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Exposure to air pollution near a steel plant and effects on cardiovascular physiology: A randomized crossover study

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ABSTRACT

Background: Iron and steel industry is an important source of air pollution emissions. Few studies have investigated cardiovascular effects of air pollutants emitted from steel plants.
Objective: We examined the influence of outdoor air pollution in the vicinity of a steel plant on cardiovascular physiology in Sault Ste. Marie, Canada.
Methods: Sixty-one healthy, non-smoking subjects (females/males = 33/28, median age 22 years) spent 5 consecutive 8-hour days outdoors in a residential area neighbouring a steel plant, or on a college campus approximately 5 kilometres away from the plant, and then crossed over to the other site with a 9-day washout. Mid day, subjects underwent daily 30-minute moderate intensity exercise. Blood pressure (BP) and pulse rate were determined daily and post exercise at both sites. Flow-mediated vasodilation (FMD) was determined at the site near the plant. Air pollution was monitored at both sites. Mixed-effects regressions were run for statistical associations, adjusting for weather variables.
Results: Concentrations of ultrafine particles, sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and carbon monoxide (CO) were 50–100% higher at the site near the plant than at the college site, with minor differences in temperature, humidity, and concentrations of particulate matter $\leq$2.5 μm in size ($PM_{2.5}$) and ozone (O_3). Resting pulse rate [mean (95% confidence interval)] was moderately higher near the steel plant [+1.53 bpm (0.31, 2.78)] than at the college site, male subjects having the highest pulse rate elevation [+2.77 bpm (0.78, 4.76)]. Resting systolic and diastolic BP and pulse pressure, and post-exercise BP and pulse rate were not significantly different between two sites. Interquartile range concentrations of SO_2 (2.9 ppb), NO_2 (5.0 ppb) and CO (0.2 ppm) were associated with increased pulse rate [0.19 bpm (−0.00, 0.38), 0.86 bpm (0.03, 1.68), and 0.11 bpm (0.00, 0.22), respectively], ultrafine particles (10,256 count/cm^3) associated with increased pulse pressure [0.85 mmHg (0.23, 1.48)], and NO_2 and CO inversely associated with FMD [−0.14% (−0.31, 0.02), −0.02% (−0.03, −0.00), respectively]. SO_2 during exercise was associated with increased pulse rate [0.26 bpm (0.01, 0.51)].
Conclusion: Air quality in residential areas near steel plants may influence cardiovascular physiology.

Introduction

In Canada, major industrial sectors contribute approximately half of air pollution emissions (Environment Canada, 2010). Given that many people live in proximity to industrial areas, it is important to understand the health impact of emissions from these point sources.

The iron and steel industry is a major industrial sector with emissions regulated by the government of Canada (Environment Canada, 2007). Although many studies have examined the adverse health effects of outdoor air pollution in urban centres (HEI International Scientific Oversight Committee, 2010; Stieb et al.,

Abbreviations: BAD, basal brachial artery diameter; BP, blood pressure; bpm, beats per minute; 95% CI, 95% confidence interval; CO, carbon monoxide; DBP, diastolic blood pressure; FMD, flow-mediated vasodilation; IQR, interquartile range; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter at aerodynamic diameter $\leq$2.5 μm; PP, pulse pressure; PR, pulse rate; SBP, systolic blood pressure; SO_2, sulphur dioxide; UFP, ultrafine particles.

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2002; WHO Working Group, 2003), few have explored the health impacts of air pollution from iron and steel industry emissions. Pope reported that in the Utah Valley hospital admissions for severe respiratory diseases decreased significantly during 1986–1987 when the local steel mill was closed (Pope, III, 1989). During periods of operation, concentrations of particulate matter with aerodynamic diameter $\leq$10 μm (PM_{10}) were associated with decreased pulmonary function and increased respiratory symptoms in children (Pope, III and Dockery, 1992). Similarly in Hamilton Ontario Canada, an area with concentrated iron and steel industry, total suspended particles in ambient air were associated with increased respiratory symptoms among children (Pouliou et al., 2008).

In general, existing evidence suggests that steel mill emissions may have negative effects on respiratory health (Pope, III, 1989; Pope, III and Dockery, 1992; Pouliou et al., 2008), but little is known about the potential cardiovascular health effects of emissions from this point source. Outdoor air pollution has been linked to increased cardiovascular hospital admissions (Burnett et al., 1995) and mortality (Villeneuve et al., 2003), and changes in blood pressure and heart rate (Brook et al., 2009; Ebelt et al., 2005; Ibald-Mulli et al., 2004; Liu et al., 2007; Urch et al., 2005; Zanobetti et al., 2004) and brachial artery function (Brook et al., 2011; Mills et al., 2005; O'Neill et al., 2005). Blood pressure, heart rate and brachial artery flow-mediated vasodilation (FMD) are physiological measurements that have been used clinically to evaluate changes in the autonomic activity of the heart and vascular system (Grassi, 2009; Joyner et al., 2010; Wallin and Charkoudian, 2007). Increased blood pressure (Franklin et al., 1999; Sesso et al., 2000) and heart rate (Hjalmarson, 2007; Singh, 2003), and reduced FMD (Corretti et al., 2002; Yeboah et al., 2007) have been established as risk factors contributing to the development of cardiovascular disease. In this study, we explored whether acute exposure to outdoor air pollution related to iron and steel plant emissions also influenced the cardiovascular physiology of human subjects using measurements of blood pressure, pulse rate and brachial artery FMD.

Materials and methods

Study design

A randomized controlled crossover design was carried out between the end of May and August 2010, in Sault Ste. Marie, Ontario, Canada. Each subject was randomly assigned to spend five consecutive eight-hour days outdoors in a residential neighbourhood at the north-west edge of a steel plant property (Bayview site), or on a college campus (College site) about five kilometres north-east from the plant (Fig. 1), and then crossed over to the other site with an intervening nine-day washout period to minimize potential effect carry-over. The plant had ten stacks, with two major stacks shown in Fig. 1. The Bayview site was within 100 metres of a major stack and within 500 metres of perpetually active coke ovens. On a study day, subjects arrived at the site with a 30-minute lag from one another, with the first subject arriving at 7:30 am, and the last subject arriving at 10:30 am. At both sites, an overhead awning protected subjects from direct sun exposure and precipitation. In the mornings prior to the exposure period and during lunch time, subjects ate low nitrate (avoid cured meats, bacon) and low fat meals. Subjects were at rest for the majority of the day, but at 2.5 hours post arrival they exercised for thirty minutes on an elliptical trainer once daily aiming for a heart rate of 65% the predicted maximum calculated as 220 – age (years). During the washout period, the subjects carried on their daily lives while avoiding occupational exposure to elevated air pollution. The study protocol was approved by the Research Ethics Boards of Health Canada and the Algoma University.

Study subjects

Subjects were recruited through advertising on local university and college campuses. Inclusion criteria were non-smoking men and women, aged 18–55 years, with no cigarette smoke exposure at home, no occupational exposure to iron and steel industry or traffic, no history of chronic cardiovascular and respiratory disease, diabetes mellitus, musculoskeletal disease, or exercise limiting disorders, and free of medications that could influence cardiovascular or respiratory function. Pregnant or breast-feeding women were excluded from the study along with residents living in the neighbourhood bordering on the steel plant property. All participants provided informed written consent prior to participating in the study. A questionnaire was administered during the first visit to collect demographic information along with medical history, and medication use.

Measurement of environmental variables

Environmental variables were determined between 8:00 and 18:00. A fixed-site ambient air quality monitor (Air Pointer® recordum Messtechnik GmbH, Jasomirgottgasse 5 City: A 2340 Mödling, Austria) was used at each of the two sites to measure hourly ambient concentrations of particulate matter at aerodynamic diameter $\leq$2.5 μm ($PM_{2.5}$, by nephelometry), sulphur dioxide (SO_2, by ultraviolet fluorescence), nitrogen dioxide (NO_2, by chemiluminescence), and ozone (O_3, by ultraviolet photometry). Ultrafine particles (UFP) (particle size 0.01–0.1 μm) were measured using a TSI® Model 3007 Ultrafine Particle Counter (http://www.tsi.com/condensation-particle-counter-3007/; Access date: January 2013). Continuous temperature and relative humidity measurements were also taken using the Air Pointer. An electronic anemometer at the study site was used to measure hourly wind direction and speed.

Measurement of blood pressure, pulse rate and flow-mediated vasodilation

Subjects were seated quietly with the arm being supported at heart level. Systolic and diastolic blood pressure was determined using a BPTRU-200 blood pressure monitor (BpTRU Medical Devices, Coquitlam, BC, Canada), following the American Heart Association's recommendations (Pickering et al., 2005). The instrument automatically took 6 consecutive readings of systolic and diastolic blood pressure, discarded the first reading and provided the mean of the remaining five readings. We calculated pulse pressure (PP), the difference between systolic and diastolic blood pressure. Pulse rate was determined using OxiMax N65 Pulse Oximeter (Covidien, Dublin, Ireland). Three readings of pulse rate were recorded with 10 seconds in between each reading. We used the mean of three readings as a representative of pulse rate for data analysis. Blood pressure and pulse rate were determined 2 hours post arrival at a site (morning), immediately post exercise, and then 5 hours post arrival (afternoon). We calculated percent changes from morning to afternoon in blood pressure and pulse rate, using formula = 100 × (afternoon value − morning value)/morning value.

Flow-mediated endothelium-dependent vasodilation (FMD) was measured using a Vivid i BT09 portable ultrasound machine (GE Healthcare, Chalfont St. Giles, Buckinghamshire, UK), following the guidelines of the American College of Cardiology (Corretti et al., 2002). Before the measurement was done the subject had fasted for approximately 2 hours. The measurement was conducted in the afternoon 6 hours after arrival at the site, a time that was logistically feasible while giving subjects the longest possible exposure period during the day. Subjects lay down in a supine position, and two-dimensional scans of their basal brachial artery diameter