical Research Ethics Board at the University of British Columbia and participants had an orientation session and a 24-h reflection period prior to signing informed consent. Prior to data collection, each participant refrained from caffeine and alcohol ingestion, as well as intense exercise for 12 h. Participants were also asked to maintain the same pretest routine including the same mode of travel to the laboratory, and pretest meal.

Procedures

Participants attended the lab on three occasions. Day 1 consisted of a maximum exercise test, a practice 20 km time trial, and familiarization with the other study procedures. For the maximal exercise test, the work rate started at 100 W, and increased by 0.5 W/s until volitional exhaustion. For each participant, days 2 and 3 began at the same time each day and were separated by at least a 7-day washout. Testing days 2 and 3 consisted of a 60-min exposure to filtered air (FA) or DE at a concentration of 300 μg/m^3 of $PM_{2.5}$ at rest, followed by a 20 km cycling time trial (Figure 1). Exposure order was randomized and participants were blinded to the condition. This dose of DE is occupationally relevant (Groves and Cain, 2000; Lewne et al., 2007) and is approximately 1 order of magnitude greater that 24-h ambient standard in Canada. For the 20 km time trial, subjects were asked to cover the distance as fast as possible; this is an exhaustive task that takes approximately 30 min. Performance was measured as the time taken to complete the distance, which is highly repeatable in trained cyclists (Sporer and McKenzie, 2007). During the 20 km time trials, participants were only provided with information on distance covered and gearing. Pulmonary function was assessed before and after exposure (KoKo PFT Spirometer, nSpire Health Inc, Longmont Co, USA), and after exercise (Figure 1). Minute ventilation (V_E), tidal volume (V_T), breathing frequency (F_B), volume of oxygen consumed (VO_2), volume of CO_2 produced (VCO_2) heart rate and oxyhemoglobin saturation (measured by pulse oximetry: SpO_2) were collected continuously during the time trials. The crossover design was chosen for its inherent power by eliminating typical concerns of between-subject variability.

Pulmonary function

Standard measures of pulmonary function were measured as per the American Thoracic Society guidelines (Miller et al., 2005), including forced vital capacity (FVC), FEV_1, and the ratio of FVC to FEV_1 (FEV_1/FVC). On tests days, participants performed the procedure three times, and the peak values were used in the analysis. If FEV_1 and FVC did not fall within 0.15 L for largest and the next largest values the procedure was repeated until acceptable repeatability was achieved.

Apparatus

All exercise tests were performed on a cycle ergometer (Velotron Pro, Racermate Inc, Seattle, WA, USA) in a climate-controlled room. During exercise tests, participants breathed through a mouthpiece attached to a low-resistance, nonrebreathing valve (NRB 2700, Hans Rudolph Inc, Kansas City, MO, USA). Mixed expired gases were drawn to a computerized metabolic cart (TrueOne® 2400, ParvoMedics, Sandy, UT, USA) and averaged every 15 s. Oxyhemoglobin saturation (SpO_2) and heart rate were measured using pulse oximetry at the finger and a polar heart rate monitor respectively.

All exposures were performed in a previously-validated environmental exposure booth (Birger et al., 2011). An Environmental Protection Agency Tier 3-compliant, 5.5 kW diesel generator was operated under discrete cyclic loads to simulate diesel on-road emissions. A portion of raw

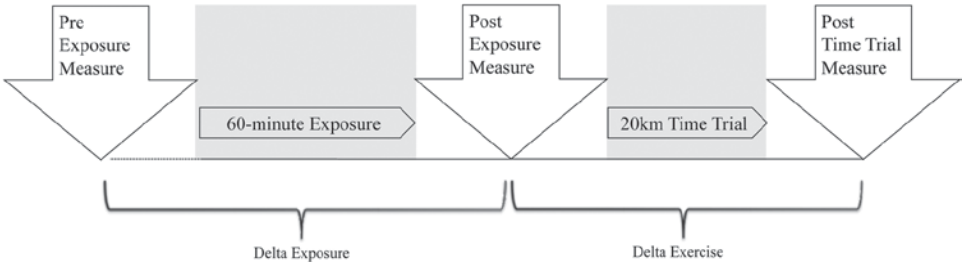

Figure 1. Experimental design.

exhaust was drawn into the primary dilution system and diluted 9:1 with compressed air to condition the exhaust to atmospheric-like exposure. The exhaust was further diluted approximately 26:1 by a stream of high-efficiency particulate air (HEPA)-filtered air to achieve the desired $PM_{2.5}$ concentration of 300 μg/m³ inside the 1.2×1.8×2.1 m exposure booth. The aging of particles from source to subject was approximately 4 min. In-booth PM mass concentration measurements were made using a Rupprecht & Pattashnick Model 1400a Tapered Element Oscillating Microbalance (TEOM) in a near-continuous manner using 10-min averages. A stream of in-chamber particulate was fed through a nephelometer to provide an instantaneous light scattering coefficient for closed-loop system control. A TSI Model 3936 Scanning Mobility Particle Scanner classified the particle size distribution. Oxides of nitrogen, CO, CO_2, relative humidity, temperature, and total volatile organic compounds were recorded every 10 s.

Data and statistical analysis

Statistical analyses were completed using SPSS software (SPSS Inc, version 11, Chicago, IL). Mixed expired gases were sampled and averaged every 15 s. Duration of time trials (which indicated exercise performance), an order effect, mean exercise V_E, V_T, F_B, SpO_2, and heart rate during time trials were analysed using paired t-tests. Pulmonary function (FEV_1 and FVC) was analysed using a 2 (condition: FA vs. DE) × 2 (change in pulmonary function: Δ exposure, Δ exercise) repeated-measures ANOVA, where delta (Δ) exposure represents the change in FEV_1 or FVC from baseline to post-exposure, and Δ exercise represents the change in FEV_1 or FVC from post-exposure to post-exercise (Figure 1). Paired t-tests were used for subsequent *post hoc* analysis. Significance was set at $p < 0.05$. All means are reported with standard deviations in parenthesis.

Results

Mean exposure data are presented in Table 1, temperature ($p = 0.115$) and humidity ($p = 0.296$) were not significantly between test days.

Mean baseline FEV_1, FVC and FEV_1/FVC values from both test days were 4.68 (0.66) L, 6.03 (0.89) L and 0.78 (0.06). Mean baseline percent predicted FEV_1, FVC and FEV_1/FVC values from both test days were 107.7 (8.9) %, 115.5 (10.5) % and 92.4 (7.2) %. There was a main effect

Table 1. Mean exposure data during a 60-min exposure to filtered air or diesel exhaust.

Condition	$PM_{2.5}$ (μg/m³)	CO (ppm)	NO (ppb)	NOx (ppb)	NO_2 (ppb)
Filtered air	13.31 (5.96)	1.85 (0.18)	36.50 (30.22)	60.53 (29.02)	24.03 (5.96)
Diesel	302.02 (11.85)	16.61 (3.60)	11394.61 (2213.30)	11802.49 (2342.57)	407.99 (134.39)

Values are displayed as mean (SD).

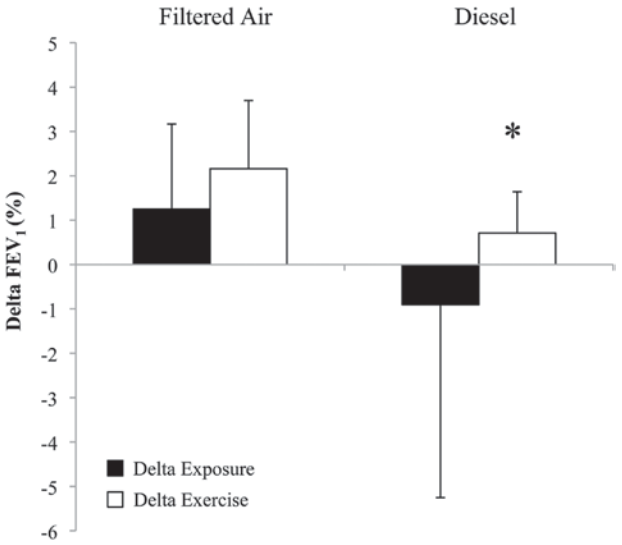

Figure 2. Mean change in FEV_1 in 8 endurance-trained males following exposure to filtered air or diesel exhaust and following a 20 km time trial. *Significantly different from Delta Exercise in filtered air ($p = 0.003$). Values above 0 indicate an improvement in FEV_1, values below 0 indicate a reduction in FEV_1. Delta Exposure represents the change in FEV_1 from baseline to post-exposure. Delta Exercise represents the change in FEV_1 from post-exposure to post-exercise. Values are depicted and mean (SD).

of exposure condition on the change in FEV_1 ($p = 0.008$) (Figure 2). *Post hoc* tests revealed that Δ exercise FEV_1 (the change in FEV_1 from post-exposure to post-exercise) was significantly attenuated in DE compare to FA (mean change = 2.2% (0.095 L) vs. 0.7% (0.03 L) for FA and DE respectively; $p = 0.003$). Figure 3 shows the percent change in FEV_1 in all eight participants.

As shown in Figure 4, there was a main effect of exposure condition on heart rate ($p = 0.023$). When participants performed a 20 km time trial that followed exposure to DE heart rate was over 6 beats/min (bpm) greater than exercise that followed FA.

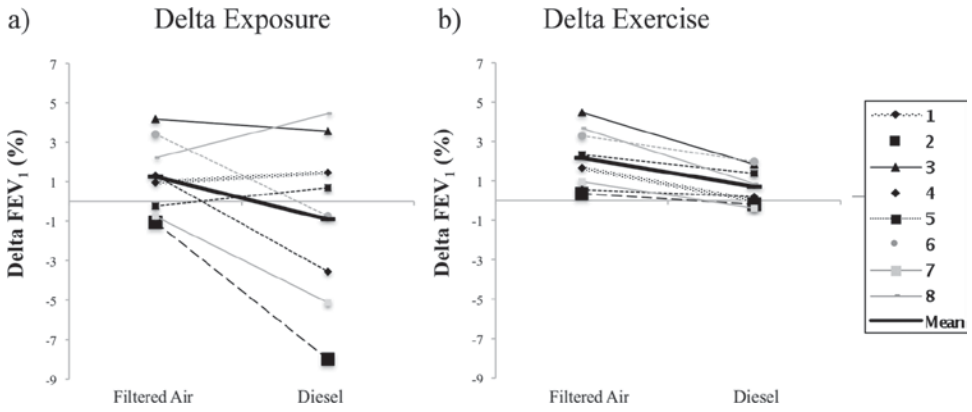

Figure 3. Change in FEV_1, in 8 endurance-trained males following exposure to filtered air or diesel exhaust and following a 20 km time trial. Values above 0 indicate an improvement in FEV_1, values below 0 indicate a reduction in FEV_1. Delta Exposure (a) represents the change in FEV_1 from baseline to post-exposure. Delta Exercise (b) represents the change in FEV_1 from post-exposure to post-exercise.

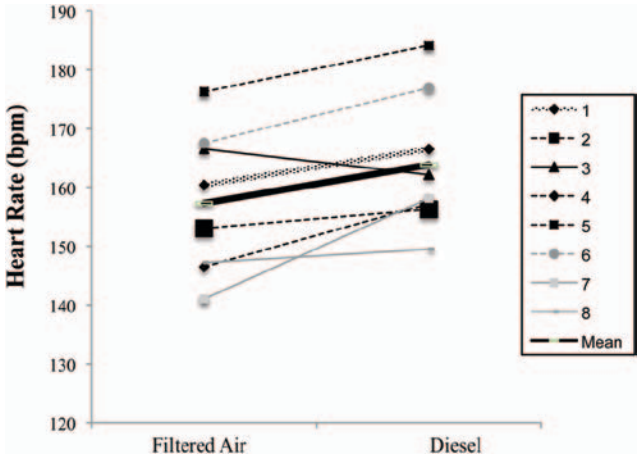

Figure 4. Mean heart rate in eight endurance-trained males during a 20 km time trial that followed a 60-min exposure to filtered air or diesel exhaust. There was a main effect of exposure condition on heart rate, $p = 0.023$.

There were no main effects of exposure condition on VO_2 ($p = 0.194$), VCO_2 ($p = 0.195$), V_E ($p = 0.36$), V_T ($p = 0.38$), F_B ($p = 0.58$), and SpO_2 ($p = 0.11$), during a 20 km time trial (Figure 5), or on FVC or time trial duration (1952.2 vs. 1980.6 s for DE and FA respectively, $p = 0.12$) (Figure 6).

Discussion

This is the first study to determine the effect of pre-exercise exposure to DE on 20-km cycling time-trial performance, pulmonary function, and cardio-respiratory parameters during exercise. In healthy endurance-trained males we found that pre-exercise exposure to DE did not impair exercise performance but attenuated exercise-induced bronchodilation, and increased exercise heart rate.

There was a main effect of condition on Δ exercise FEV_1, implying that DE attenuated exercise-induced bronchodilation. Some studies indicate that exposure to air pollution may affect pulmonary function immediately following exposure cessation (McCreanor et al., 2007; Gong et al., 2008; Rundell et al., 2008). In contrast, but similar to others,, we found that immediately following PM exposure there was no significant difference in FEV_1 between conditions (Gong et al., 1986; Ghio et al., 2000; Gong et al., 2008; Graff et al., 2009; Strak et al., 2010; Zuurbier et al., 2011). However, when the post-exposure lung function measure was delayed, PM exposure may affect FEV_1 (Gong et al., 2008; Strak et al., 2010). This indicated that the timing of post-exposure measures may be important and within the current study when pulmonary function was measured after exercise (approx 1-h post-exposure), DE exposure significantly affected lung function. Despite the differences in timing of exposure between the above studies and the current study (before exercise vs. during exercise), it could be suggested that the combination of exercise and exposure to air pollution could adversely affect pulmonary function. This is particularly important when considering travel to an exercise/competition location, as any pre-exercise exposure to particulate matter could adversely affect the pulmonary system.

Compared to FA, we found that pre-exercise exposure to DE significantly increased heart rate during exercise. Pulmonary irritants such as PM stimulate the sympathetic nervous system, and inhibit the parasympathetic nervous system, causing an increase in heart rate (Paton, 1998; Neas, 2000; Brook et al., 2010). In support of this, elevated PM_{10} levels in the previous 1–5 days have been associated with an increase in resting heart rate (Dockery et al., 1999). Therefore, it is possible that in the current study, the increase in exercise heart rate following DE exposure may be due to an up-regulation of sympathetic activity. In contrast to this study, Rundell et al. (Rundell and Caviston, 2008) did not find that heart rate was affected during exercise in a high PM_1 environment. However, the differing results between the current study and Rundell et al. (Rundell and Caviston, 2008), could be related to exposure duration (60 vs. 6 min), exposure timing (before exercise vs. during exercise) and fuel type (diesel vs. gasoline). Collectively, the above findings indicate that 60-min exposures to DE prior to exercise, and multi-day exposures to PM_{10} play a role in elevating heart

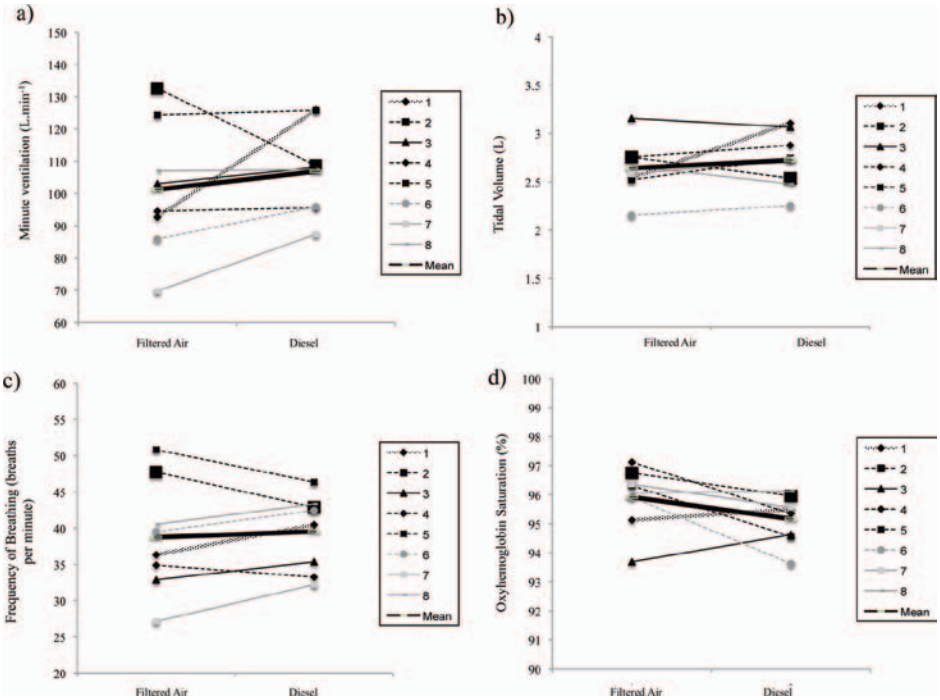

Figure 5. Minute Ventilation (a), tidal volume (b), frequency of breathing (c) and (d) oxyhemoglobin saturation in 8 endurance-trained men during a 20 km cycling time trial. Values depict mean (SD).

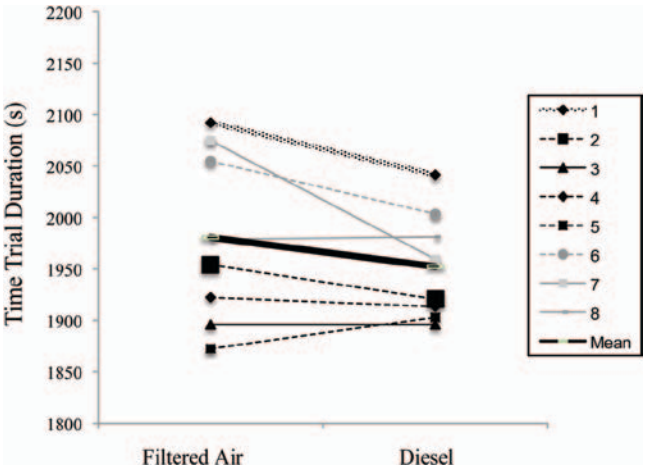

Figure 6. 20 km cycling time-trial duration in 8 endurance-trained males following exposure to filtered air or diesel exhaust.

rate. However, a very brief exposure to gasoline exhaust during exercise may not. While this elevated heart rate may not be problematic for healthy individuals during exercise, it may be of concern to those with pre-existing cardiovascular disease who cannot accommodate the additional cardiovascular strain.

To mimic travel to an exercise location we chose to expose individuals to DE prior to exercise. As method of travel can significantly alter individual exposure to pollutants (Kingham et al., 1998; McNabola et al., 2008; Kaur and Nieuwenhuijsen, 2009; Zuurbier et al., 2010), we reasoned that any PM exposure experienced en route to an exercise location might cause physiological changes, which could also affect exercise performance. However, we found that 20 km time trial performance was not significantly impaired following exposure to DE. This contrasts with Rundell et al. (Rundell and Caviston, 2008) and Cutrufello et al. (Cutrufello et al., 2011), who found that exercise performance was significantly impaired with high PM_1 exposure. During the experiment by Rundell et al. (Rundell and Caviston, 2008) participants were exposed to 2 low PM_1 days separated by 3 days, then a 7-day washout period followed by 2 high PM_1 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.

Pre-exercise exposure to DE did not significantly alter ventilatory parameters during exercise. Gong et al. (Gong et al., 1986) found that cyclists exposed to high levels of ozone during exercise showed significant decreases in peak V_E VO_2 and V_T. Based on this finding, we reasoned that DE exposure would irritate the airways, during exercise this would be manifested as a higher respiratory rate and a smaller tidal volume, however, this was not the case.

It is possible that some of the health effects observed in this study could be due to components of DE other than the PM. However, using a particle trap during a DE exposure (300 μg/m^3 $PM_{2.5}$) reduced any observed cardiovascular impairments (Lucking et al., 2011). Gases such as nitrogen dioxide (NO_2) have pulmonary and cardiovascular consequences, however, concentrations of NO_2 greater than 1000 ppb are necessary to induce

changes in pulmonary function in healthy adults (World Health Organisation, 2005), and levels of 4000 ppb do not adversely affect the cardiovascular system in healthy adults (Langrish et al., 2010). Within the current study levels of NO_2 were only 407.99 ppb during DE exposure.

With a significant proportion of the population exercising in climate-controlled environments following a period of DE/PM exposure, this study provides a better understanding of the interactive effects of exercise and DE inhalation on exercise performance, pulmonary function and cardio-respiratory parameters during exercise. The higher exercise heart rate may be of concern for diseased or at-risk populations. Interestingly, athletes exposed to high levels of PM or DE prior to exercise may not experience an immediate decrement in performance.

Future research should investigate the effects of pre-exercise exposure on other cardiovascular, respiratory and systemic parameters, such as endothelial and cardiac function, and lung and systemic inflammation. Additionally the effects of PM exposure during exercise on cardio-respiratory parameters and endurance performance would be beneficial. The study was limited to males only and therefore future research should aim to include females.

Conclusion

Much of the research investigating exercise and air pollution focuses on exposure to air pollution during exercise however, we are aware that physiological health effects occur beyond cessation of exposure. Therefore this research attempts to understand the physiological and exercise performance implications of exposure to DE prior to exercise. For athletes and exercising individuals it is important to understand the exercise and health implications of their environment not only during exercise but also prior to exercise. This study addressed how the respiratory system and heart rate are affected by air pollution exposure prior to exercise, and suggested that a 60-min exposure to DE (300 $\mu g/m^3$ of $PM_{2.5}$) prior to exercise significantly attenuated exercise-induced bronchodilation and increased heart rate during exercise. Pre-exercise exposure to DE did not significantly alter breathing pattern during exercise or impair 20 km cycling time trial performance in endurance-trained males.

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Declaration of interest

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