	I N A		6 1 2	B	Dispatch: 29.6.09	Journal: INA	CE: Krishna Sarma
	Journal Name	Manuscript No.			Author Received:	No. of pages: 7	PE: Radhika

Indoor Air 2009
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INDOOR AIR
doi:10.1111/j.1600-0668.2009.00612.x

The effects of indoor particles on blood pressure and heart rate among young adults in Taipei, Taiwan

Abstract This study aims to evaluate whether indoor particles are associated with elevated blood pressure (BP) and heart rate (HR). We recruited 40 young, healthy students from universities in Taipei. We made four home visits in which we took consecutive 48-h measurements of systolic BP, (SBP) diastolic BP (DBP), and HR in each participant. Particulate matter less than 10 μm in diameter (PM_{10}), 2.5 μm in diameter ($PM_{2.5}$), and nitrogen dioxide levels were measured at each participant's home. Participants were asked to keep their windows open during the first two visits, and keep their windows shut during the last two visits. We used linear mixed-effects models to associate BP and HR with indoor air pollutants averaged over 1- to 8-h periods prior to physiological measurements. We found indoor PM_{10} and $PM_{2.5}$ exposures at 1- to 4-h means were associated with an elevation in SBP, DBP, and HR. Effects of indoor PM_{10} and $PM_{2.5}$ on BP and HR were greatest during the visits with windows open. During windows-closed visits, participants showed no significant change in BP and HR with indoor PM_{10} exposure. We concluded that exposures to infiltrated outdoor particles are associated with short-term increases in BP and HR in young and healthy students. Closing windows can reduce indoor PM concentrations and modify the effect of PM_{10} on BP and HR in young adults.

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Key words: Indoor particles; Air pollution; Blood pressure; Heart rate; Epidemiology.

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Received for review 31 January 2009. Accepted for publication 15 June 2009.

Practical Implications

Particulate matter exposure, high blood pressure (BP) and heart rate (HR) have been reported to be associated with increased risk of cardiovascular morbidity and mortality. Exposure to indoor particles is found to be associated with Elevated BP and HR. Closing windows may reduce indoor particles concentrations and modify the effect of particles on BP and HR in young adults in heavily polluted cities.

Introduction

Exposure to ambient air pollution, especially particulate matter (PM) has been reported to be associated with increased cardiovascular mortality and morbidity (Pope and Dockery, 1999, 2006). These associations have been partially supported by the association between PM, blood pressure (BP), and heart rate (HR) changes, a possible mechanism linking ambient PM to increased risk for cardiovascular diseases (Brook et al. 2004) in several panel studies. A survey of adults in Germany found that higher levels of total suspended PM in the air was associated with higher BP in subjects (Ibald-Mulli et al., 2001). A repeated-measures study of patients with chronic obstructive pulmonary disease (COPD) in Los Angeles showed an association between PM less than 10 μm in diameter (PM_{10}) and BP (Linn et al., 1999). The effect of PM less than 2.5 μm in diameter ($PM_{2.5}$) on BP in cardiac rehabilitation patients was reported in a Boston study (Zanobetti et al., 2004). An association between

increased HR and air pollution episode was observed in Germany (Peters et al., 1999). Our study found an association between submicrometer particles and BP and HR in patients with lung function impairment in Taipei, Taiwan (Chuang et al., 2005).

Ambient PM, especially traffic-related PM, may cause adverse effects on the cardiovascular system and travel indoors. Previous studies showed that living near a major road with heavy traffic was associated with increased mortality (Hoek et al., 2002; Hoffmann et al. 2007). Indoor penetration of ambient particles is variable, and there are many indoor sources of particles, such as cooking, heating appliances, and tobacco smoke (Lanki et al., 2007; Polidori et al., 2006) that worsen indoor air quality and have cardiovascular effects in humans. However, studies of the adverse effects of indoor air quality on BP and HR among young and healthy humans are lacking. The aim of this study was to investigate association between indoor particles and BP and HR in 40 young adults by using four consecutive 48-h periods to monitor each participant's BP and HR, and indoor PM levels in each private home. For two 48 h periods, participants were requested to keep their windows closed so we could monitor the effect of environmental airflow on PM and its consequent physiological sequelae.

Materials and methods

Study participants and design

This panel study was designed to monitor changes in air pollution levels and BP simultaneously and continuously in study participants in indoor environments. The selection criteria for study participants were as follows: the participant was not taking any medication that might affect cardiac rhythm; the participant must have no cardiovascular disease or history of cardiovascular disease such as coronary artery disease, arrhythmia, hypertension, diabetes mellitus, and dyslipidemia. We recruited a panel of young, healthy nonsmokers through an on campus advertisement in Taipei. Sixty-eight students responded to our advertisement, and 40 were willing to participate in our study after we detailed our monitoring protocols to them (Response Rate = 54%).

The protocol included four home visits that entailed consecutive 48-h BP and HR monitoring at approximately 1-week intervals from April to August 2008. The sampling date and time were recorded for each participant to correlate with air pollution and weather data in different exposure periods.

During the first visit, a baseline screening questionnaire was administered in which age, sex, body mass index (BMI), medications, pulmonary and cardiac symptoms, home characteristics, environmental tobacco smoke exposures, and time-activity patterns were noted and then 48-h BP and HR monitoring was performed. On subsequent visits, participants were administered a brief questionnaire regarding cardiac and respiratory symptoms, home characteristics, environmental tobacco smoke exposures, time-activity patterns, and then underwent 48-h BP and HR monitoring. All participants were requested to stay home and keep gas stoves off during all visits, keep windows open at the first two visits, and keep windows closed at the last two visits.

The study design was reviewed and approved by the Human Subject Committee of the St. Mary's Medicine Nursing and Management College.

BP and HR monitoring and recording

We continuously recorded each participant's BP and HR every hour during visits using a portable BP monitoring system (DynaPulse model 5000A; Pulse Metric, San Diego, CA, USA). The DynaPulse system can measure a participant's arterial pulsation signals, known as the arterial wave form, through a noninvasive cuff device. The systolic blood pressure (SBP), diastolic blood pressure (DBP), and HR readings taken by DynaPulse have been validated against the traditional mercury sphygmomanometer measurements (Brinton et al., 1998). Each participant carried a DynaPulse system for 48 h to complete his/her continuous BP and HR monitoring. To avoid sleep effects on BP and HR, we used the monitor measurements when the participants were awake between 7:00 AM and 11:00 PM in our data analysis. Each participant obtained approximately 128 successful BP and HR measurements from four visits (32 measurements for each visit) for data analysis.

Indoor air pollution and meteorological data

Exposures to indoor PM were measured using a portable dust monitor (DUST-check portable dust monitor, model 1.108; Grimm Labortechnik Ltd., Ainring, Germany) which measured and recorded 1-min mass concentrations of PM_{10} and $PM_{2.5}$, as well as 1-min temperature and humidity. Collocated Rupprecht and Patashnick 1400a tapered element oscillating microbalance samplers (Thermo Electron Corporation, East Greenbush, NY, USA) were used to calibrate the mass concentrations of PM_{10} and $PM_{2.5}$ measured by DUST-check monitor before and after each visit. Indoor nitrogen dioxide (NO_2) levels were also measured continuously by chemiluminescence NO-NO_2-NOx analyzer (model S-5012; Sistemas Instalaciones Redes, Madrid, Spain).

Statistical analysis

To compare the levels of BP, HR, indoor air pollution, and meteorological data during each visit, one-way analysis of variance (ANOVA) with Scheffe mean

comparison test was used. Linear mixed-effects regression models were applied to examine the association between indoor air pollution, BP, and HR in young adults by running S-PLUS 2000 (MathSoft Inc., Cambridge, MA, USA). The outcome variables were SBP, DBP, and HR, and the exposure variables were PM_{10}, $PM_{2.5}$, and NO_2 at previous 1- to 8-h means in single-pollutant models. The moving average was computed only if 75% of the data were present. Each regression model included fixed effects for age, sex, BMI, windows status (open or closed), day of the week, and visit order, and random effects for participant-specific terms.

The models also adjusted for several smooth function terms as fit by Nature Spline. These terms included: hour of the day, temperature and humidity. The smooth term hour of the day accounts for serial autocorrelation among measurements taken on the same day above that explained by the participant-specific intercepts in the model. Autocorrelation plots of the residuals were checked to see whether this term sufficiently accounted for autocorrelation in the data. Estimates of the effects of air pollutants were scaled to interquartile range (IQR), the difference between the 25th and the 75th percentile, in levels for the appropriate hour mean of indoor air pollutants.

Results

Forty young adults were recruited for this study and 160 home visits with 5120 hourly physiological measurements were included in data analyses. The median age of the population was 22.0 years (s.d. = 1.4 year). Their BMI averaged 22.3 kg/m^2 (s.d. = 2.7 kg/m^2), and the male/female ratio was 1:1. None of the participants had cardiopulmonary disease or medication or tobacco smoke exposure.

Table 1 summarizes BP, HR, indoor air pollution, and meteorological data averaged hourly for the 40 young adults during each visit. The BP and HR were within the normal range (Chobanian et al. 2003, Limmer et al., 2005). Although indoor air pollution was comparatively low, especially with regard to PM_{10} and $PM_{2.5}$, participants had higher PM exposures (indoor PM and infiltrated outdoor PM) and higher BP and HR responses during the first two visits (windows open) as compared with the last visits (windows closed) (ANOVA, $P < 0.05$). There were no significant differences in indoor NO_2 levels and meteorological data between the four visits: the weather was pleasant with a temperature range of 24.6–26.0°C and a relative humidity range of 49.3–51.0% during visits.

Figure 1 depicts mean levels of indoor PM_{10} and $PM_{2.5}$ for individual hours during visits with and without window open. Levels of PM_{10} rose early in the morning at 04:00 AM and peaked at 08:00 AM during the visits with the windows open (visit 1 and visit 2); in contrast, levels of PM were relatively stable during the visits with window closed. On the contrary, levels of $PM_{2.5}$ during visits with window closed had a similar pattern compared with the pattern seen during the visits with window open. Taken overall, levels of particulate air pollution during visits with windows open were higher than levels during visits with

Table 1 Summary statistics for hourly blood pressure, heart rate, indoor air pollution, and meteorological data at 1-h mean of 40 young adult with 1280 measurements during each visit

	First visit (windows open)	Second visit (windows open)	Third visit (windows closed)	Fourth visit (windows closed)	ANOVA *P*-value	Scheffe test
SBP, mmHg						
Mean ± s.d.	114.8 ± 24.0	119.1 ± 25.0	109.3 ± 22.9	107.1 ± 22.4	<0.01	1st/4th; 2nd/3rd;
Range	74.6–152.3	77.4–158.1	71.0–145.0	69.6–142.1		2nd/4th
DBP, mmHg						
Mean ± s.d.	83.7 ± 22.7	86.9 ± 23.5	79.7 ± 21.6	78.1 ± 21.2	<0.01	1st/3rd; 1st/4th;
Range	49.4–121.8	51.2–126.4	47.0–116.0	46.1–113.7		2nd/3rd; 2nd/4th
HR, beats/min						
Mean ± s.d.	76.8 ± 5.9	79.7 ± 6.1	73.1 ± 5.6	71.6 ± 5.5	<0.05	1st/4th; 2nd/3rd;
Range	65.1–92.4	67.6–95.9	62.0–88.0	60.8–86.2		2nd/4th
PM_{10}, $\mu g/m^3$						
Mean ± s.d.	37.9 ± 20.7	39.0 ± 21.2	32.5 ± 17.7	33.0 ± 18.0	<0.01	1st/3rd; 1st/4th;
Range	7.2–97.9	7.4–100.7	6.2–83.9	6.3–85.3		2nd/3rd; 2nd/4th
$PM_{2.5}$, $\mu g/m^3$						
Mean ± s.d.	26.5 ± 14.5	27.3 ± 14.9	22.7 ± 12.4	23.1 ± 12.6	<0.05	1st/3rd; 2nd/3rd;
Range	5.0–68.5	5.2–70.5	4.3–58.7	4.4–59.7		2nd/4th
NO_2, ppb						
Mean ± s.d.	19.3 ± 12.7	19.9 ± 13.1	18.8 ± 12.4	19.0 ± 12.6	0.12	–
Range	1.3–86.5	1.4–88.9	1.3–84.0	1.3–85.2		
Temperature, °C						
Mean ± s.d.	25.7 ± 2.1	24.6 ± 2.0	24.8 ± 2.0	26.0 ± 2.1	0.46	–
Range	22.0–29.8	21.0–28.5	21.2–28.7	22.2–30.1		
Relative humidity, %						
Mean ± s.d.	49.9 ± 7.7	49.3 ± 7.6	50.4 ± 7.7	51.0 ± 7.8	0.32	–
Range	25.7–66.8	25.5–66.0	26.0–67.5	26.3–68.3		

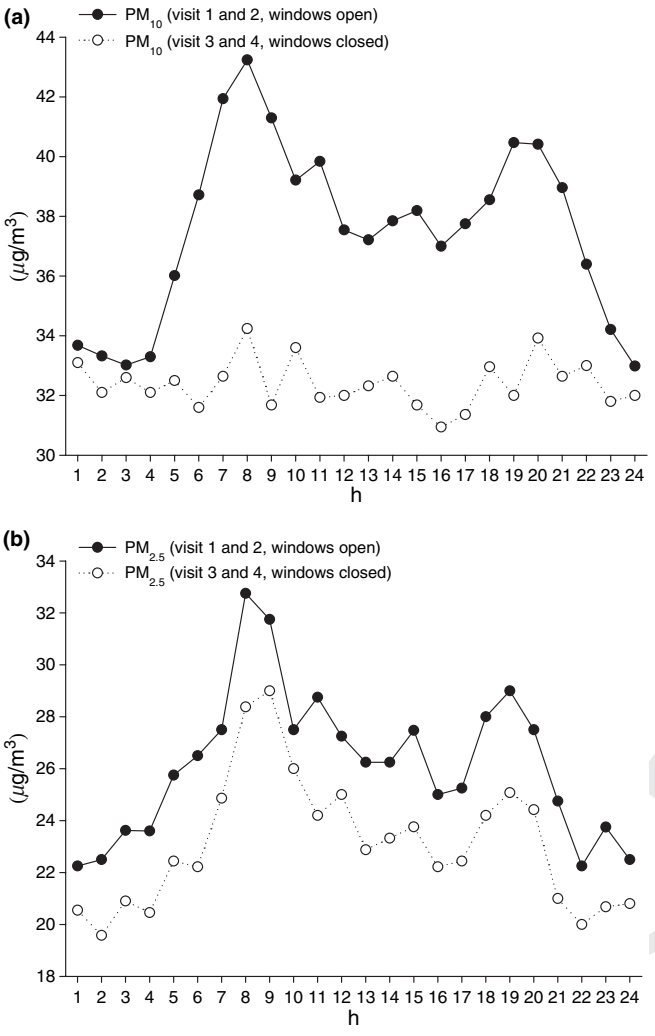

Fig. 1 Arithmetic mean of hourly indoor levels of particulate matter less than 10 μm in diameter (PM_{10}) (a) and particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) (b) for individual hour during the first two visits (windows-open) and the last two visits (windows-closed)

windows closed. Pearson correlations showed high correlations between indoor PM_{10} and $PM_{2.5}$ ($r = 0.77$, P-value < 0.01) during visits with window open and low correlations ($r = 0.39$, P-value $= 0.18$) during the visits with window closed.

The associations between indoor particulate air pollution, BP, and HR estimated by the single-pollutant mixed-effects models are shown in Table 2. With age, sex, BMI, windows status, day of the week, and visit order adjusted in our single-pollutant mixed-effects models, we found that an IQR increased the mean 1- to 4-h PM_{10} and $PM_{2.5}$ levels-predicted elevation of hourly averaged BP and HR. For BP, SBP increased by 1.85–2.64 mmHg per IQR of PM_{10} at 1- to 4-h means (30.5, 29.8, 28.5, and 27.8 $\mu g/m^3$) and 1.94–2.99 mmHg per IQR of $PM_{2.5}$ at 1- to 4-h means (21.5, 20.3, 19.6, and 18.8 $\mu g/m^3$), respectively. Likewise, DBP increased by 1.47–2.10 mmHg per IQR of

Table 2 Changes (95%CI) in blood pressure and heart rate for interquartile range changes in indoor air pollution estimated by single-pollutant mixed-effects models

	Exposure matrix	PM_{10}	$PM_{2.5}$	NO_2
SBP	1-h mean	1.85 (0.44, 3.26)	1.94 (0.46, 3.41)	1.78 (−0.22, 3.78)
	2-h mean	2.11 (0.50, 3.72)	2.29 (0.55, 4.03)	1.58 (−0.42, 3.58)
	4-h mean	2.64 (0.63, 4.65)	2.99 (0.71, 5.27)	2.11 (−0.39, 4.61)
	8-h mean	1.23 (−0.29, 2.75)	1.18 (−0.28, 2.64)	1.81 (−0.19, 3.81)
DBP	1-h mean	1.47 (0.05, 2.90)	1.54 (0.06, 3.04)	1.41 (−0.59, 3.41)
	2-h mean	1.68 (0.06, 3.31)	1.82 (0.07, 3.59)	1.26 (−0.74, 3.26)
	4-h mean	2.10 (0.08, 4.14)	2.38 (0.09, 4.69)	1.68 (−0.32, 3.68)
	8-h mean	0.98 (−0.10, 2.06)	0.94 (0.03, 1.85)	1.44 (−0.56, 3.44)
HR	1-h mean	2.10 (1.47, 2.73)	2.20 (1.54, 2.86)	1.81 (−0.69, 4.31)
	2-h mean	2.40 (1.68, 3.12)	2.60 (1.82, 3.38)	2.10 (−0.40, 4.60)
	4-h mean	3.00 (2.10, 3.90)	3.40 (2.38, 4.42)	1.66 (−0.34, 3.66)
	8-h mean	1.40 (−0.10, 2.90)	1.34 (0.94, 1.74)	3.01 (−0.99, 7.01)

PM_{10} at 1- to 4-h means and 1.54–2.38 mmHg per IQR of $PM_{2.5}$ at 1- to 4-h means, respectively. Elevations in HR of 2.10–3.00 beats/min and 2.20–3.40 beats/min were also observed per IQR changes of 1- to 4-h mean PM_{10} and $PM_{2.5}$. The cumulative effects of BP (Figure 2a) and HR (Figure 2b) were strongest at 4-h mean and waned after 5-h mean. PM at 7- to 8-h were not associated with BP and HR in our young adults. Indoor NO_2 exposure was not associated with BP and HR elevation in our study participants.

We further performed effect modification of association of indoor PM exposure with BP and HR by windows status (open vs. closed) (Table 3) and found a consistent effect modification of PM effect with windows open status. During visits with windows open, participants' SBP, DBP, and HR increased by 2.98 mmHg, 1.97 mmHg and 2.78 beats/min per IQR of $PM_{2.5}$ at 4-h mean, respectively. During windows-closed visits, participants showed no significant change in BP (interaction P-value < 0.01). Similar effect modification was suggested in models evaluation $PM_{2.5}$ effects on BP and HR, although the statistical significance of the interaction was weaker than those in models evaluation PM_{10}.

Discussion

The relationship between indoor PM and BP and HR in a panel of young adults has not yet been documented. Our study results suggest that indoor PM_{10} and $PM_{2.5}$ levels could lead to elevated HR and BP, which represents an increased risk of cardiovascular morbidity and mortality (Chobanian et al. 2003; Walldius et al., 2001). The effects of indoor PM_{10} on HR increase in our subjects were consistent with previous findings of PM_{10} on HR increase in cardiopulmonary patients (Peters et al., 1999). The magnitude of the effect of indoor PM_{10} on SBP and DBP (0.09/0.08 mmHg increase in SBP/DBP per 1 $\mu g/m^3$ indoor PM_{10} at 4-h mean) in our study was less than that of ambient PM_{10} on subjects with severe

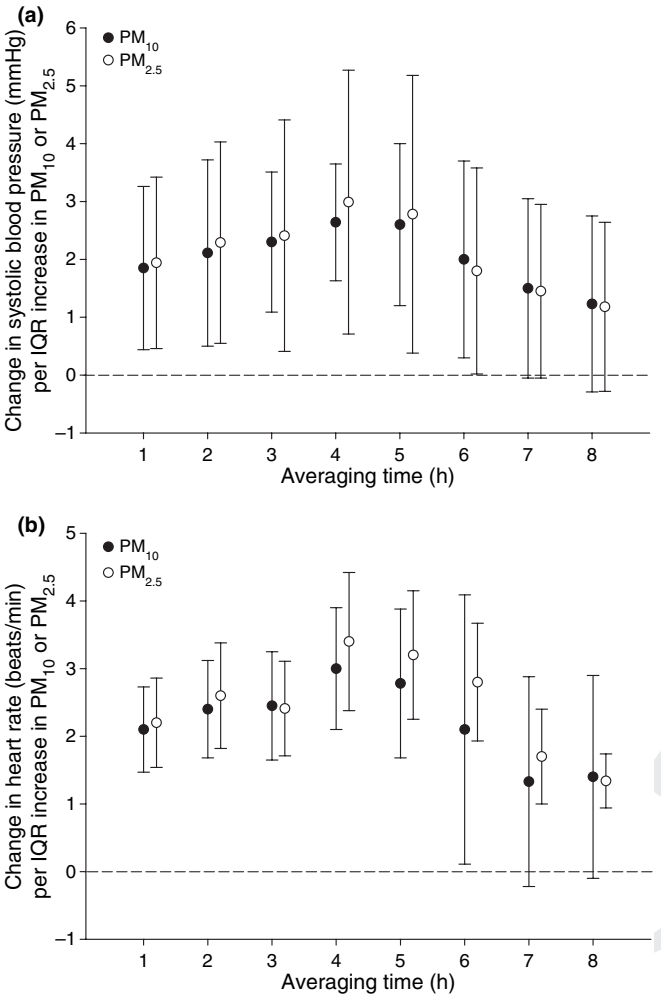

Fig. 2 The effects of particulate matter less than 10 μm in diameter (PM_{10}) and particulate matter less than 2.5 μm in diameter ($PM_{2.5}$) on systolic blood pressure (a) and heart rate (b) estimated by single-pollutant mixed-effects regression models, scaled to interquartile range increase in levels for individual hour mean. Error bars indicate 95% confidence intervals

COPD reported by Linn et al. (1999) (0.17/0.10 mmHg increase in SBP/DBP per 10.5 $\mu g/m^3$ ambient PM_{10} at 1-day mean). The effects of ambient $PM_{2.5}$ on cardiac rehabilitation patients reported by Zanobetti et al. (2004) (2.8/2.7 mmHg increase in SBP/DBP per 10.5 $\mu g/m^3$ ambient $PM_{2.5}$ at 5-day mean) were also greater than the effects of indoor $PM_{2.5}$ on physiological parameters presented in our study (3.0/2.4 mmHg increase in SBP/DBP per 18.8 $\mu g/m^3$ indoor $PM_{2.5}$ at 4-h mean). Our previous study showed a 3.4 mmHg increase in SBP, a 2.2 mmHg increase in DBP, and a 1.2 beats/min increases in HR among patients with lung function impairments for 10,000 particles/cm^3 increases in ambient submicrometer particles at 2-h mean (Chuang et al., 2005). These findings were more dramatic than indoor PM effects observed in this study.

Table 3 The effect modification of association of indoor particles exposure with blood pressure and heart rate by windows status

	Windows status		
	Open	Close	*P*-value, interaction
Systolic blood pressure, mmHg			
4-h mean PM_{10} (95%CI)	2.98 (2.75, 3.22)*	−0.41 (−1.02, 0.20)	0.002
4-h mean $PM_{2.5}$ (95%CI)	2.21 (1.21, 3.20)*	0.68 (−0.02, 1.38	0.060
Diastolic blood pressure, mmHg			
4-h mean PM_{10} (95%CI)	1.97 (1.42, 2.52)*	0.22 (−0.02, 0.46)	<0.001
4-h mean $PM_{2.5}$ (95%CI)	1.48 (0.71, 2.25)*	0.91 (−0.09, 1.90)	0.629
Heart rate, beats/min			
4-h mean PM_{10} (95%CI)	2.78 (1.21, 4.35)*	0.45 (−0.01, 0.92)	0.035
4-h mean $PM_{2.5}$ (95%CI)	3.01 (1.51, 4.50)*	−0.31 (−2.31, 1.69)	0.576

*P-value <0.05.

Although comparisons can only be approximate, these findings imply that young adults are less susceptible to PM exposure or that indoor PM may have less of an effect on BP compared with ambient PM. Another interesting finding of this study was that windows status (open vs. closed) seems to modify the particulate effects on BP and HR: greater PM effects on BP and HR were observed during visits with windows-open compared with those during the visits with windows-closed. Such findings implied that exposure to infiltrated outdoor particles and indoor particles might have greater effects on BP and HR. Closing windows could reduce indoor PM concentrations and modify the effect of PM_{10} on BP and HR in young adults in Taipei, Taiwan.

Modulation of the autonomous nervous system plays an important role in hypertension and alteration of BP and HR (Grassi, 1998; Malpas, 1998). Our findings related to indoor PM exposure on BP and HR in this study, and ambient PM on BP and HR in our previous study (Chuang et al., 2005) suggest that PM can have both immediate and cumulative effects on cardiac autonomic function in human subjects. Studies have also shown that vascular endothelial perturbation (Bouthillier et al., 1998) and systemic inflammation (Peters et al., 1997; Schwartz, 2001), alter sympathetic nerve activity and BP (Nakamura et al., 1999) and occurs hours and even days after PM exposures in human subjects or experimental animals. Accordingly, we believe particles-induced systemic inflammation could indirectly result in BP and HR changes in the delayed phase after indoor PM exposures at 3- to 4-h means.

The association between indoor PM and increase in BP and HR found in the study could be confounded by unavailability of indoor exposure data for other air pollutants. The lack of indoor exposure to ozone, sulfur dioxide, and particle

compositions, such as elemental contents, carbon contents, or soluble ions, may still confound the observed associations between indoor PM and increase in BP and HR. As these unmeasured air pollutants are usually correlated with PM, the outcomes of this study may be bias for either positive or null results (Zeger et al., 2000). Moreover, the association between indoor PM and BP could be also confounded by the reduction of outdoor noise, which has been reported to be associated with BP change (Penney and Earl, 2004). Regardless of this limitation, we believe our data generally support that changes in indoor PM can lead to increases in BP and HR. Reduction of particles in home by closing windows can improve autonomic function in a panel of healthy, nonsmoking, young students.

Although elevated BP and HR can serve as markers for altered autonomic activity (Bootsma et al., 1994; Grassi, 1998) and increased risks of cardiovascular diseases (Kannel, 1990), it is still unknown whether short-term and small fluctuations of autonomic activity are associated with higher risks of cardiovascular diseases clinically. Therefore, further studies are needed to determine whether the indoor PM-associated autonomic activity fluctuations observed in this panel study will have meaningful implications of cardiovascular mortality clinically.

Acknowledgements

This study was supported by grants (NSC 095-SAF-I-564-602-TMS and NSC 97-2314-B-562-001) from National Science Council of Taiwan.

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