Acute Blood Pressure Responses in Healthy Adults During Controlled Air Pollution Exposures

Bruce Urch,1,2 Frances Silverman,1,2,3 Paul Corey,1,2 Jeffrey R. Brook,1,2,4,5 Karl Z. Lukic,1 Sanjay Rajagopalan,6 and Robert D. Brook7

1Gage Occupational and Environmental Health Unit, St. Michael’s Hospital, Toronto, Ontario, Canada; 2Department of Public Health Sciences, 3Department of Medicine, and 4Department of Chemical Engineering, University of Toronto, Toronto, Ontario, Canada; 5Air Quality Research Branch, Meteorological Service of Canada, Environment Canada, Toronto, Ontario, Canada; 6Mount Sinai School of Medicine, New York, New York, USA; 7Department of Internal Medicine, University of Michigan, Ann Arbor, Michigan, USA

Exposure to air pollution has been shown to cause arterial vasoconstriction and alter autonomic balance. Because these biologic responses may influence systemic hemodynamics, we investigated the effect of air pollution on blood pressure (BP). Responses during 2-hr exposures to concentrated ambient fine particles (particulate matter < 2.5 µm in aerodynamic diameter: PM2.5) plus ozone (CAP+O3) were compared with those of particle-free air (PFA) in 23 normotensive, non-smoking healthy adults. Mean concentrations of PM2.5 were 147 ± 27 versus 2 ± 2 µg/m³, respectively, and those of O3 were 121 ± 3 versus 8 ± 5 ppb, respectively (p < 0.0001 for both). A significant increase in diastolic BP (DBP) was observed at 2 hr of CAP+O3 [median change, 6 mm Hg (9.3%); binomial 95% confidence interval (CI), 0 to 11; p = 0.013, Wilcoxon signed rank test] above the 0-hr value. This increase was significantly different (p = 0.017, unadjusted for basal BP) from the small 2-hr change during PFA (median change, 1 mm Hg; 95% CI, −2 to 4; p = 0.24). This prompted further investigation of the CAP+O3 response, which showed a strong association between the 2-hr change in BP and (mean arterial pressure) and the concentration of the organic fraction of PM2.5 (r = 0.53, p < 0.01; r = 0.56, p < 0.01, respectively) but not with total PM2.5 mass (r = 0.25, p = 0.27). These findings suggest that exposure to environmentally relevant concentrations of PM2.5 and O3 rapidly increases DBP. The magnitude of BP change is associated with the PM2.5 carbon content. Exposure to vehicular traffic may provide a common link between our observations and previous studies in which traffic exposure was identified as a potential risk factor for cardiovascular disease. Key words: air pollution, blood pressure, hypertension, ozone, particulate matter, PM2.5, Environmental Health Perspectives 113:1052–1055 (2005). doi:10.1289/ehp.7785 available via http://dx.doi.org/ [Online 19 May 2005]

Address correspondence to B. Urch, Gage Occupational and Environmental Health Unit, 223 College St., Toronto, Ontario, Canada M5T 1R4. Telephone: (416) 978-5886. Fax: (416) 978-2608. E-mail: bruce.urch@utoronto.ca

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BP and HR measures. BP and HR are highly variable, and even minor alterations in temperature and physical activity can substantially alter readings (Parati et al. 2003). In order to minimize effects not mediated by air pollution, BP and HR were measured throughout the actual exposure period while subjects were resting quietly and at constant temperature (~23°C). Participants were provided with an automated oscillometric BP monitor (Oscar-1; SunTech Medical Instruments, Inc., Raleigh, NC, USA) immediately before entering the exposure enclosure, with the arm-cuff secured on their left upper arm. In addition, a PC-based real-time 12-lead electrocardiogram (ECG; PC-ECG 1200; Norav Medical Ltd., Kiryat Bialik, Israel) was connected. After sitting inside the exposure enclosure, the door was sealed and concentrator pumps turned on. When resting quietly, subjects were asked to place their left forearm on their left leg and then turn on the BP monitor with their right hand (0 hr baseline exposure measure). BP measures were repeated at 30-min intervals during exposure, with the final BP measurement made at 2 hr, immediately before the end of the exposure. HR was determined at 30-min intervals by the 12-lead ECG. The technician recording the BP and HR was blinded to the exposure treatments (CAP+O3 vs. PFA).

Statistical methods. Intraexposure comparisons of BP (SBP, DBP, mean) and HR outcome variables were made using a 0-hr value as the baseline reference to assess the change over the course of the 2-hr exposure. The respective exposure values at 2 hr were calculated as a linear change (Δ = 2 hr – 0 hr) for each participant. The nonparametric Wilcoxon signed rank test was used to compare the difference in the 0- to 2-hr change between the two exposure treatments and was confirmed using percentage change, as well as the slopes of the individual straight lines fitted over the five 30-min measurements. Further support was provided by Student’s t-test of the linear and percentage changes and by a repeated-measures analysis of the linear trend over time. Linear regression analysis was also used to estimate the slope of the relationship between the 2-hr change in BP (SBP, DBP, mean) and the concentration of individual PM constituents (organic and elemental carbon, inorganic ions, and trace elements) as well as the total mass concentration. PM constituent measurements have previously been described (Urch et al. 2004). An unpaired t-test was used for interexposure comparisons of the mean PM2.5, O3, and environmental values. The Wilcoxon signed rank test was used for inter-exposure comparison of the number of symptoms reported during exposure. All analyses were performed using SAS for Windows (release 8.02; SAS Institute Inc., Cary, NC, USA).

Results

The participants ranged in age from 19 to 50 years, with a mean (± SD) age of 32 ± 10 years, and included 13 males and 10 females (Table 1).

Table 1. Participant characteristics (n = 23; 13 male and 10 female).

| Characteristic | Mean ± SD |
|----------------|-----------|
| Age (years)    | 32 ± 10   |
| Height (cm)    | 173 ± 8   |
| Weight (kg)    | 72 ± 11   |
| SBP (mm Hg)    | 117 ± 10  |
| DBP (mm Hg)    | 77 ± 9    |
| HR (beats/min) | 70 ± 11   |

The mean exposure concentration (± SD) of the PM2.5 total mass was 147 ± 27 with a range of 102–214 µg/m³ (Table 2). The mean O3 concentration was less variable, with a mean (± SD) of 121 ± 3 and range of 115–128 ppb. As expected, the PM2.5 total mass and O3 concentrations were significantly different between exposure treatments (p < 0.0001 for both). The exposure concentrations of the other copollutants measured, which included nitrogen oxides, sulfur dioxide, and carbon monoxide, were low and less than measured ambient levels. There were no significant treatment differences for any of these copollutants, for temperature, or for relative humidity.

Symptoms reported by subjects during exposures were few, if any (mean number of symptoms reported ± SD was 1.0 ± 1.9 for CAP+O3 vs. 0.5 ± 1.2 for PFA; p = 0.039). For example, only 4 of 23 subjects (17%) reported smelling an odor during CAP+O3 exposure, compared with 1 during PFA, which is not surprising because the O3 generation began at the exposure start (time 0 hr) and progressively increased over the first 10 min, then remained stable at 120 ppb over the duration of the exposure. Subjects were not able to identify the pollutant (CAP+O3) exposure with any precision, because only 52% (12 of 23) responded “yes” immediately afterward to the question, “Do you think you were exposed to a pollutant?” After PFA exposure, 30% (7 of 23) responded “yes” they were exposed to a pollutant, including 4 of the 12 who said “yes” after CAP+O3.

Table 2. Particle, O3, and environmental exposure measures (mean ± SD; n = 23).

| Measure            | CAP+O3 | PFA  |
|--------------------|--------|------|
| PM2.5 (µg/m³)      | 147 ± 27* | 2 ± 2* |
| O3 (ppb)           | 121 ± 3*  | 8 ± 5  |
| NO2 (ppb)          | 55 ± 18   | 51 ± 23 |
| SO2 (ppb)          | 3 ± 2     | 4 ± 5  |
| CO (ppm)           | 0.6 ± 0.2  | 0.5 ± 0.2 |
| Temperature (°C)   | 23.0 ± 1.3 | 23.3 ± 1.3 |
| Relative humidity (%) | 49 ± 8 | 48 ± 9 |

NO2, nitrogen oxides.
*p < 0.0001, unpaired t-test for CAP+O3 versus PFA.

Table 3. Median (binomial 95% CI) DBP and SBP (mm Hg) over 2-hr exposures (n = 23).

| Exposure time | 0 hr | 0.5 hr | 1 hr | 1.5 hr | 2 hr | 2 hr Δ² |
|---------------|------|--------|------|--------|------|--------|
| CAP+O3 DBP    | 74 (69–78) | 75 (71–76) | 75 (70–76) | 78 (71–82) | 78 (71–82) | 0 (0–11)* |
| CAP+O3 SBP    | 118 (112–127) | 119 (111–126) | 118 (112–124) | 121 (113–124) | 121 (113–124) | −1 (−5–4) |
| PFA DBP       | 74 (69–78) | 73 (71–76) | 72 (68–80) | 76 (70–81) | 73 (70–76) | 1 (2–4) |
| PFA SBP       | 117 (113–124) | 115 (107–121) | 118 (110–123) | 120 (114–131) | 121 (112–123) | 0 (2–5) |

*p = 0.017 for CAP+O3 DBP 2 hr Δ versus PFA DBP 2 hr Δ, Wilcoxon signed rank test.

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Discussion

For individuals exposed to CAP+O₃ for 2 hr, we observed a significant increase of 6 mm Hg (9.3%) in DBP. A particularly interesting finding is the strong association between the size of the 2-hr DBP change (and MAP) and the carbon content of the fine particles. This result provides additional validity and biologic plausibility to the observed blood pressure increase. The levels of both pollutants in this study, although high, were at environmentally relevant concentrations [U.S. Environmental Protection Agency (EPA) 2002]. These findings shed further light upon the biologic mechanisms that link air pollution exposure to enhanced cardiovascular risk.

The significant correlation between the organic carbon fraction of the PM₂·₅ and BP change suggests that PM composition is an important factor in cardiovascular health effects and that the carbonaceous fraction in particular warrants further study. It is also possible that carbon is only a measurement proxy for the pollutant actually responsible. A source apportionment study of local ambient PM₂·₅, over the same time period, has shown that 40–50% of the organic carbon was motor vehicle related—through either combustion or road dust (Lee et al. 2003). Exposure to urban traffic has been identified as a potential risk factor for cardiovascular disease (Peters and Pope 2002). Of particular note, a recent study reported an association between exposure to urban traffic and the onset of myocardial infarction, as soon as 1 hr afterward (Peters et al. 2004). Although we observed no association between other pollutants (e.g., O₃) and the change in BP, further studies will be required to clarify the specific pollutant(s) responsible. In particular, studies will need to be performed using concentrated particles with and without added O₃ to determine if there are important additive or synergistic interactions.

Of substantial interest is that the size of the 2-hr change in blood pressure (and MAP) and the carbon content of the fine particles. Although this may help lend biologic plausibility to this observation, the responsible pollutant(s) and the change in BP, further studies will be required to clarify the specific pollutant(s) responsible. In particular, studies will need to be performed using concentrated particles with and without added O₃ to determine if there are important additive or synergistic interactions.

O heart and MAP 2002; 105:1534–1536. The risks for myocardial infarctions (Peters et al. 2001, 2004), as well as strokes (Hong et al. 2002; Tsai et al. 2003), increase in response to acute elevations in air pollution. These observations suggest that a factor common to the etiology of both adverse outcomes, such as an elevated BP, may play a significant pathophysiologic role. It is well established that relatively small sustained increases in DBP even within the normotensive range, as observed in this study (median change of 6 mm Hg), increase the long-term risk for coronary events and strokes by approximately 30% and 40%, respectively (Lewington et al. 2002; Vasan et al. 2001). However, the cardiovascular risk imposed by the brief vasoressor response observed in this study is less certain. Nevertheless, two hypothetical scenarios may be envisioned. Exposure to high ambient concentrations of air pollutants may initiate a rapid hypertensive response, thus promoting acute cardiovascular events in susceptible individuals. In conjunction, if this vasoressor response continues unabated, gradients in personal exposure to air pollution could contribute to long-term differences in interindividual BP levels. Continued exposure to air pollution could thereby increase the risk for developing chronically elevated BP and possibly overt hypertension.

Conclusions

We observed a rapid and a statistically significant increase of diastolic BP among individuals exposed to ambient fine particles and O₃. Lending biologic plausibility to this observation is that the size of the 2-hr change in blood pressure is associated with the carbon content of the fine particles. Although this may help explain the mechanisms whereby air pollution increases cardiovascular risk, the clinical significance of this finding, the responsible pollutants, biologic mechanisms, and the duration of the response require further investigation. Exposure to vehicular traffic in urban centers may provide a common link between our findings and previous studies in which this exposure source was identified as a potential risk factor for cardiovascular disease.

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