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Original Investigation

Environmental tobacco use and indicators of metabolic syndrome in Chinese adults

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Abstract

Introduction: Exposure to environmental tobacco smoke (ETS) is a widespread source of nicotine exposure, and an estimated 540 million Chinese are exposed to ETS in mainland China. We aimed to investigate associations of ETS exposure and metabolic syndrome (MetS) as well as its individual components independent of active smoking status in Chinese adults.

Methods: A cross-sectional data of 304 randomly selected Chinese households with fourth (elementary school) and seventh (middle school) graders in Qingdao city was used. Assessments of fat mass, metabolic biomarkers, personal history of illness, and health behaviors were conducted.

Results: Proportions of current smokers were 3% in women and 60.5% in men, and more men reported exposure to ETS 5–7 days per week than women (60.8% vs. 48.1%). Exposure to ETS was significantly associated with enhanced risks of MetS (odds ratio [*OR*] = 2.8, $p = .01$), hypertriglyceridemia (*OR* = 2.1, $p = .02$), and central obesity (*OR* = 2.7, $p < .001$) and reduced levels of high-density lipoprotein cholesterol (*OR* = 1.9, $p = .02$) and elevated mean levels of fasting insulin ($p < .01$). These observed associations were independent of active smoking status and were successfully replicated in female never-smokers.

Conclusions: Results of our study support the hypothesis that ETS exposure is independently associated with MetS and its individual components. Further large-scale studies with longitudinal design and objective assessment of ETS exposure are needed to elucidate the underlying mechanisms and the causal effects of passive smoking on MetS. Findings of this work emphasize the importance of developing community intervention to reduce smoking, ETS, and promote healthy lifestyle.

Introduction

China is the world's largest producer and consumer of tobacco according to the report from the World Health Organization Framework Convention on Tobacco Control (WHO FCTC). As revealed in findings from 1996 (Yang et al., 1999) and 2003 National Surveys (Shi, Liu, Zhang, Lu, & Quan, 2008), about 60% of men and 3% of women smoke, equivalent to an estimated population of 350 million. A growing body of literature indicates that tobacco smoke is associated with glucose intolerance (Houston et al., 2006), insulin resistance (Facchini, Hollenbeck, Jeppesen, Chen, & Reaven, 1992), dyslipidemia (increased low-density lipoprotein [LDL] cholesterol and decreased high-density lipoprotein [HDL] cholesterol; Li, Xu, & Xia, 2006; Tonstad & Svendsen, 2005), endothelial dysfunction, and a hypercoagulable state (Ferrence, Slade, Room, & Pope, 2000) and unhealthy body fat distribution (Bamia, Trichopoulou, Lenas, & Trichopoulos, 2004; Canoy et al., 2005), which are all related to development of metabolic syndrome (MetS), a clinical condition defined by the clustering of several risk factors of diabetes and cardiovascular diseases including impaired glucose tolerance and insulin resistance, high blood pressure, abnormal lipid profiles, and central obesity (International Diabetes Federation, 2005). Growing attention has been paid to exposure to environmental tobacco smoke (ETS) in forms of secondhand or passive smoking and its adverse health outcomes. Exposure to ETS is a widespread source of nicotine exposure (Hammond, 1999), and an estimated 540 million Chinese are exposed to secondhand smoke according to the recent national tobacco control report (Shi et al.). Passive smoke contains similar toxins to active smoke but is produced at different temperatures and different reducing conditions; therefore, some toxic substances may even be more concentrated in passive smoke (U.S. Environmental Protection Agency, 1992; Chan-Yeung & Dimich-Ward, 2003; Houston

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et al.; National Cancer Institute, 1999). A positive association between self-reported and/or objective measures of ETS exposure and circulating concentrations of nicotine and/or biomarkers of nicotine was often reported, and nicotine exposure from ETS can engender plasma nicotine concentrations that are equivalent to levels produced by tobacco smoking (Okoli, Kelly, & Hahn, 2007). The adverse metabolic effects of ETS can be larger than one would expect on the basis of the risks associated with active smoking and the relative doses of tobacco smoke delivered to smokers and nonsmokers (Barnoya & Glantz, 2005). Rapidly accumulating evidence suggests that multiple metabolic systems and functions including platelet and endothelial function, arterial stiffness, atherosclerosis, glucose tolerance and insulin resistance, inflammation, energy metabolism, and body fat distribution are exquisitely sensitive to the toxins in ETS, and adverse effects of even brief (minutes to hours) passive smoking are often nearly as large (averaging 80%–90%) as chronic active smoking (Barnoya & Glantz).

Accompanying its rapid economic development, China is undergoing a remarkably fast but undesirable nutrition transition (Popkin, 2001a, 2001b), which has been related to recent dramatic increase of MetS. The estimated prevalence of MetS was 13.2% in a study of a large professional population in Beijing from 2003 to 2004 (Li et al., 2006) and 9·8% in men and 17·8% in women from a nationally representative sample of Chinese adults aged 35–74 years in 2000–2001(Gu et al., 2005). In this paper, we investigate associations of exposure to ETS and MetS as well as its individual components independent of active smoking status. The independent effects of ETS were first analyzed in the overall sample with adjustments for active smoking status and other covariates, and then a subgroup of female never-smokers was analyzed to cross-validate findings in the overall sample.

Methods

Sample selection and procedures

A cross-sectional pilot household study was conducted among 304 randomly selected fourth (elementary school) and seventh (middle school) graders and their parents living in Qingdao city, Shandong province, China, between December 2005 and March 2006. The purpose was to assess capacity and training needs required to carry out large-scaled household data collection in support of future research to develop, implement, evaluate, and disseminate community-based family interventions on smoking prevention and cessation, environmental tobacco exposure, and overweight/obesity prevention in both youths and parents. A stratified sampling strategy was utilized to randomly select primary and middle school students. Two city districts with the highest and lowest residential incomes were identified. Within each identified district, the primary and middle schools were grouped according to low and high academic performance. Two primary schools and two middle schools were randomly selected from each of four (two levels × two districts) clusters to participate in the study. One class each from fourth (elementary school) and seventh (middle school) grades was randomly selected, and half of the students in each selected class were recruited for the study. Parents or guardians of the students were approached by research staff at Qingdao Centers of Disease Control and Prevention (CDC). Parents were invited and at least one parent (e.g., mother, father, or both) participated in the study. Parents were asked to complete consent forms for students and themselves and were scheduled to come to the CDC with students for data collection.

Both parents and students were required to fast overnight and arrive at CDC early in the morning. A small amount of blood (10 ml) was drawn by research staff and transferred to two 5-ml tubes (one heparinized) for a free health screening on serum lipids and fasting plasma glucose (FPG) and insulin. Anthropometric assessments (i.e., weight, height, waist and hip circumferences, skin fold thickness at multiple sites, seated blood pressure), questionnaire survey and interviews on demographic information, personal history of illness and treatment/therapy (i.e., diabetes, hypertension, cardiovascular diseases, and cancers), and health behaviors were conducted after participants finished a free continental breakfast provided by CDC. Prior to the data collection, all research staff received training of assessment procedure based on a detailed protocol. All study procedures and survey instruments were approved by both the University of Southern California and Chinese Institutional Review Boards.

Measurements of cigarette smoking and ETS

Cigarette smoking

The following two questions were asked in the questionnaire to assess cigarette smoking: "Have you ever tried cigarette smoking, even a few puffs?" (0 = "no," 1 = "yes") and "During the past 30 days, on the days you smoked, how many cigarettes did you smoke per day?" The responses included, "I did not smoke during the past 30 days," "less than 1 cigarette per day," "1 cigarette per day," "2 to 5 cigarettes per day," "6 to 10 cigarettes per day," "11 to 20 cigarettes per day," or "more than 20 cigarettes per day." Based on the responses to these two items, subjects were further classified as never-smokers (never smoked in their lifetime), ex-smokers (smoked but not in the past month), and current smokers (smoked in the past month).

Environmental tobacco smoke

One question was asked in the questionnaire to assess exposure to ETS: "Think about the past seven days (one week). On how many of those days were you in a room or vehicle (buses, cars, ships, trains) with someone who was smoking?" There is no objective and clear consensus on the cutoff number of days for ETS exposure. The responses of number of days were further dichotomized as 0 for less than or equal to 4 days in the past week and 1 for 5–7 days in the past week.

Anthropometric measurements

All anthropometric measures were taken by trained research staff according to a standard protocol. Measurements were recorded to the nearest 0.1 cm (height, waist and hip circumference), 0.50 mm (skin fold thickness) and 0.1 kg (weight), while subjects were without shoes or thin socks and lightly clothed. Two measurements were taken for each measure and the average of two measurements was used for analysis. Height and weight were measured using a standard calibrated scale and stadiometer. Body mass index (BMI; weight in kilograms divided by height in meters squared) was used to quantify overweight and obesity status. Overweight and obesity were defined based on BMI of 25 and 30 Kg/m^2 as cutoff points recommended by

the World Health Organization (WHO) as well as BMI of 24 and 28 Kg/m^2 as cutoff points recommended by the Working Group on Obesity in China (WGOC; Zhou, 2002). Waist circumference was measured around the smallest circumference between the lowest rib and iliac crest, or, for obese subjects with no natural waist, midway between the lowest rib and iliac crest. Hip circumference was measured horizontally at the level of the greatest lateral extension of the hips. Waist-to-hip ratio was calculated using the waist and hip circumferences. Skin fold thickness at three sites (biceps, triceps, and subscapular) was measured with prior calibrated caliper.

Laboratory assessments and blood pressure measurement

After the participants had fasted overnight, blood samples were drawn to measure serum total cholesterol (TC), HDL cholesterol (HDL-C), triacylglycerols (TG), and plasma glucose and insulin. Blood specimens were stored in a −20°C refrigerator and immediately transported to the laboratory in batch in ice-cooled containers. Plasma glucose was determined by a glucose oxidase method within 3 hr after sampling. Fasting insulin was measured by radioimmunoassay. TC, HDL-C, and TG were measured enzymatically using commercial reagents. LDL cholesterol (LDL-C) concentrations were calculated using the Friedewald equation for those participants who had a TG concentration of <400 mg/dl (or 22.2 mmol/l): LDL-C = TC-HDL-C-TG/5 (Wildman, Gu, Reynolds, Duan, & He, 2004). Only one subject had TG level greater than 400 mg/dl and his/her LDL-C level was treated as missing value for further analysis. Seated blood pressure was measured by trained research staff using a standard mercury sphygmomanometer as standard protocol. Two blood pressure readings were taken with the participant resting for 10 min, and the mean of the two readings was calculated, as recommended by the American Heart Association (Perloff et al., 1993).

Definition of MetS

The MetS was diagnosed by the International Diabetes Federation (IDF) consensus worldwide definition of the MetS (International Diabetes Federation, 2005). With this new IDF definition, MetS is diagnosed when a subject meets criteria of central obesity (waist circumference ≥90 cm for Chinese men, ≥80 cm for Chinese women) plus any two of the following four factors: (a) raised TG level (≥150 mg/dl [1.7 mmol/L] or specific treatment for this lipid abnormality); (b) reduced HDL-C (<40 mg/dl [1.03 mmol/L] for men and <50 mg/dl [1.29 mmol/L] for women or specific treatment for this lipid abnormality); (c) raised blood pressure (systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥85 mm Hg or treatment of previously diagnosed hypertension); (d) raised FPG (FPG ≥100 mg/dl [5.6 mmol/L] or previously diagnosed type II diabetes).

Other sociodemographic and behavioral measures

Education attainment

Subjects' education levels were surveyed in categorical increments ranging from illiterate to college diploma or higher. The attainment of education was collapsed into three categories: below high school, high school, and college or above.

Family income

Monthly family income was determined by subjects' response to the question "What is your total monthly family income from all sources?" The response options ranged from "<100 Yuan/month" (<$12 USD/month) to ">10,000 Yuan/month" (>$1,200 USD/month) and was collapsed into "≤500 Yuan/month" (≤$60 USD/month) "501–2,000 Yuan/month" ($61–$240 USD/month) and ">2,000 Yuan/month" (>$240 USD/month).

Alcohol use

One question was asked to assess drinking status: "During the last 12 months, how often did you usually have any kind of drink containing alcohol? (Alcohol is a generic term for liquor, wine, beer, yellow wine [Huang Jiu], and fruit wine)." The responses included "I never drank any alcohol in my life," "I did not drink any alcohol in the past year, but I did drink in the past," and responses ranged from "every day" to "1 or 2 times in the past year." Based on the response, subjects were classified as never-drinkers (never drank in their lifetime), ex-drinkers (drank but not in the last 12 months), or current drinkers (drank in the last 12 months).

Data analysis

Descriptive statistics (mean, standard deviation, and percentage) were calculated to reflect the background characteristics of the sample. General linear mixed-effect models implemented in SAS Proc Mixed procedure were conducted to compare mean levels of BMI, peripheral and general fat mass, blood pressure, lipid profile, and fasting glucose and insulin between subjects with ETS exposure ≤4 days per week and their counterparts with ETS 5–7 days per week. Similarly, generalized estimating equation (GEE) models implemented in SAS Proc Genmod procedure were fitted to compare odds of having MetS or each of five related metabolic components (i.e., impaired glucose, high blood pressure, hypertriglyceridemia, reduced HDL-C, and central obesity) between groups of subjects with different exposure levels of ETS. Both mixed-effect and GEE models were enabled to account for interdependence of spouse data within family during the analysis (Fitzmaurice, Laird, & Ware, 2004; Littell, Milliken, Stoup, & Wolfinger, 1996). Two sets of analysis were conducted. First, we examined independent effects of ETS in the overall sample combining never-smokers, ex-smokers, and current smokers with adjustments for active smoking status as well as other covariates including age, gender, education attainment, family income, and alcohol drinking status. As majority of women (76.1% in Table 1) were never-smokers, we reexamined ETS effects within this subgroup. General linear models and logistic regression were employed. In addition, moderating effects of smoking status (never-smokers and ex-smokers vs. current smokers) and weight status (overweight and obese vs. not overweight) on relationships between ETS and MetS and related metabolic components were explored. Subcategories of never-smokers and ex-smokers and overweight and obese were combined to avoid low cell frequency problem during the moderation analysis. Statistical analyses were carried out using SAS (v. 8.0; SAS Institute, Cary, NC).

Table 1. General characteristics of the sample

	Mother ($n = 208$)	Father ($n = 181$)	Overall ($N = 389$)
Age, years, mean (*SD*)	37.7 (3.7)	39.6 (4.2)	38.6 (4.0)
Education (*n*, %)			
Below high school	51 (25.0%)	39 (22.0%)	90 (23.6%)
High school	74 (36.3%)	63 (35.6%)	137 (36.0%)
College or above	79 (38.7%)	75 (42.4%)	154 (40.4%)
Annual family income, Yuan, mean (*SD*)			36,571.9 (42,235.8)
Active smoking status (*n*, %)			
Never-smoker	153 (76.1%)	19 (12.1%)	172 (48.0%)
Ex-smoker	42 (20.9%)	43 (27.4%)	85 (23.7%)
Current smoker	6 (3.0%)	95 (60.5%)	101 (28.2%)
ETS exposure			
≤4 days/week	108 (51.9%)	71 (39.2%)	179 (46.0%)
5–7 days/week	100 (48.1%)	110 (60.8%)	210 (54.0%)
Drinking status (*n*, %)			
Never drinker	107 (54.9%)	33 (18.3%)	140 (37.3%)
Ex-drinker	0 (0%)	26 (14.4%)	26 (6.9%)
Current drinker	88 (45.1%)	121 (67.2%)	209 (55.7%)
Weight status (*n*, %)			
WHO definition			
Overweight (25 $Kg/m^2 \leq$ BMI $<$ 30 Kg/m^2)	62 (30.1%)	96 (53.0%)	158 (40.8%)
Obesity (BMI $\geq$30 Kg/m^2)	5 (2.4%)	11 (6.1%)	16 (4.1%)
WGOC definition			
Overweight (24 $Kg/m^2 \leq$ BMI $<$ 28 Kg/m^2)	71 (34.5%)	82 (45.3%)	153 (39.5%)
Obesity (BMI $\geq$28 Kg/m^2)	16 (7.8%)	39 (21.6%)	55 (14.2%)
Metabolic syndrome (*n*, %)	12 (5.9%)	38 (21.1%)	50 (13.0%)
MetS components (*n*, %)			
Impaired glucose level	3 (1.5%)	12 (6.7%)	15 (3.9%)
High blood pressure	26 (12.5%)	62 (34.6%)	88 (22.7%)
Hypertriglyceridemia	18 (8.7%)	72 (40%)	90 (23.3%)
Reduced HDL-C	67 (32.5%)	53 (29.4%)	120 (31.1%)
Central obesity	56 (27.1%)	73 (40.3%)	129 (33.3%)

Note. BMI = body mass index; ETS = environmental tobacco smoke; MetS = metabolic syndrome; WGOC = Working Group on Obesity in China; WHO = World Health Organization.

Results

A total of 396 students were approached and 304 students (144 fourth and 160 seventh graders) agreed to participate in this study, including 236 of their mothers and 203 of their fathers and male guardians. The response/participation rate was 76.8%. Among participating households, 389 (64%) adults provided valid responses on item pertaining to ETS. For the purposes of this paper, parents' data with valid ETS responses ($N = 389$) were used for analysis to address our research questions as described in the introduction. The general characteristics of subjects are summarized in Table 1. Among 389 participants with valid responses on item related to ETS, 358 (92.0%) adults provided valid responses on items of active smoking status. The age of subjects ranged from 30 to 54 years. While a majority of women were never-smokers (76.1%) and never-drinkers (54.9%), 60.5% and 67.2% of men were current smokers and drinkers, respectively ($p < .01$). Relatively, more men reported exposure to ETS 5–7 days per week than women (60.8% vs. 48.1%, $p < .05$). Significant gender differences were observed in prevalence of overweight ($p < .001$), obesity ($p = .07$ for WHO definition and $p < .01$ for WGOC definition), MetS ($p < .001$), and related metabolic components ($p < .001$), except for reduced HDL-C ($p = .5$).

We first evaluated independent associations of ETS with indicators of MetS by comparing adjusted least-squared means of fat mass and metabolic biomarkers between subjects with different levels of exposure to ETS in the overall sample with combination of never-smokers and ex-smokers and current smokers (in Table 2). With an adjustment of active smoking status and other covariates, subjects with exposure to ETS ≤4 days per week had significantly lower levels of BMI, peripheral (hip circumference and skin fold thickness at biceps and triceps) and central (waist circumference and skin fold thickness at scapular) fat mass, smaller ratio of waist–hip ratio, higher level of serum HDL-C, and lower level of fasting plasma insulin than those with exposure to ETS 5–7 days per week. The independent effects of ETS on risks of MetS and each of the five related metabolic components were further evaluated (in Table 2). Additionally, with the adjustment of active smoking and other covariates, subjects with ETS 5–7 days per week had more than twofold greater odds of having MetS than those with ETS ≤4 days per week with adjusted odds ratio [*OR*] of 2.8 ($p = .01$).

Table 2. Comparison of fat mass and biomarkers between ETS levels

Mixed-effect models[a]	ETS ≤4 days/week Adjusted mean (*SE*)	ETS 5–7 days/week Adjusted mean (*SE*)	*p* value
BMI	23.8 (0.3)	25.2 (0.3)	<.001
Peripheral fat mass			
Biceps skin fold thickness	9.9 (0.5)	11.2 (0.4)	.02
Triceps skin fold thickness	14.3 (0.6)	16.61 (0.5)	<.001
Hip circumference	95.2 (0.6)	97.16 (0.6)	<.01
Central fat mass			
Scapular skin fold thickness	18.9 (0.6)	21.04 (0.6)	<.01
Waist circumference	79 (0.9)	82.2 (0.8)	<.01
Waist–hip ratio	0.8 (0.01)	0.85 (0.01)	.02
Blood pressure			
SBP	118.3 (1.4)	118.3 (1.2)	.9
DBP	78.9 (1.0)	79.65 (0.9)	.5
Lipid profile			
TG	1.5 (0.3)	1.44 (0.2)	.8
LDL-C	2.3 (0.1)	2.28 (0.09)	.9
HDL-C	1.4 (0.04)	1.22 (0.04)	<.01
TC	4.9 (0.1)	4.75 (0.09)	.2
Diabetes components			
Fasting glucose	4.2(0.1)	4.26(0.1)	.9
Fasting insulin	5.0(0.4)	6.56(0.4)	<.01

GEE models[b]	ETS 5–7 days/week vs. ETS ≤4 days/week *OR*	95% *CI*	*p* value
Metabolic syndrome	2.8	1.2–6.6	.01
Components			
Impaired glucose	1.1	0.3–4.1	.8
High blood pressure	1.1	0.6–1.9	.7
Hypertriglyceridemia	2.1	1.1–3.9	.02
Reduced HDL-C	1.9	1.1–3.1	.02
Central obesity	2.7	1.6–4.5	<.0001

Note. [a]Covariates controlled in mixed-effect models: age, gender, education, income, alcohol drinking, and active smoking status. BMI = body mass index; DBP = diastolic blood pressure; ETS = environmental tobacco smoke; GEE = generalized estimating equation; OR = odds ratio; SBP = systolic blood pressure; TC = total cholesterol; TG = triacylglycerols.

[b]Covariates controlled in GEE models: age, gender, education, income, alcohol drinking, and active smoking status. Reference group for *OR*: ETS ≤4 days per week.

Similar effects of ETS were observed in the risks of having hypertriglyceridemia (adjusted *OR* = 2.1, $p = .02$), reduced HDL-C (adjusted *OR* = 1.9, $p = .02$), and central obesity (adjusted *OR* = 2.7, $p < .001$). In addition, we explored moderation effects of smoking and weight status with ETS on MetS and related metabolic components (Table 3). Significant moderating effects of active cigarette smoking were observed in waist–hip ratio ($p = .02$) and level of serum TC ($p = .03$). Namely, effects of ETS were significant (for waist–hip ratio) or more pronounced (TC) only in never-smokers or ex-smokers, and not in current smokers. Being either overweight or obese significantly moderated the effects of ETS on waist circumference, waist–hip ratio, and fasting plasma insulin levels. The adverse effects of ETS were only observed among overweight or obese subjects.

As a result of the majority of women being never-smokers (76.1%), we further reexamined effects of ETS within female smokers only (in Table 4). Comparisons of adjusted mean levels of fat mass and biomarkers revealed significantly lower levels of BMI ($p < .01$), peripheral ($p = .01$ for skin fold thickness at biceps and triceps and borderline $p = .07$ for hip circumference) and central fat mass ($p < .01$ for skin fold thickness at scapular and $p = .01$ for waist circumference), smaller waist–hip ratio ($p = .02$), and significantly higher level of serum HDL-C ($p < .001$) and lower level of fasting plasma insulin ($p = .01$). A total of seven (4.7%) female never-smokers met the diagnosis criteria for MetS. Proportions of female never-smokers who met the diagnosis criteria for individual component were 2.0% ($n = 2$) for impaired glucose, 14.4% ($n = 22$) for high blood pressure, 8.6% ($n = 13$) for hypertriglyceridemia, 27.8% ($n = 42$) for reduced HDL-C, and 25.7% ($n = 39$) for central obesity. Due to the issue of low cell frequency, we could not fit logistic regression models for MetS and impaired glucose. Logistic regression models for risks of other components showed adjusted *OR* of 1.4 (95% *CI* = 0.5–3.7, $p = .2$) for high blood pressure, *OR* of 4.6 (95% *CI* = 1.2–17.4, $p = .03$) for hypertriglyceridemia, adjusted *OR* of 3.9 (95% *CI* = 1.7–9.0, $p = .001$) for reduced HDL-C, and adjusted *OR* of 2.5 (95% *CI* = 1.1–5.6, $p = .008$) for central

Table 3. Moderation effects of active smoking and overweight/obesity status

	ETS ≤4 days/week	ETS 5–7 days/week	
	Adjusted mean (*SE*)	Adjusted mean (*SE*)	*p* value
	Moderation effect of active smoking status[a]		
Waist–hip ratio	*p* for interaction = .02		
Never-smokers and ex-smokers	0.8 (0.01)	0.8 (0.01)	<.01
Current smokers	0.9 (0.01)	0.9 (0.01)	.6
TC	*p* for interaction = .03		
Never-smokers and ex-smokers	1.1 (0.2)	1.3 (0.2)	.09
Current smokers	2.8 (0.9)	1.7 (0.7)	.3
	Moderation effect of overweight status[b]		
Waist circumference	*p* for interaction = .03		
Not overweight (BMI <25 Kg/m^2)	74.6 (0.9)	74.5 (0.9)	.9
Overweight/obesity (BMI ≥25 Kg/m^2)	85.6 (1.1)	89.1 (0.8)	.02
Waist–hip ratio	*p* for interaction = .01		
Not overweight (BMI <25 Kg/m^2)	0.8 (0.01)	0.8 (0.01)	.4
Overweight/obesity (BMI ≥25 Kg/m^2)	0.9 (0.01)	0.9 (0.01)	.02
Fasting insulin	*p* for interaction = .04		
Not overweight (BMI <25 Kg/m^2)	4.5 (0.4)	4.9 (0.4)	.4
Overweight/obesity (BMI ≥25 Kg/m^2)	5.9 (0.8)	8.2 (0.6)	.03

Note. [a]Covariates: age, gender, education, income, and alcohol drinking status. BMI = body mass index; ETS = environmental tobacco smoke; TC = total cholesterol.

[b]Covariates: age, gender, education, income, alcohol drinking, and active smoking status.

obesity. We also explored the possible moderating effects of smoking and weight status and did not find any significant effects, which may be attributable to the small sample size.

Discussion

In this study, exposure to ETS was significantly associated with MetS and some individual components, including increased BMI, increased peripheral and central fat mass accumulation, reduced levels of HDL cholesterol, elevated levels of insulin resistance, enhanced risks for hypertriglyceridemia, and central obesity. These observed significant associations were independent of active smoking status and were successfully replicated in female never-smokers, who represent a majority of the female sample in this study. Our results were consistent to some previous studies reviewed by Barnoya and Glantz (2005).

The unfavorable effects of ETS exposure on the increase in high triglyceride level and decrease in HDL-C level may be in part attributed to the atherogenic change of blood vessels. Effects of ETS exposure have been attributed to the mediational influence of inflammation, plate aggregation, and/or endothelial dysfunction (Venn & Britton, 2007). Several inflammation markers such as circulating white blood cell count and C-reactive protein have been found to be significantly associated with enhanced risks of cardiovascular diseases and MetS and its individual components among passive smokers (Ford, 2003; Ishizaka et al., 2004; Ishizaka et al., 2007; Nagasawa et al., 2004; Wannamethee et al., 2005). ETS effects on the elevated insulin levels observed in our study were consistent with the findings from the Insulin Resistance Atherosclerosis Study, in which low insulin sensitivity was significantly associated with self-reported passive smoking (Henkin et al., 1999). Previous studies have shown that many long-term cigarette smokers are insulin resistant and hyperinsulinemic (Ishizaka et al., 2005; Nakanishi, Takatorige, & Suzuki, 2005; Oh et al., 2005; Weitzman et al., 2005). Proinflammatory cytokines, such as tumor necrosis factor-α and adiponectin, may explain the association between smoking and MetS (de Alvaro, Teruel, Hernandez, & Lorenzo, 2004). Decreased adiponectin is thought to play a major role in the development of insulin resistance and insulin insensitivity, which may in part explain observed enhanced risks of MetS in passive smokers (Facchini et al., 1992; Henkin et al.). In our study, ETS exposure was not significantly associated with high blood pressure. The literature on passive exposure to ETS and blood pressure is still limited and unclear.

Lower BMI in cigarette smokers compared with nonsmokers or quitters (i.e., those who stop smoking) has been reported in the literature and also linked to a negative implication for smoking cessation because of weight gain after quitting smoking (Froom, Melamed, & Benbassat, 1998; Hughes, 1992; Patten & Martin, 1996; Piasecki, Fiore, & Baker, 1998). However, associations of tobacco smoke with metabolically adverse fat distribution profile and central obesity have been consistently documented in a small but growing body of literature (Bamia et al., 2004; Canoy et al., 2005; Leffondre, Abrahamowicz, Siemiatycki, & Rachet, 2002). In our study, exposure to ETS was positively associated with the subject's BMI and was related to increase in peripheral and central fat mass accumulation as well as enhanced risk of central obesity. The underlying biological mechanism of the observed significant association between smoking and the pattern of regional fat distribution is unclear. The possible impacts of tobacco smoke on sex hormones and on adipocytes have been suggested (Klesges, Eck, Isbell, Fulliton, & Hanson, 1990; Shimokata, Muller, & Andres, 1989; Troisi, Heinold, Vokonas, & Weiss, 1991; Visser, Launer, Deurenberg, & Deeg, 1999). For example, tobacco smoke may have an antiestrogenic effect by increasing the 2-hydroxylation of estradiol or inducing an imbalance

Table 4. Comparison of fat mass and biomarkers between ETS levels in female never-smokers

	ETS ≤4 days/week	ETS 5–7 days/week	
	Adjusted mean (*SE*)	Adjusted mean (*SE*)	*p* value
BMI	22.6 (0.4)	24.4 (0.4)	<.001
Peripheral fat mass			
Biceps skin fold thickness	11.1 (0.7)	13.5 (0.7)	.01
Triceps skin fold thickness	16.9 (0.7)	19.3 (0.7)	.01
Hip circumference	95.1 (0.8)	97.1 (0.8)	.07
Central fat mass			
Scapular skin fold thickness	19 (0.7)	22 (0.7)	<.01
Waist circumference	72.9 (0.9)	76.4 (1.0)	.01
Waist–hip ratio	0.8 (0.01)	0.8 (0.01)	.02
Blood pressure			
SBP	112.1 (1.7)	113.7 (1.8)	.5
DBP	74.2 (1.2)	76.4 (1.2)	.2
Lipid profile			
TG	1.0 (0.07)	1.0 (0.08)	.6
LDL-C	2.1 (0.1)	2.1 (0.1)	.6
HDL-C	1.6 (0.04)	1.4 (0.05)	<.001
TC	4.8 (0.1)	4.7 (0.1)	.3
Diabetes components			
Fasting glucose	4.0 (0.09)	4.2 (0.1)	.3
Fasting insulin	5.0 (0.4)	6.5 (0.4)	.01

Note. Covariates controlled in general linear models: age, education, income, and alcohol drinking. DBP = diastolic blood pressure; ETS = environmental tobacco smoke; SBP = systolic blood pressure; TC = total cholesterol; TG = triacylglycerols.

in androgenic to estrogenic activity in male and female smokers, which may finally influence regional distribution of body fat mass (Canoy et al.; Carney & Goldberg, 1984). Cigarette smoking may have a direct influence on adipose tissues for upregulation of the uptake and storage of fat mass (Canoy et al.; Carney & Goldberg). Accordingly, the adverse metabolic effects of ETS may even be exacerbated among overweight or obese subjects. This consideration is empirically supported by the observed significant moderation effects of overweight and obesity in our study. The negative effects of ETS exposure on waist circumference, waist–hip ratio, and fasting insulin levels were pronounced and only significant within overweight or obese subjects in our study.

One major limitation of our study is lack of objective measures of ETS exposure, such as air nicotine monitors (both passive and personal monitors) and biological markers (e.g., cotinine and/or nicotine) from various sources (e.g., serum, urine, hair, toenail, semen, saliva). ETS exposure could have been based on living with a spouse who is a smoker even if the smoker says he or she never smokes inside the home. The potential issue of misclassification due to lack of objective measure of ETS exposure may bias our results. In addition, we do not have data on how long and how frequently passive smokers were exposed to ETS. The amount of nicotine and other tobacco-related toxins from ETS exposure are highly related to the concentration, duration, and frequency of exposure (Howard & Thun, 1999; Jaakkola & Jaakkola, 1997). Due to the pilot nature of this study, the sample size is relatively small. As a result, we have few cases of men and women who were never-smokers and diagnosed with MetS and impaired glucose tolerance, which limited us to further explore ETS effect on risks of having MetS and impaired glucose tolerance directly. Finally, our results may only be generalizable to the Chinese population, which may have a different cardiovascular disease risk profile than other populations such as Americans.

In conclusion, findings of our study support the hypothesis that ETS exposure is independently associated with MetS and its individual components. Further large-scale studies with longitudinal design and objective assessment of ETS exposure are needed to elucidate the underlying mechanisms and the causal effects of passive smoking on MetS.

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Declaration of Interests

None declared.

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