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ARTICLE

Associations between air pollution and cardio-respiratory physiological measures in older adults exercising outdoors

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ABSTRACT

We examined whether exercising indoors vs. outdoors reduced the cardio-respiratory effects of outdoor air pollution. Adults ≥55 were randomly assigned to exercise indoors when the Air Quality Health Index was ≥5 and outdoors on other days (intervention group, n = 37), or outdoors everyday (control group, n = 35). Both groups completed cardio-respiratory measurements before and after exercise for up to 10 weeks. Data were analyzed using linear mixed effect regression models. In the control group, an interquartile range increase in fine particulate matter ($PM_{2.5}$) was associated with increases of 1.4% in heart rate (standard error (SE) = 0.7%) and 5.6% (SE = 2.6%) in malondialdehyde, and decreases of 5.6% (SE = 2.5%) to 16.5% (SE = 7.5%) in heart rate variability measures. While the hypothesized benefit of indoor vs. outdoor exercise could not be demonstrated due to an insufficient number of intervention days (n = 2), the study provides evidence of short-term effects of air pollution in older adults. ISRCTN #26552763.

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KEYWORDS
Air pollution; exercise; cardiovascular physiology; respiratory function tests

Introduction

Air quality indices (AQIs) and advisories are provided based on the premise that they furnish information that people can use to reduce their exposure to air pollution and, as a consequence, the risk of adverse health effects. The Air Quality Health Index (AQHI) is an aggregate measure of nitrogen dioxide (NO_2), ozone (O_3) and fine particulate matter ($PM_{2.5}$) which was developed to address identified deficiencies in existing AQIs, notably their inability to reflect potentially additive effects among multiple pollutants and the occurrence of adverse effects at low levels of exposure, i.e. without a threshold (Stieb et al. 2008a). We previously reported associations of the AQHI and individual air pollutants with cardio-respiratory physiological measures in panel

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Supplemental data for this article can be accessed here.

studies of summer and winter outdoor physical activity in older adults conducted in a predominantly rural area and small northern industrial city, respectively (Stieb et al. 2017, 2018). These findings provided evidence supporting the utility of the AQHI in predicting health risks for diverse health outcomes and types of communities not accounted for in developing the AQHI, which was based on the effects of air pollution on mortality in large urban centres. However, empirical evidence to support the effectiveness of AQIs and advisories in actually reducing exposures and health risks is mixed (Bickerstaff and Walker 2001; Semenza et al. 2008; Stieb et al. 2008b; Smallbone 2009, 2015; Wen et al. 2009; Neidell 2009; Maheswaran et al. 2010; Licskai et al. 2013; Mullins and Bharadwaj 2015; Radisic et al. 2016; Lyons et al. 2016; D'Antoni et al. 2017; Chen et al. 2018) and to our knowledge there have been no previous experimental studies based on individual-level data. The present study was designed as a randomized controlled trial of the AQHI in which the intervention comprised advising participants to exercise indoors rather than outdoors on days when the maximum AQHI was forecast to be 5 or higher. We hypothesized that as a result of reducing exposure to outdoor air pollution on designated days, associations of physiological measures with air pollution in the intervention group would be attenuated relative to the control group. During the study period of approximately 70 days, however, there were only 2 intervention days, substantially fewer than anticipated based on historical data. Nonetheless, we analyzed the data according to original group assignment, in keeping with intention to treat analysis. Given this limitation, our results cannot readily address our hypothesis but may provide additional evidence of the AQHI as a predictor of health risk in diverse settings.

Materials and methods

Methods were described in detail in our previous papers reporting findings from our summer and winter panel studies (Stieb et al. 2017, 2018). They are summarized briefly here.

Study location and participant recruitment

The study was conducted in London, Ontario, a city of approximately 500,000 in southwestern Ontario. It has no large industrial emitters but is situated along the busy Quebec City–Windsor transport corridor and is subject to regional smog episodes related to long-range transported pollutants from the US and elsewhere in southern Ontario (City of London 2018; Middlesex-London Health Unit 2019). Data were collected during June through early September of 2015. To avoid biasing study participants in relation to perceived air quality, the study hypothesis was not disclosed. Participants were told that the study pertained to indoor and outdoor physical activity and health. Inclusion criteria were: age ≥55 years old, non-smokers, non-exposed at home to environmental tobacco smoke and without seasonal allergies. Exclusion criteria were: unstable angina, atrial flutter, atrial fibrillation, paced rhythm, left bundle branch block, an implanted cardioverter-defibrillator, or allergy to latex or adhesives. The study was approved by Health Canada and Western University Research Ethics Boards and written consent was obtained from all study participants. The International Standard Randomised Controlled Trial Number (ISRCTN) is 26552763.

Exposure assessment

A dedicated Airpointer® (Recordum Messtechnik GmbH, Vienna, Austria) monitor was deployed at the site used for weekly health measures, recording continuous hourly measures of carbon monoxide (CO), NO_2, O_3, $PM_{2.5}$, sulphur dioxide (SO_2) and temperature. Missing values were filled using data from a nearby (6.7 km) National Air Pollution Surveillance program monitor. The AQHI is calculated according to Equation 1:

$$AQHI = \frac{10}{10.4} \times \left(100 \times \left(e^{0.000871 \times NO_2} - 1 + e^{0.000537 \times O_3} - 1 + e^{0.000487 \times PM_{2.5}} - 1\right)\right) \quad (1)$$

where all pollutants are entered as 3 h moving average concentrations in ppb (NO_2, O_3) or μg/m^3 ($PM_{2.5}$) (Stieb et al. 2008a).

Collection of health data

Personal characteristics, health history data and housing characteristics were determined at study enrolment using a baseline health questionnaire, and daily and weekly questionnaires documented medication use, symptoms, indoor exposures and outdoor activity. Participants completed daily measurements of blood pressure and peak expiratory flow rate (PEFR), and weekly measurements of heart rate variability (HRV), reactive hyperemia index as a measure of endothelial function, spirometry, fraction of exhaled nitric oxide (FeNO) and urinary malondialdehyde (MDA) before and after 30 min of prescribed physical activity. Pre-exercise measures for daily (at home) exercise were carried out immediately prior to exercise after sitting for 15 min. Postexercise measures were carried out after sitting for 15 min following exercise. For weekly measures, pre-exercise measures were carried out up to 1.5 h before and 2 h after exercise. Details of instrumentation and measurement protocols are described elsewhere (Stieb et al. 2017). We asked participants to engage in daily light exercise (e.g. walking), at the same time, location and level of effort each day. We advised them not to exercise outdoors when there was a severe weather warning (e.g. thunderstorm). On days when participants underwent weekly measurements, prescribed walking routes were employed. On other days, exercise was unsupervised, but participants recorded the duration, time of day and type of activity. We monitored each participant for up to 10 weeks with weekly measurements carried out at the same time of day and day of the week.

Study participants assigned to the intervention group were advised to exercise indoors rather than outdoors on days when the maximum AQHI was forecast to be 5 or higher, while those assigned to the control group exercised outdoors. Group allocation was carried out using block random allocation with random block sizes of 2 and 4, stratified by gender. Allocation was carried out by Health Canada study coordination staff using participant ID numbers provided by field staff. The intervention group received instructions for a simple indoor exercise routine (stationary walking) that could be completed at home or at the weekly testing site on designated days. Participant blinding and placebo control were not feasible. However, participants in both groups were instructed not to disclose their group status to other participants or field staff conducting health measurements, such that the study was single-blind. At the weekly testing site, a separate room was provided for study participants where those in the intervention group could complete their indoor exercise routine out of view of field staff while control group participants exercised outdoors. Flow of participants was supervised by a field staff member who was aware of group assignment, but not involved in health measurements. The risk of contamination of the control group as a result of public AQHI information was low, since AQHI information for London was not highly publicized at the time. Moreover, advice to reduce outdoor activity for otherwise healthy individuals does not appear until AQHI values of 7 or greater and our experience in previous rounds of data collection is that participants are highly committed to completing their daily outdoor activity, even when outdoor conditions are inclement. Intervention days occurred July 29 and September 2.

Statistical analysis

Prior to modeling associations with air pollution, outcome variables were log transformed if necessary to reduce skew. Associations with both pre and postexercise measures were examined.

We employed linear mixed effect regression models in order to account for repeated measures among study participants. The model is represented by Equation 2:

$$y = X\beta + Z\gamma + \varepsilon \tag{2}$$

where y is a vector of observed responses, X is the design matrix of fixed effects, β is a vector of fixed effect parameters, Z is the design matrix of random effects, γ is a vector of random effects parameters and ε is a vector of random errors (SAS Institute Inc 2015). Participants were treated as random effects and time-invariant individual covariates as well as time-variant environmental variables were treated as fixed effects. Age, sex, Body Mass Index (≤25, >25), smoking history (never, ever), and dichotomous variables for medication use (statins, other cardiovascular drugs) were included as covariates in all models. Height and use of respiratory medication were also included in spirometry models. First-order autoregressive models were used to account for serial correlation. We accounted for effects of time (trend and temporal cycles) using a linear function of time and day of week variables. In a previous study, we found no consistent associations with sub-daily air pollution exposures in the hours prior to health measures (Stieb et al. 2017), thus only daily 3-h maximum exposures (daily maximum of 3-h trailing averages) were considered. Air pollution variables were entered into models at individual lags of 0–2 days, each with natural spline functions of temperature with 3 degrees of freedom at individual lags of 0–2 days (9 models per pollutant). Effect of group assignment was determined by including an interaction between group and air pollution concentration. Percent change in health measures associated with air pollution was calculated according to equations 3 and 4:

For untransformed variables,

$$\Delta y(\%) \cong 100 \times (\beta \times \Delta x)/\bar{y} \tag{3}$$

and for log-transformed variables,

$$\Delta y(\%) = 100 \times (e^{\beta \times \Delta x} - 1) \tag{4}$$

where β is the regression coefficient, Δx is the increment in pollution concentration and $\bar{y}$ is the mean value of the health measure. Since percent change was calculated differently for log-transformed and untransformed variables, they cannot be compared directly (Buteau and Goldberg 2016). Statistical analyses were conducted in SAS EG (64 bit) version 5.1 (SAS Institute, Cary, NC, USA) and R version RX64 3.2.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Figure 1 summarizes allocation of participants to study groups. More participants in the intervention (n = 6) than control group (n = 1) withdrew before completing the full duration of the study. Table 1 summarizes participant characteristics. The control and intervention groups were similar with respect to age, sex, race, previous smoking and prevalence of chronic disease, but overweight (BMI 25–29), asthma and air conditioning were more prevalent in the control group.

Table 2 summarizes air quality and temperature data during the study. Pollutant levels were generally quite low in comparison to historical data for southern Ontario. There was 1 day (August 17) when an extreme heat advisory was issued and participants were encouraged to complete their exercise during the morning or evening hours and not during peak heat hours, or not to complete their outdoor exercise if they felt it was too hot for them and would impact their safety.

Table 3 presents the distributions of cardio-respiratory physiological measures in control and intervention groups. There were no striking differences between groups.

Supplemental Table S1 shows the percent change in cardiovascular outcomes and MDA per pollutant interquartile range, by group and pre vs. postexercise period. Results for the air pollution lag time for which the strongest effect was observed (largest t-ratio regardless of direction) in the

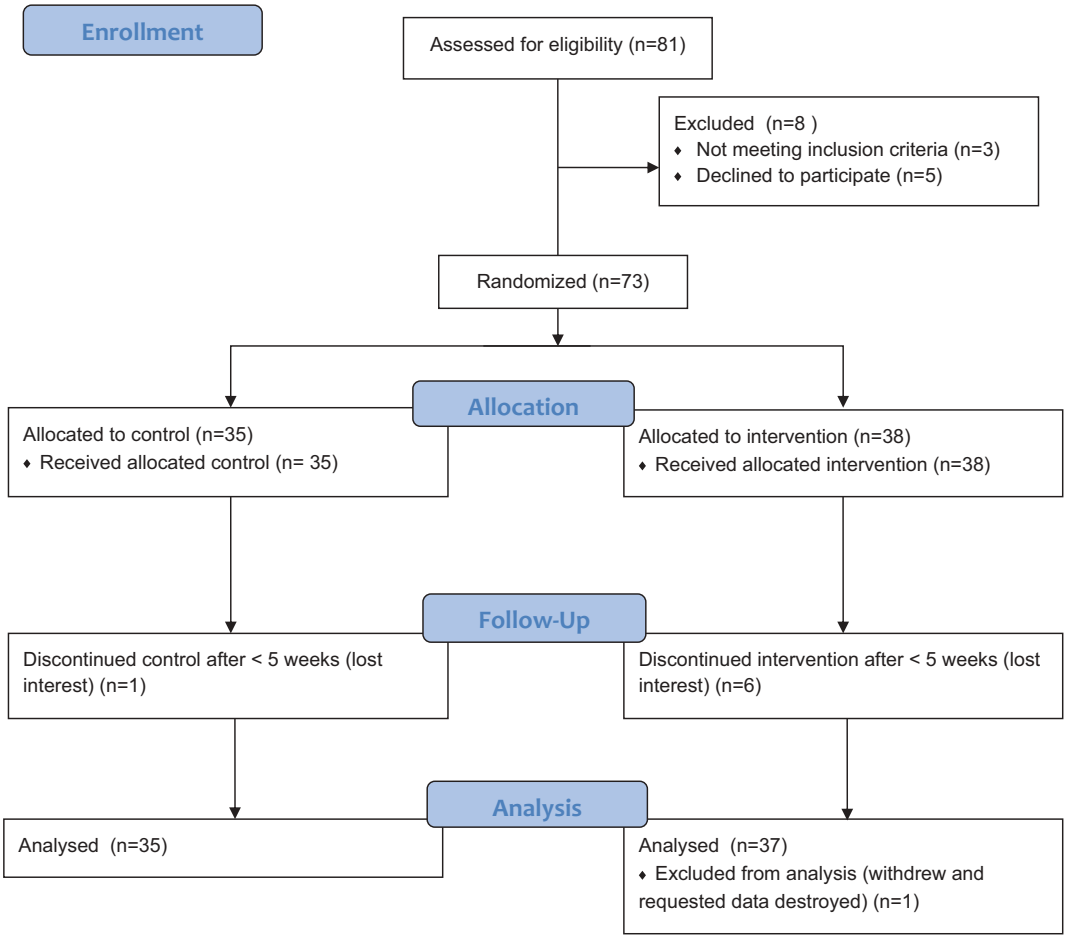

Figure 1. CONsolidated standards of reporting trials (CONSORT) flow diagram.

Table 1. Baseline characteristics of participants*.

	Control (n=35)	Intervention (n=37)
Female n (%)	20 (57.1)	20 (54.1)
Mean age (range)	65 (55-79)	66 (55-81)
Race n (%) Caucasian	35 (100.0)	33 (89.2)
Ever smoked n (%)	17 (48.6)	21 (56.8)
BMI		
<25 n(%)	11 (31.4)	18 (48.6)
25-29 n (%)	18 (51.4)	13 (35.1)
30+ n(%)	6 (17.1)	6 (16.2)
Statins n (%)	7 (20.0)	9 (24.3)
Other cardiovascular drugs n (%)	12 (34.3)	13 (35.1)
Asthma n (%)	4 (11.4)	0 (0.0)
Heart disease n (%)	2 (5.7)	2 (5.4)
Single family home n (%)	21 (60.0)	22(59.5)
Air conditioning n (%)	34 (97.1)	31 (83.8)
Dehumidifier n (%)	18 (51.4)	15 (40.5)

*includes 1 control participant and 6 intervention participants who participated for less than half of the study (<5 weeks).

Table 2. Descriptive summary of air pollution data (daily 3-h maximum).

				Percentile					
Variable*	n	Mean	Standard Deviation	0	25	50	75	100	Interquartile Range
AQHI	81	3.0	0.8	1.2	2.5	3.0	3.4	4.7	0.89
CO (ppm)	73	0.2	0.1	0.1	0.1	0.2	0.2	0.5	0.09
NO_2 (ppb)	81	6.6	2.7	2.4	4.5	6.0	8.2	14.6	3.69
O_3 (ppb)	81	43.8	10.3	14.7	36.9	44.4	52.1	62.8	15.23
$PM_{2.5}$ ($\mu g/m^3$)	81	15.2	9.0	3.1	7.4	13.2	22.3	40.1	14.87
SO_2 (ppb)	74	1.3	1.1	0.0	0.6	1.0	1.6	7.4	1.02
Temperature (°C)	81	24.6	3.3	15.6	22.5	24.2	27.3	31.1	4.86

*AQHI-Air Quality Health Index, scale is 1–3, low health risk, 4–6, moderate health risk, 7–10, high health risk, >10, very high health risk, CO-carbon monoxide, NO_2-nitrogen dioxide, O_3-ozone, $PM_{2.5}$-particulate matter of median aerodynamic diameter <2.5 µm, SO_2-sulfur dioxide.

Table 3. Descriptive summary of pre-exercise physiological measures data by group.

Measure*	Group	n	Mean	Standard Deviation	Percentile 25th	Percentile 75th
Daily						
Heart rate	Control	2026	66.7	10.6	60.0	72.0
(beats/min)	Intervention	1874	68.7	9.7	62.0	75.0
Systolic BP	Control	2026	121.2	17.3	107.3	133.3
(mm Hg)	Intervention	1877	126.0	15.8	115.7	134.7
Diastolic BP	Control	2025	71.1	9.5	63.7	77.7
(mm Hg)	Intervention	1873	74.2	9.0	67.0	81.0
FEV1	Control	2025	2.5	0.7	2.0	2.8
(L)	Intervention	1877	2.2	0.6	1.7	2.5
PEFR	Control	2026	7.6	2.0	6.2	9.1
(L/s)	Intervention	1878	7.3	2.0	5.5	9.0
Weekly						
Heart rate	Control	290	63.7	9.8	57.0	69.0
(beats/min)	Intervention	269	64.6	8.5	59.0	70.0
HF	Control	290	265.1	362.9	53.6	361.5
($msec^2$)	Intervention	269	186.8	199.5	67.7	244.2
LF	Control	290	416.1	587.9	112.8	504.8
($msec^2$)	Intervention	269	350.4	442.6	129.7	407.7
pNN50	Control	290	9.7	13.0	0.6	16.1
(%)	Intervention	269	6.7	8.8	0.9	8.5
rMSSD	Control	290	29.7	17.1	17.0	38.0
(msec)	Intervention	269	26.4	11.5	19.0	32.0
SDNN	Control	290	51.9	20.3	38.0	62.0
(msec)	Intervention	269	48.0	17.8	36.0	58.0
RHI	Control	287	2.1	0.7	1.6	2.4
	Intervention	270	2.3	0.7	1.7	2.7
MDA	Control	290	2.2	1.0	1.6	2.7
(nmol/mg creatinine)	Intervention	272	2.3	0.7	1.9	2.7
FEF25-75%	Control	259	2.0	0.7	1.4	2.4
(L/s)	Intervention	232	1.9	0.7	1.4	2.3
FEV1	Control	259	2.7	0.7	2.1	3.1
(L)	Intervention	232	2.5	0.6	2.1	3.0
FVC	Control	259	3.8	1.0	2.8	4.6
(L)	Intervention	232	3.5	0.9	2.9	4.1
PEFR	Control	259	6.6	1.7	5.4	7.6
(L/s)	Intervention	232	6.5	1.5	5.5	7.6
FENO	Control	279	25.4	18.2	13.5	30.3
(ppb)	Intervention	263	23.3	15.2	16.0	24.0

*BP, blood pressure; FEV_1, forced expiratory volume in 1 s; PEFR, peak expiratory flow rate; HF, high frequency power; LF, low frequency power; pNN50, percentage of successive normal cardiac interbeat intervals greater than 50 msec; SDNN, standard deviation of NN (normal RR) intervals; RMSSD, root mean square of successive differences; RHI, reactive hyperemia index; MDA, malondialdehyde; FEF25-75%, forced expiratory flow at 25–75% of forced vital capacity; FVC, forced vital capacity; FeNO, fraction of exhaled nitric oxide.

control or intervention group are shown, together with results for the same lag for the other group. Significant associations of air pollutants with increased heart rate and decreased heart rate variability measures were more commonly observed in the pre vs. postexercise period. In the pre-exercise period, significant associations were only observed in the control group (with the exception of RHI, e.g. 1.4% increases in heart rate (standard error (SE) = 0.7%) and 5.6% (SE = 2.6%) in MDA, and decreases of 13.8% (SE = 5.6%) in High Frequency power, 16.3% (SE = 5.5%) in Low Frequency power, 16.5% (SE = 7.5%) in percentage of successive normal cardiac interbeat intervals greater than 50 msec, 5.6% (SE = 2.5%) in root mean square of successive differences, and 5.8% (SE = 2.1%) in standard deviation of NN (normal RR) intervals, per interquartile range $PM_{2.5}$). In some instances, differences in effect size between control and intervention groups were significant ($p < 0.05$). Associations were generally similar in direction but smaller in magnitude in the postexercise period. Figure 2 illustrates the typical pattern of these associations, also showing the general lack of sensitivity of associations to the lag of temperature.

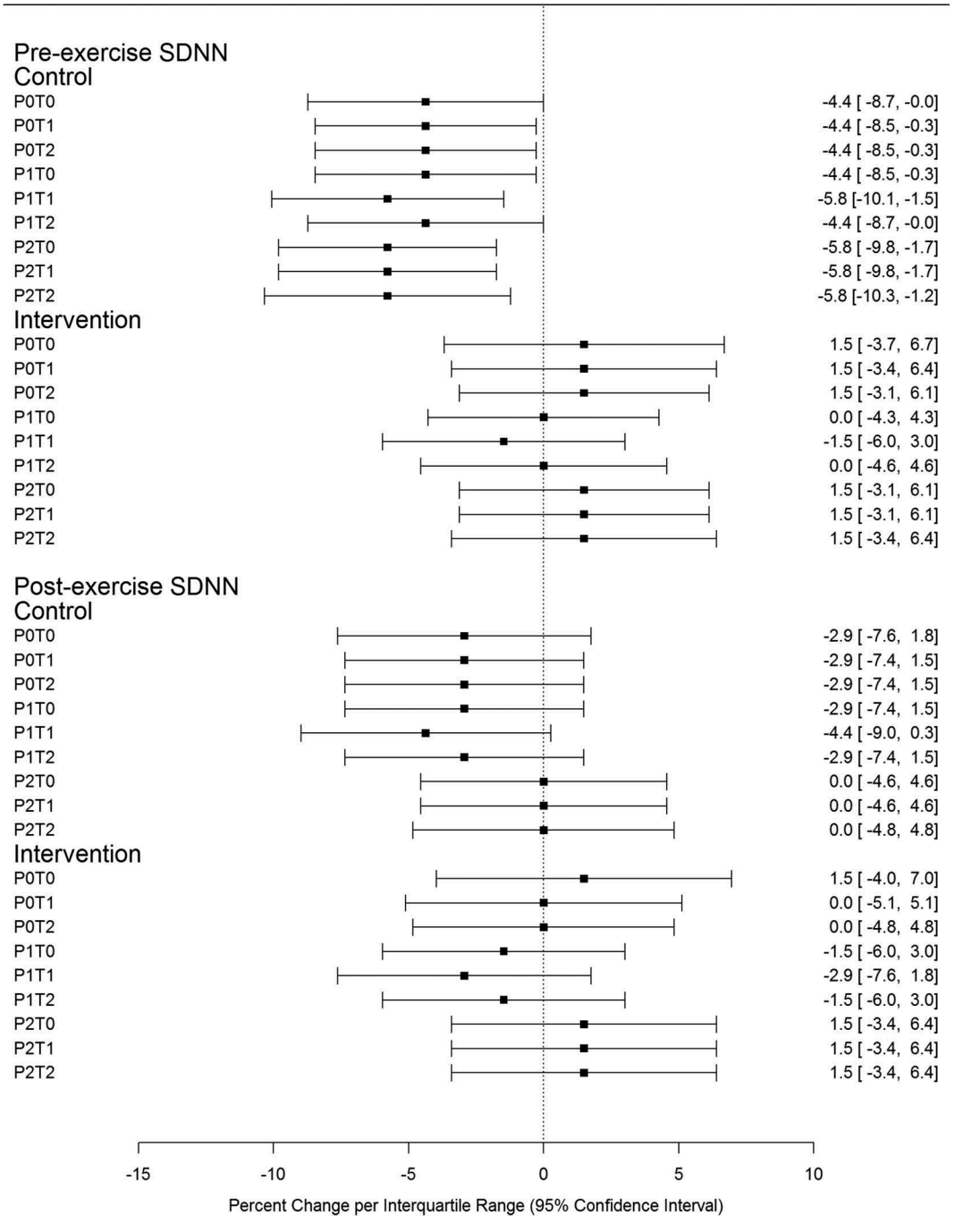

Figure 2. Percent change in standard deviation of NN (normal RR) intervals (SDNN) per $PM_{2.5}$ interquartile range, by pollutant (P) and temperature (T) lag in days.

Findings were generally less consistent for respiratory outcomes (see Supplemental Table S2). Significant increases in FENO were observed only in the intervention group in the preexercise period, while significant decreases were observed in the intervention group in the postexercise

period. No consistent associations were observed between air pollution and daily physiological measures (see Supplemental Table S3).

As reported in our earlier panel studies, we again observed significant improvements in several cardiovascular measures over the duration of the study, but only in the postexercise period (Figure 3). Of daily measures, only postexercise heart rate declined significantly during the study period (see Supplemental Figure S4). In a sensitivity analysis including an interaction term between group and day of study, there was no consistent evidence that changes over the duration of the study differed significantly between the intervention and control groups (not shown).

Discussion

We observed associations of the AQHI and individual air pollutants with several cardiovascular measures and a urinary marker of oxidative stress (MDA). These are consistent in magnitude and direction with those observed in our previous observational studies of summer and winter outdoor physical activity in older adults (Stieb et al. 2017, 2018), and provide additional evidence supporting the utility of the AQHI as a predictor of health risk for diverse health outcomes and types of communities not accounted for in developing the AQHI (Stieb et al. 2008a).

Given the small number of intervention days, our findings are difficult to interpret relative to our hypothesis that as a result of reducing exposure to outdoor air pollution on designated days, associations of physiological measures with air pollution in the intervention group would be attenuated relative to the control group. Associations between air pollution and weekly pre-exercise heart rate and heart rate variability measures as well as MDA were consistently stronger in the control group. A possible explanation is that intervention group participants, despite being instructed to exercise indoors on only 2 days, may have done so on other days, or may have been more cautious in their prescribed activity than control group participants. Also, more participants withdrew from the intervention group than the control group before completing the full duration of the study, which could have reduced statistical power. However, associations with RHI were stronger in the intervention group and associations with weekly respiratory and daily measures were inconsistent. There did not appear to be significant differences between the control and intervention group participants which would explain the differential air pollution associations in these groups. Eleven percent of the control group had asthma (vs. 0% in the intervention group), but one would not expect this to make the group more susceptible to cardiovascular effects of air pollution. There was also a higher prevalence of air conditioning in the control group (97% vs. 83%) but if anything this would be expected to confer greater protection from outdoor air pollution exposure in the control group. The prevalence of overweight (BMI 25–29) was higher in the control group compared to the intervention group (51.4% vs. 35.1%, respectively), but to our knowledge, this has not been determined to modify associations of air pollution with cardiovascular outcomes. It has been reported that adverse cardiovascular effects of air pollution may be greater in obese individuals (BMI ≥30) (Weichenthal et al. 2014), but the prevalence of obesity was similar in the control and intervention groups.

Although levels of air pollution exposure observed in this study were relatively low, we were nonetheless able to detect significant associations with several health measures, similar to our findings in two previous panel studies in other locations with relatively low exposures (Stieb et al. 2017, 2018). More broadly, there is also extensive literature in Canada documenting adverse effects of air pollution at relatively low levels of air pollution compared to elsewhere in the world (Stieb and Liu 2013). The AQHI is intended to strike a balance between encouraging outdoor activity when air pollution levels are lower, and recommending measures to reduce exposure at higher air pollution concentrations. Since there appears to be no threshold in the concentration-response relationship between a number of air pollutants and diverse health outcomes, it is not possible to define a single value as the boundary between 'safe' and 'unsafe' levels of exposure. As 5 represents the middle of the 'moderate health risk' category, we felt it was an appropriate reference value to trigger the

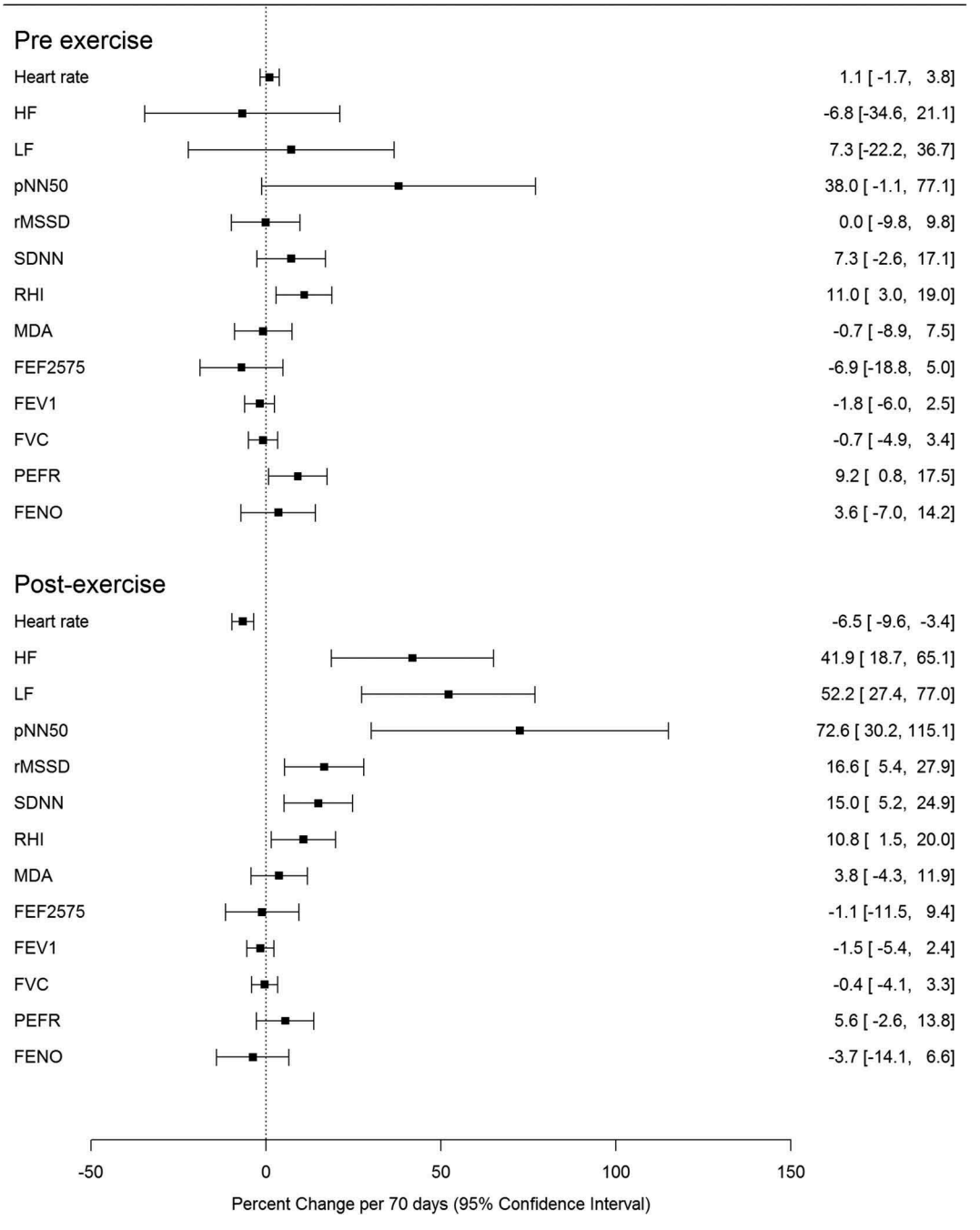

Figure 3. Percent change in cardio-respiratory measures over study duration (70 days) (HF, high frequency power; LF, low frequency power; pNN50, percentage of successive normal cardiac interbeat intervals greater than 50 msec; SDNN, standard deviation of NN (normal RR) intervals; RMSSD, root mean square of successive differences; RHI, reactive hyperemia index; MDA, malondialdehyde; FEF25-75%, forced expiratory flow at 25–75% of forced vital capacity; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; PEFR, peak expiratory flow rate; FeNO, fraction of exhaled nitric oxide).

intervention, and at the same time representative of real-world conditions in that some informed individuals would be expected to take action at this level. While lowering the trigger value to 4 for the purposes of an intervention study could be considered, the lower the value, the less representative the study would be of typical behaviour in response to the AQHI. Replication of the study

during additional summers or conducting the trial in areas with historically higher air pollution concentrations and over multiple years to reduce the probability of encountering unusually low exposures in a single year could also be considered.

Evidence regarding the effectiveness of air quality alerts/advisories has been mixed. Though they have the potential to reduce pollution-related adverse health effects, it is unclear whether behaviour is driven by the alerts themselves or by people's own perceptions of poor air quality or both (D'Antoni et al. 2017). If air quality advisories don't corroborate an individual's own perception or experience, they will likely be ignored (Bickerstaff and Walker 2001) and in fact, it has been reported that individuals' perception of air quality may not correlate very strongly with air quality data (Johnson 2012). Some studies have shown that air quality advisories have little impact on changing public behavior or the incidence of adverse health effects. In a study by Semenza et al., only about a third of study participants were aware air quality advisories were available, and of those, the small percentage of people who voluntarily changed their behaviour did so more from their perception of low air quality rather than from the advisory (Semenza et al. 2008). Even those responsible for the care of at risk individuals displayed only minor modifications in their behaviour (Semenza et al. 2008). Smallbone found that participants without pollution-related health conditions were unlikely to be aware of the air quality index or advisories and that even among those with respiratory disease, approximately a third stated they would not or could not alter their behaviours (Smallbone 2015). Analyzing direct delivery of air quality data, Smallbone found impediments to behavior change 'result from a lack of awareness or understanding of the relationship between air pollution and health rather than any operational problems' (Smallbone 2009). However, those receiving air quality data directly were more aware of the relationship between health and the air quality and more likely to modify their behavior (Smallbone 2009). Radisic et al. also found that lack of knowledge was an important barrier, as well as lack of time and dependence on one's own perceptions and media reports (Radisic et al. 2016). Though only about a third of participants in an asthma study said they would change their behaviour when aware of an air quality advisory, Wen et al. suggested this percentage could be increased with the addition of health-care professional advice (Wen et al. 2009).

With respect to effects on incidence of adverse health effects, one study found increased health-care use following air quality advisories (Lyons et al. 2016), while another, looking at the association between an alert service and COPD, found no change in admissions (Maheswaran et al. 2010). Chen et al. reported that air quality advisories reduced asthma-related emergency room admissions by 25%, while there was no impact on visits for cardiovascular outcomes (Chen et al. 2018). Also using a regression discontinuity design, Neidell found that smog alerts significantly reduced attendance at outdoor facilities and that in a daily time-series analysis, the effect of ozone on asthma hospitalizations was significantly larger when models included an indicator for air quality alerts, suggesting that individuals do in fact take action to reduce risk (Neidell 2009). In a Chilean study, where temporary measures to reduce pollution were enacted in addition to alerts sent on days with poor air quality, a reduction in mortality among the elderly was observed (Mullins and Bharadwaj 2015). Licskai et al. found a decrease in urgent health-care visits in a smartphone asthma application study, though the application included other advice in addition to air quality information, and the results were not statistically significant (Licskai et al. 2013). We also previously reported that following typical protective advice which accompanies air quality indices or advisories significantly reduced exposure to some pollutants at certain times of day, while also increasing exposure to other pollutants at other times (Stieb et al. 2008b).

Although not related specifically to the impact of AQIs or advisories, McCreanor et al. found that adults with asthma exercising in parks had significantly reduced exposure to multiple pollutants, and reduced magnitude adverse changes in lung function parameters compared with exercise along an urban roadside (McCreanor et al. 2007). Similarly, Weichenthal et al. found that adults cycling outdoors experienced adverse changes in some heart rate variability parameters, that were not seen in those cycling indoors (Weichenthal et al. 2011).

As in our previous panel studies (Stieb et al. 2017, 2018), we found that several cardiovascular measures improved over the duration of the study, including reduced postexercise heart rate, increased heart rate variability, and increased RHI, suggesting that participants experienced beneficial training effects. We recognize however that we cannot infer a causal association in the absence of a control group that did not exercise.

Strengths of our study include employment of well-established fieldwork procedures developed and implemented in our previous panel studies and recruitment of participants in an important sensitive sub-population. The study was sufficiently powered in that there were several significant differences between intervention and control groups consistent with our hypothesis. However, these differences could not plausibly be attributed to the intervention given that there were only two intervention days. Nonetheless, study power would have been greater if there had been a higher frequency of AQHI values of 5 or greater, which would have increased the difference in exposure to outdoor air pollution between the intervention and control groups.

Conclusions

In this study, we observed associations of air pollution with several cardiovascular measures and a urinary marker of oxidative stress (MDA). Our findings provide additional evidence supporting the utility of the AQHI as a predictor of health risk for diverse health outcomes and types of communities not accounted for in developing the AQHI. While associations with heart rate, HRV and MDA were consistently of greater magnitude in the control group, in keeping with our hypothesis that reduced outdoor activity on higher pollution days would attenuate associations in the intervention group, this was not the case for all outcome measures, and interpretation is difficult in light of the much smaller than expected number of intervention days. Replication of this design during additional summers may address this limitation if a greater frequency of intervention days occurs, or if the threshold triggering the intervention were lowered. Conducting the trial in areas with historically higher air pollution concentrations and over multiple years to reduce the probability of encountering unusually low exposures in a single year could also be considered. Replication of other study designs using population-level data will also strengthen the evidence base in this area.

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Declaration of Interest

No potential conflict of interest was reported by the authors.

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