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Clinical research

Effects of air pollution on blood pressure and heart rate variability: a panel study of vehicular traffic controllers in the city of São Paulo, Brazil

Ubiratan de Paula Santos[1]*, Alfésio Luís Ferreira Braga[2,3], Dante Marcelo Artigas Giorgi[4], Luiz Alberto Amador Pereira[2], César Jose Grupi[5], Chin An Lin[2], Marcos Antonio Bussacos[6], Dirce Maria Trevisan Zanetta[7], Paulo Hilário do Nascimento Saldiva[2], and Mario Terra Filho[1]

[1] *Divisão de Pneumologia, Instituto do Coração (InCor), Av. Dr Enéas de Carvalho Aguiar, 44, CEP 05403-000, São Paulo, SP, Brazil*
[2] *Laboratory of Experimental Air Pollution, Department of Pathology, University of São Paulo Medical School, São Paulo, Brazil*
[3] *Environmental Pediatrics Program, University of Santo Amaro Medical School, São Paulo, Brazil*
[4] *Hypertension Unit, Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil*
[5] *Eletrocardiology Unit, Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil*
[6] *Division of Occupational Health, FUNDACENTRO, São Paulo, Brazil*
[7] *Department of Epidemiology and Public Health, São José do Rio Preto Medical School, São José do Rio Preto, Brazil*

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Aims Evaluating the effects of air pollution on São Paulo city's vehicular traffic controllers by means of risk indicators for cardiovascular diseases.
Methods and results Twenty-four hour blood pressure recordings and an electrocardiogram were obtained in 48 healthy, non-smoking vehicular traffic controllers, aged 31–55 years, during three periods: winter 2000, summer 2001, and winter 2001. Effects of air pollutants on the outcomes were estimated using linear regression based on generalized estimated equations, controlling for age, body mass index, humidity, and temperature. An interquartile range (IQR) increase in CO (1.1 p.p.m.) was associated with increases of 2.6 mmHg (95% CI 1.0, 4.2), 1.8 mmHg (95% CI 0.8, 2.8), and 2.4 mmHg (95% CI 1.1, 3.6) in systolic, diastolic, and mean 24 h ambulatory blood pressures. SO_2 also had relevant effects on blood pressure. On heart rate variability, an IQR increase of SO_2 (9.6 $\mu g/m^3$) was negatively associated with the standard deviation (SD) of normal RR intervals (SDNN) −7.93 ms (95% CI −15.3, −0.6).
Conclusion This study supplies biological plausibility for observational studies on air pollution-related cardiovascular morbidity and mortality.

* Corresponding author. Tel/fax: +55 11 3082 7040.
E-mail address: pneubiratan@incor.usp.br

Introduction

Observational[1–5] and experimental[6,7] studies have repeatedly reported associations between air pollution and morbidity and mortality due to cardiorespiratory diseases. It has been estimated that ~800 000 people worldwide die every year due to the adverse health effects of air pollution.[8]

In a cohort study, Pope *et al.*[9] followed up 500 000 adults and found a 9% increase in cardiopulmonary mortality associated with a 10 $\mu g/m^3$ increase in long-term particulate matter smaller than 2.5 μm ($PM_{2.5}$) exposure and smaller mortality increases attributed to sulfate and sulfur dioxide (SO_2) increases. In a complementary analysis of the same cohort, Pope *et al.*[10] showed that PM exposures were most strongly associated with mortality due to ischaemic heart disease, dysrhythmias, heart failure, and cardiac arrest. Hoek *et al.*,[11] in a prospective cohort study, found that people living by major roads have an increased cardiopulmonary mortality risk. This study reinforces the role of automotive fleets on the air pollutant emissions issue. Daniels *et al.*,[12] in a time-series study of short-term exposure, found a linear relationship between particle exposure and mortality, reinforcing the concept of a dose–response relationship already suggested by Dockery *et al.*[1] in their prospective cohort long-term exposure study of six cities. These results indicate the lack of a security threshold for particle exposure. However, this concept is not yet established for other pollutants.

The magnitude of this problem in terms of public health has demanded new studies that can clarify the pathophysiological mechanisms responsible for the adverse respiratory and cardiovascular effects attributed to air pollution. In terms of cardiovascular diseases, studies have focussed on potential effects of pollutants on inflammatory blood markers and on autonomic nervous system control.[13,14] Heart rate, its variability, and blood pressure have been shown to be susceptible to the effects of air pollution.[6,15–30]

Despite the clear evidence of the association between air pollution and cardiovascular diseases, most of the studies have used well-known susceptible groups like the elderly,[16–18,21–23,25,27–29] people with chronic obstructive pulmonary disease,[21,22] and/or cardiac diseases,[16,26,28] or unusual exposure levels.[19,30] Also, published blood pressure studies have used conventional sphygmomanometry measurements,[21–23,26] which are less accurate than ambulatory blood pressure monitoring (ABPM) and a potential source of measurement errors.[31] Based on the hypothesis that air pollution exposure may induce cardiovascular changes mediated by the autonomic system not only in susceptible populations, the present study has assessed the effects of air pollution on heart rate variability and blood pressure in healthy vehicular traffic controllers in the city of São Paulo, using 24 h electrocardiographic and blood pressure monitoring.

Methods

Site and study population

São Paulo city is 800 m above sea level, with 10 500 000 inhabitants, and an area of 1500 km^2. Its 5 400 000 vehicles are the main source of air pollution. We conducted a panel study with 48 male vehicular traffic controllers selected from 250 employees of the São Paulo Traffic Engineering Company (CET), who worked at three high-traffic avenues, two of them riverside avenues, which are part of the loop around the city's central area (*Figure 1*). The inclusion criteria were the following: >3 years working for CET and ≥1 year working at the riverside

Figure 1 Brazil, state of São Paulo, metropolitan region, and São Paulo city. The white lines represent the avenues where the traffic controllers worked.

avenues; age <60; being a non-smoker or having quit smoking for >1 year; having no other occupational activity with risk of gas or particle inhalation; absence of signs and symptoms of chronic obstructive pulmonary disease, asthma, heart failure, or cardiac arrhythmia; and no regular use of corticosteroids, anti-arrhythmic medication, beta-blockers, anti-inflammatory drugs, or aspirin. All study participants had the same educational level (11 years of completed high school) and a monthly salary from U$ 400.00 to U$ 500.00.

The study participants were evaluated in three periods: period 1, 31 July 2000 to 22 August 2000 (winter); period 2, 22 January 2001 to 21 February 2001 (summer); and period 3, 31 July 2001 to 23 August 2001 (winter).

Analysed parameters

A self-response questionnaire was used to obtain information on morbidity and medicine intake. All subsets underwent clinical examination, chest X-ray, and carbon monoxide (CO) measurement in exhaled air (MicroCO Meter, Micro Medical Ltd, Rochester, Kent, UK).

The following parameters were measured in each period:

(i) Clinical parameters: changes in work routine, presence of respiratory symptoms, medicine intake.
(ii) Twenty-four hour electrocardiographic Holter monitoring (GE, Marquette, MARS, version 4.0a) according to standards defined elsewere.[32] Records were analysed for quality and to obtain heart rate (HR) mean and variability (HRV) indicators: standard deviation (SD) of normal RR intervals (SDNN) and standard deviation of averages of normal RR intervals (SDANN) every 5 min, the square root of the mean of squared differences between NN adjacent intervals (rMSSD), low frequency (LF), high frequency (HF), and the LF/HF ratio.
(iii) Twenty-four hour ABPM, using 24 h SpaceLabs 90207 monitors (Spacelabs Medical, Inc., USA). Blood pressure was measured every 10 min when the volunteers were awake (6:00 a.m. to 11:00 p.m.) and every 20 min during sleep (11:00 p.m. to 6:00 a.m.). Records with less than 20% errors, at least 70 valid measures, and length of more than 21 h were accepted. We used systolic, diastolic, and mean blood pressure measurements for the analyses.

Holter and ABPM were measured simultaneously. Each participant filled out a daily activity card to record any unusual occurrence during the day. This information was used to control any unexpected record on both Holter and ABPM. Usually, the physical activity required to perform the role of a traffic controller is of low intensity.

Air pollution and weather

Daily records of CO, particulate matter with an aerodynamic profile $\leq$10 μm (PM_{10}), nitrogen dioxide (NO_2), and SO_2 were obtained from São Paulo State Sanitary Agency (CETESB). Fourteen monitoring stations were spread around the city, although not all of them measured all pollutants. The measurement adopted for CO (p.p.m.) was the highest 8 h moving average at eight stations. NO_2 ($\mu g/m^3$) concentrations were computed as the highest hourly average at seven stations. The 24 h averages were adopted for PM_{10} ($\mu g/m^3$), measured in 14 stations, as well as SO_2 ($\mu g/m^3$), measured in six stations. Air pollutant measurements have been analysed around the city in the last 20 years, and air pollution levels recorded in each station are highly correlated with the others. Therefore, the average of all stations that measured each pollutant was adopted as a citywide exposure status. These computations follow standards adopted by CETESB in agreement with international standards.[33] All pollutants were measured from 1:00 a.m. to 12:00 p.m. Mean daily temperature and mean relative humidity values were obtained from the Institute of Astronomy and Geophysics (IAG) of the University of São Paulo.

Statistical analysis

Descriptive analyses were made for the variables included in the study. We assessed the linear correlation between the pollutants using simple correlation coefficients (Pearson product–moment correlation coefficients). The repeated measures analysis of variance (ANOVA) was used to test for dependent variable differences in the three study periods. In the analysis, we used an exchangeable correlation structure, which assumes equal variances and covariances between measurements within a subject, to take into account the correlation of repeated measurements. Scheffe's *post hoc* test was used and the two-tailed test of significance was adopted. The effect of air pollutants on blood pressure and HRV was estimated using linear regression models based on generalized estimating equations (GEE) considering fixed effects for repeated measurements in the S-Plus 2000 Professional Release 3 (MathSoft Inc., Seattle, WA, USA), adjusting for age, body mass index (BMI), temperature, and humidity using an exchangeable correlation structure as a working matrix, which assumes equal correlation for measurements in each subject.

The regression model was:

$$E[Y_{ij}] = \alpha + \beta_1\ \text{pollutant}_{ij} + \beta_2\ \text{age}_{ij} + \beta_3\ \text{bmi}_{ij} + \beta_4\ \text{temperature}_{ij} + \beta_5\ \text{humidity}_{ij} \quad (1)$$

where Y is the dependent variable, i is the subject, and j is the period.

The lag structure between air pollutants and adverse effects was assessed using lags 0 to 5 days and moving averages of 2, 3, and 5 days. For instance, a 2 day moving average is the mean of the levels in the concurrent and previous day assigned for the concurrent day. Single-pollutant models were used for the analyses. If more than one pollutant had a significant effect on an outcome, two-pollutant models were adopted. Effects were reported as increases in the outcomes [with the respective 95% confidence interval (CI)] for an interquartile range (IQR) increase in each pollutant.

The study was approved by the Heart Institute Research Ethics Committee of the University of São Paulo Medical School.

Results

Table 1 presents descriptive characteristics of the study population. Mean age was 39 years and the average work time at CET was 10 years. In general, the group was overweight and had high cholesterol levels.

For Holter analyses, 48 individuals had three follow-up examinations (144 records) while only 46 completed blood pressure follow-up (138 records).

Table 2 presents descriptive analysis and ANOVA for the outcomes in the study period. Holter and blood pressure variables had variable-specific seasonal patterns.

Table 3 presents descriptive analysis and ANOVA of pollutants and weather variables. Primary pollutants had higher levels in winter but all of them had huge variations (*Figure 2*). For PM_{10}, SO_2, and, to a lesser extent NO_2, differences between winter and summer levels were statistically significant.

Primary pollutants had significantly high Pearson correlation coefficients, ranging from 0.65 for CO–SO_2 to 0.9 for SO_2–PM_{10}.

The effects of air pollutants on blood pressure are shown in *Figure 3*. CO and SO_2 had positive and statistically significant effects on blood pressure. CO had significant acute (lag 0) and subacute (lag 4) effects, whereas SO_2 effects were mainly lagged. When a two-pollutant model was used to test the robustness of the associations, only the CO effect remained statistically significant.

Only SO_2 presented a statistically significant association with HRV (*Table 4*). This effect was negative in almost all lag structures and increased according to the length of lag structures for SDANN and was less consistent for SDNN. This pattern of lag structure-effect is similar to that observed for blood pressure. SO_2 was also associated with decreased r-MSSD and mean HR increase, but the most relevant effect was observed only at lag zero. An IQR increase was associated with r-MSSD −2.28 ms (95% CI −4.67, 0.11) decrease, and with HR 1.7 b.p.m. (95% CI 0.2, 3.3) increase in 24 h mean at lag 0. No statistically significant association was observed with frequency domain HRV measurement.

PM_{10} and NO_2 had no statistically significant effects either on blood pressure or on HRV indicators.

Table 1 Characteristics of the 48 participants

General	Mean (SD)	Range
Age (years)	39.2 (6.4)	31–55
Working time (years)	9.7 (4.6)	5–27
Body mass index (kg/m^2)[a]	27.9 (4.2)	21.3–41.9
Total cholesterol (mg/dL)[a]	205.5 (41.3)	120–378
HDL-C (mg/dL)[a]	47.1 (11.7)	26–93
Tobacco, morbidity, and medication	Number (%)	
Non-smokers	26 (54.2)	
Ex-smokers[b]	22 (45.8)	

HDL-C, high density lipoprotein cholesterol
[a]Average of the three measurements for all participants along the study.
[b]Ex-smokers for at least 1 year.

Discussion

There are many hypotheses to explain the effects of air pollutants on cardiovascular diseases. Among them, changes in blood markers associated with increased cardiovascular risks,[13,34] ischaemic response in the myocardium,[35] and effects on the autonomic nervous system,[4,15–19,23] which are related to blood pressure and HRV changes. The cardiovascular system and HR are permanently under the influence of the sympathetic and parasympathetic nervous systems, with a predominance of the latter. Mental stress, physical exertion, and stimuli promoted by outdoor air pollution, tobacco smoke, and weather variables may change the balance between the two systems, decreasing parasympathetic influences and increasing the sympathetic tone. This new situation leads to increases in blood pressure and HR, decreases in HRV, and lowers the ventricular fibrillation threshold.[36] Hence, the risks of cardiac arrhythmia, sudden death, and other cardiovascular events are higher.[14,32,36–40]

The present study showed that air pollutants might affect the cardiovascular system, including blood pressure and HRV in a group of healthy, non-smoking adults, with a similar degree of education and socioeconomic status. It is a unique study because of the characteristics

Table 2 Descriptive statistics and analysis of variance of HRV and blood pressure for each period[a]

Variable	Mean (SD)			*P*-value
	Period 1	Period 2	Period 3	
HRV ($n = 48$)				
SDNN (ms)	135.2 (34)	143.8 (33.8)	140.3 (31.6)	0.1012
SDANN (ms)	119.1 (32.8)	127.7 (33.3)	125.3 (31.8)	0.0940
r-MSSD (ms)	29.9 (9)	34.2 (12.4)	31.4 (10.5)	0.0024[b]
LF (ms^2)	29.1 (8.4)	32.2 (11.5)	29.5 (8.5)	0.0049[c]
HF (m/s^2)	14.1 (4.9)	16.1 (6.8)	14.2 (5.2)	0.0030[c]
LF/HF	2.1 (0.4)	2.1 (0.5)	2.2 (0.5)	0.3347
Heart rate (b.p.m.)	77.81 (6.82)	74.54 (8.07)	77.06 (8.86)	0.0033[c]
ABPM (mm Hg) ($n = 46$)				
Systolic pressure	129.5 (11.0)	122.5 (9.5)	124.3 (9.6)	0.0001[d]
Diastolic pressure	82.4 (9.2)	77.7 (8.1)	78.5 (9.4)	0.0001[d]
24 h mean pressure	98.11 (9.5)	92 (8.6)	93.5 (8.5)	0.0001[b]

n, number of participants.
[a]Winter (31 July 2000 to 22 August 2000), summer (22 January 2001 to 21 February 2001), winter (31 July 2001 to 23 August 2001).
[b]Period 1 ≠ period 2.
[c]Period 2 ≠ (period 1 = period 3).
[d]Period 1 ≠ (period 2 = period 3).

Table 3 Descriptive statistics and analysis of variance of air pollutants, temperature, and humidity during study periods

Variable	Period[a]	Mean (SD)	IQR	25–75%	*P*	*n*
CO (p.p.m.)	Period 1	2.9 (1.1)	1.6	1.9–3.5		23
	Period 2	2.3 (0.4)	0.7	1.9–2.6		31
	Period 3	2.6 (1.0)	1.3	1.9–3.2		24
	Three periods	2.6 (0.9)	1.1	1.9–3.0	0.06	78
SO_2 ($\mu g/m^3$)	Period 1	21.6 (11.4)	15.4	11.5–26.9		23
	Period 2	12.1 (2.3)	2.8	11.0–13.8		31
	Period 3	19.2 (5.3)	7.7	15.7–23.4		24
	Three periods	17.1 (8.1)	9.6	11.3–20.9	0.00[b]	78
NO_2 ($\mu g/m^3$)	Period 1	107.4 (49.7)	82.4	62.9–145.3		23
	Period 2	87.5 (21.3)	23.2	73.6–96.8		31
	Period 3	133.9 (45.1)	47.3	107.0–154.3		24
	Three periods	107.6 (43.2)	56.4	76.7–133.1	0.00[c]	78
PM_{10} ($\mu g/m^3$)	Period 1	58.7 (24.9)	34.4	39.7–74.1		23
	Period 2	37.4 (7.5)	7.4	33.9–41.3		31
	Period 3	63.5 (18.9)	23.0	49.0–72.0		24
	Three periods	51.7 (21.1)	33.7	34.6–68.4	0.00[b]	78
Temperature (°C)	Period 1	15.4 (3.3)	4.7	13.3–18.0		23
	Period 2	23.7 (0.9)	1.5	22.9–24.6		31
	Period 3	17.0 (1.3)	1.0	16.4–17.4		24
	Three periods	19.2 (4.2)	7.3	16.0–23.3	0.00[b]	78
Humidity (%)	Period 1	78.2 (11.5)	14.0	72.4–86.4		23
	Period 2	80.6 (4.5)	7.7	77.3–85.0		31
	Period 3	76.6 (7.3)	9.4	72.0–81.4		24
	Three periods	78.6 (8.0)	10.4	74.3–84.7	0.12	78

P, ANOVA *p*-value; *n*, number of days.
[a]Winter (31 July 2000 to 22 August 2000), summer (22 January 2001 to 21 February 2001), winter (31 July 2001 to 23 August 2001).
[b]Period 2 $\neq$ (period 1 = period 3).
[c]Period 2 $\neq$ period 3.

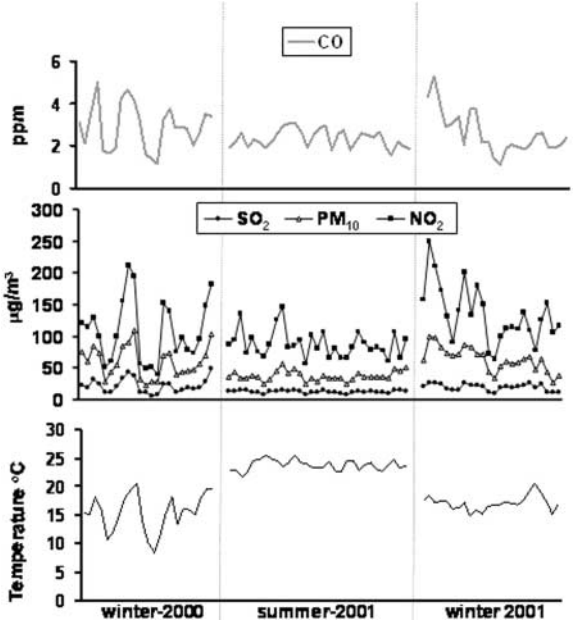

Figure 2 Daily air pollutants and temperature during study periods. Winter (31 July 2000 to 22 August 2000), summer (22 January 2001 to 21 February 2001), winter (31 July 2001 to 23 August 2001).

of the study population and the methods chosen to measure blood pressure (24 h ABPM).

The analysis methods took into account statistical tools used for panel studies, and the controlling variables included in the models were those that can potentially affect the dependent variables. It is possible that other unusual confounders or effect modifiers of the association between air pollution and cardiovascular risk may have been missed. Among them are stress and noise control.

Most of the daily and inter-period variability in air pollutant levels can be explained by changes in the number of vehicles, small dispersion of pollutants, and the number and height of thermal inversions. In addition, the winter in São Paulo is dryer than the summer.

The exploratory analysis using ANOVA showed increases in both air pollution and the outcomes indicating a potential relationship between them. However, this approach is not enough to identify potential causal relationships.

Blood pressure

The magnitude of the effects observed in this study, in particular blood pressure changes, is greater than that observed in other studies. Moreover, no statistically significant effects were observed for PM_{10}.[21–24] However, other authors have

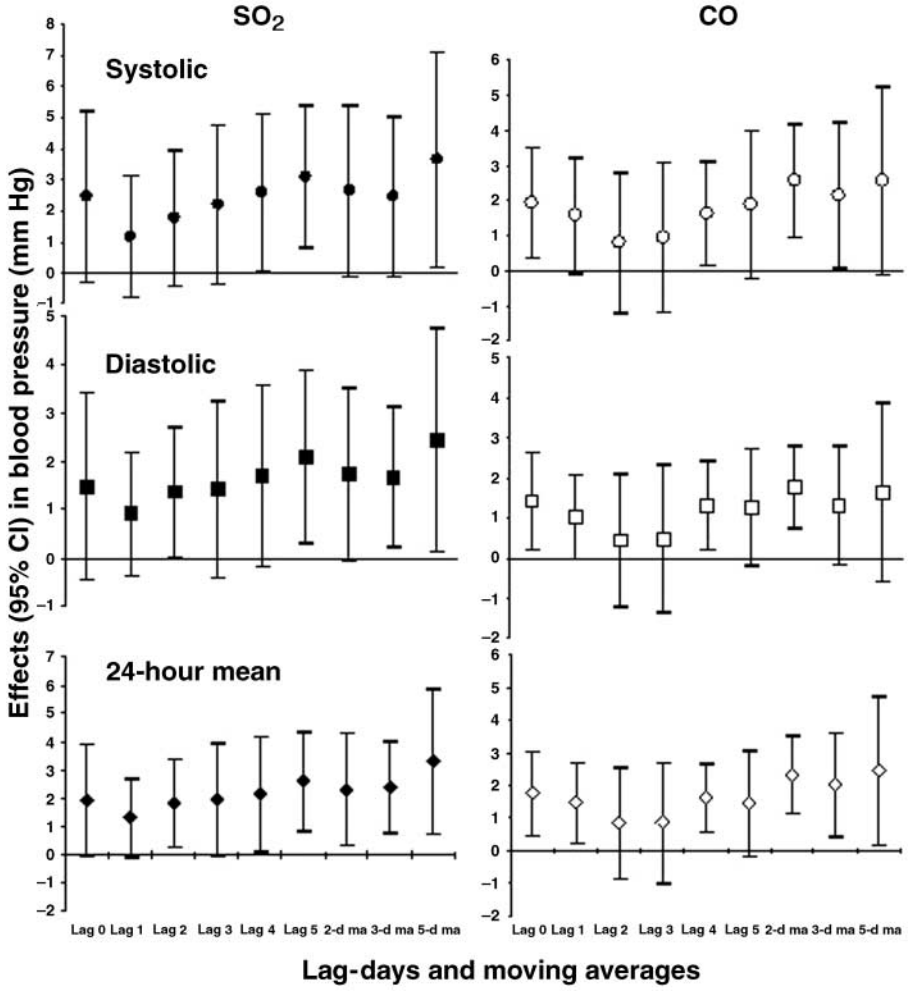

Figure 3 Effects and 95% CI on 24 h systolic, diastolic, and mean blood pressures due to IQR increases in SO_2 and CO levels. SO_2IQR = 9.6 μg/m^3; CO IQR = 1.1 p.p.m.; ma = moving average.

Table 4 Effects (ms) and 95% CI on HRV using different lag structures for an IQR in SO_2 (9.6 μg/m^3)

SO_2	SDNN	SDANN	r-MSSD
Lag 0	−4.91 (−10.90; 1.08)	−3.93 (−9.67; 1.81)	−2.28 (−4.67; 0.11)
Lag 1	−0.80 (−7.72; 6.13)	−2.19 (−10.04; 5.66)	0.82 (−1.10; 2.74)
Lag 2	1.33 (−5.02; 7.67)	−1.18 (−7.34; 4.99)	1.11 (−0.79; 3.01)
Lag 3	−1.96 (−6.89; 2.96)	−2.83 (−7.89; 2.23)	−0.14 (−1.48; 1.20)
Lag 4	−7.93 (−15.31; −0.55)*	−7.30 (−14.82; 0.21)	−0.77 (−2.54; 1.00)
Lag 5	−2.60 (−8.42; 3.21)	−0.81 (−7.37; 5.76)	−1.04 (−2.74; 0.65)
2 day ma	−5.41 (−12.63; 1.82)	−5.58 (−13.22; 2.06)	−1.21 (−3.54; 1.12)
3 day ma	−3.56 (−12.74; 5.62)	−5.77 (−15.70; 4.17)	0.003 (−0.236; 0.242)
5 day ma	−7.38 (−16.47; 1.71)	−10.22 (−20.27; −0.18)*	0.96 (−1.94; 3.86)

ma, moving average. *$P < 0.05$.

reported lag structures between exposure and effects that are similar to those presented in this study.[23]

Some studies have shown different results. Ibald-Mulli *et al.*,[26] in a study of air pollution cardiovascular effects in cardiac patients, found negative associations between particles and blood pressure, mainly among those with previous myocardial infarction. The authors suggest that medication intake and disease status, both affecting the autonomic control of the heart, may explain the differences with previous studies.[22,23]

The use of 24 h ABPM instead of instantaneous measurements, a procedure with more measurement errors used as standard until now,[31] and the characteristics of the participants in our study (younger, healthier, and more homogeneous than those of other studies) may have contributed to the final results.

Two different pathways have been related to vascular constriction. One of them is based on the induction of reflex increase in the activity of the sympathetic nervous system;[35,41] the other is the acute increase in the release of vascular endothelin.[23,24] It is possible that air pollutants promote vascular constriction through both mechanisms.

Brachial artery vasoconstriction after exposure to air pollutants was observed in a study by Brook *et al.*[24] with 25 healthy and non-smoker adults. The authors suggest that a similar event may occur in the coronary arteries, as both groups of arteries have highly correlated reactivity.

In our study, despite using a different study design, we observed that effects of air pollution on blood pressure were remarkable and that they may occur in different time lags. CO had both acute and lagged effects, whereas SO_2 effects were mainly lagged. Although the blood pressure variations observed in this study may have no acute clinical implications in healthy individuals, minor blood pressure variations may trigger arrhythmia, myocardial infarction, and stroke in those with cardiovascular co-morbidities.[36,38,42,43]

Heart rate variability

HRV reflects the autonomic nervous system's modulation of heart rate and has been used as a risk marker for arrhythmia and sudden death.[32,36,37,39] HRV allows adequate reactions of the heart to environmental changes. HRV decrease reflects an imbalance of the autonomic system and may affect the capacity to adapt to several stimuli that occur daily.

Pollution impacted on HRV indicators during the winter. SO_2 effects on SDNN and SDANN were mainly negative. Changes were observed for SO_2 concentration on the concurrent day (lag 0) and the prior 4 and 5 days (*Table 4*). Its behaviour is similar to that of blood pressure and suggests both acute and subacute effects. This pattern of effect is somewhat in agreement with studies that found increased risks for both myocardial infarction[42] and discharges in patients with implanted cardioverter defibrillators associated with increases in particulate and gaseous pollutants.[4]

Although frequency domain variables have shown statistically significant variations between winters and summer (*Table 2*), we did not find statistically significant effects of air pollution on those variables. However, the r-MSSD, an estimator of short-term variation in heart rate modulated by the parasympathetic nervous system and correlated with HF, was acutely affected by SO_2, as reported by other authors.[16–19] The lack of effects of air pollutants on frequency domain parameters may be partially explained by the difficult interpretation of 24 h Holter records.[32]

The acute effect on r-MSSD and the lagged effects on SDNN and SDANN suggest that the imbalance of the autonomic system may be driven by a decrease of parasympathetic activity followed by sympathetic stimulation increase.

In addition, we did not find effects of PM on HRV indicators. This may be partially explained by the lack of $PM_{2.5}$ data pointed out in recent studies as the fraction of inhalable particles with the strongest toxicity.[9,13,17–20] Another explanation may be that analyses were performed over a 24 h period instead of a more acute exposure (hours after the exposure).[18,19,25]

The demographic characteristics of our study group may have contributed to the lack of effects in many outcomes, as observed by Devlin *et al.*[25] Most of the studies carried out elsewhere have analysed elderly persons or people with co-morbidities.[16–18,21–23,25]

The study by Magari *et al.*[19] is one of the few published reports evaluating healthy, non-elderly workers which has found effects of $PM_{2.5}$ on HRV. However, the ambient concentration of the pollutant was very high, almost 10 times the PM_{10} concentration in São Paulo during our study.

Pekkanen *et al.*[35] provided new evidence to support that air pollutants may induce increased myocardial susceptibility causing ischaemic heart diseases. These results suggest that myocardial ischaemia may be induced by air pollution exposure increasing the risk of cardiovascular diseases. However, artery constriction or blood pressure increases may also explain this outcome,[24] well-known risk factors for coronary diseases.[38]

Our study has some limitations that should be explained. First of all, the lack of noise and stress control on the autonomic nervous system. However, we believe there is a minimum adaptation after at least 3 years of daily work as traffic controllers, where no significant changes in vehicle traffic occur. No dramatic changes were recorded by the traffic controllers in their daily activity report cards. Thus, we may assume there were no major respiratory frequency changes that could have influenced the results. Secondly, and more relevant is the lack of individual monitoring exposure data in the work sites. However, the air pollution monitoring stations are located, almost exclusively, in high traffic ways around the city. Therefore, the mean air pollutant levels recorded by those monitoring stations are not far from the real environment faced daily by the automotive traffic controllers included in our study.

Controlling air pollution in São Paulo requires a deep change in traffic policies, the main source of pollutants. Studies carried out in São Paulo in the last 10 years have contributed to the implementation of some public policies as the restriction on vehicle circulation, implemented in the 1990s, the vehicular emission control programme, and the renovation of the public transportation system. Nowadays, being an automotive traffic controller in a city with 5.4 million cars and working in the busiest avenues is a risky job. Until these policies provide the expected results, traffic controllers must work reduced shifts in different work sites.

In summary, our results show that in adult and healthy workers directly exposed to automotive traffic-generated air pollution, increases in primary gaseous pollutants were associated with changes in blood pressure and HRV. Despite the high correlation between primary pollutants, the effects for PM_{10} and NO_2 were not found. Because fossil fuel and alcohol burning generate pollutants in São Paulo, the toxicological patterns of those

pollutants may be altered. This study supplies biological plausibility for observational studies on the cardiovascular effects of air pollution and provides additional evidence that public policies must be adopted to reduce air pollution in São Paulo. However, due to the dissimilarities among studies carried out in different places with specific air pollution characteristics and different populations, further investigations are required on a research topic that is far from concluded.

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