Particulate matter (PM) from diesel exhaust (DE) is damaging to health, and has been associated with myocardial infarction, stroke, atherosclerosis, bronchitis and asthma (1-6). Fire fighters, airport and ferry employees, police officers, construction workers, mining workers, vehicle testing workers and mechanics may be exposed to high levels of PM at work. Exposures in these jobs are typically higher than ambient concentrations and can range from 106-3300µg/m³ PM$_{2.5}$ (7-10). In a physical job demands analysis (11), many of the above workers are not sedentary and are required to perform moderate levels of physical exertion as part of their job. Physical exertion increases particle deposition (12), and as exertion increases predominantly oral breathing (13) may further increase particle deposition. Some research implies that PM exposure during physical exertion can affect the cardiovascular and respiratory system (14-16). If we assume that any adverse health effects are related to dose, these may be exacerbated as exertion levels increase. Given the well-established benefits that physical activity has on disease prevention understanding the relationship between exertion and air pollution has important implications.

**Purpose:** To determine the effect of DE exposure during different exertion levels on vascular function, lung inflammation, heart rate variability and white blood cell count (WBCC).

**Hypothesis:** We hypothesize that DE exposure will impair vascular function and heart rate variability, and increase lung inflammation and white blood cells. These responses will be exacerbated as exertion levels increase.

**Methods:** Eight males, age 23 (7) yrs, height 1.79 (0.76) m, weight 74.2 (10.5) kg (mean (sd)), participated in the study. Participants attended the lab on 7 occasions and exertion levels were precisely controlled using a stationary bike. Following a familiarization session the remaining six sessions consisted of a 30-minute exposure to either DE (300µg/m³ of PM$_{2.5}$) or filtered air while the participant was resting, or performing low or high intensity exertion. Low and high intensity exertion levels were set at 30% (96w) and 60% (192w) of the maximum work that was achieved during the familiarization session. Both the intensity of exertion (rest, low or high) and environmental condition (DE or filtered air) were randomized. Diesel exhaust was generated in a previously validated exposure chamber. All outcomes measures were taken prior to and 2-hours post exposure.
Outcome measures

Heart rate variability: heart rate was recorded for 5 minutes (Polar S810). Heart rate variability was analyzed offline using custom software (Kubios HRV, Kuopio, Finland), that performed time and frequency domain analysis, including VLF, LF, and HF power, LF:HF, RMSSD, pNN50, RR triangular index. Fraction of Exhaled Nitric Oxide (FeNO) was measured using a NIOX MINO® Airway Inflammation Monitor, as a surrogate for airway inflammation. White blood cell count was measured in venous blood. Vascular function: was measured using a validated technique known as flow mediated dilation (FMD) and was calculated as the % change in brachial artery diameter from baseline to peak diameter following cuff deflation. Brachial artery diameter was recorded for 1 minute prior to, and 3 minutes following a 5-minute forearm occlusion at 200mmHg. Videos were then analyzed using a custom-designed edge detection and wall-tracking software (Medical Image Applications, Vascular Research Tolls 5, Coralville, IA). DOSE of PM$_{2.5}$ and particle number concentration (PNC) were calculated as the mean minute ventilation multiplied by the mean PM$_{2.5}$ concentration and PNC during tests. Data and statistical analysis: Delta values for each variable were calculated as the difference from baseline and then used for analysis. Data were analyzed using a 2-condition (DE vs. Filtered air) x 3-intensity (rest vs. 30% vs. 60%) repeated measures ANOVA.

Results

Estimated particulate dose increased with the intensity of exertion. Mean FMD, brachial artery diameter, peak diameter and time to peak dilations across all test days was 6.35 (2.8) %, 4.35 (0.3) mm, 4.62 (0.4) mm and 57.62 (16.1) s respectively. Mean FeNO on all testing days was 16.0 (5.4) ppb. There were no main effects of condition or intensity on delta FMD, baseline or peak brachial artery diameter, FeNO or heart rate variability. There was a main effect of exertion intensity on WBCC (p=0.001), neutrophils (p=0.000), and monocytes (p=0.013); however, DE exposure did not modify this.

Conclusions: Thirty minutes of exertion in DE does not acutely affect vascular function, FeNO, or affect heart rate variability. High intensity exertion significantly increases WBCC, neutrophils and monocytes; however, this response is not modified by DE exposure. This preliminary data implies that air pollution exposure during different exertion levels does not acutely modify...
certain cardiovascular, systemic and respiratory endpoints in healthy individuals. Suggesting that short duration, low and high intensity exertion in a work place with high levels of PM may not acutely affect aspects of inflammation, vascular function or cardiac autonomic control. However, more research is required to determine the effects of longer exposures to DE with exertion, as well as monitoring of physiological changes for a longer duration post exposure.