The association between smoking and hypertension in a population-based sample of Vietnamese men

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Objective The association between tobacco smoking and blood pressure in epidemiological studies remains unclear despite experimental evidence that smoking elevates blood pressure. This study examined the association between smoking and hypertension in a population-based sample of Vietnamese men.

Methods The study utilized a population-based sample of men ($n = 910$) from a survey of risk factors of noncommunicable diseases in Vietnam. Measurements including behavioural risk factors, body composition, and blood pressure were performed according to internationally standardized protocols. Poisson regression was used to obtain prevalence ratios and 95% confidence intervals (CIs). All analyses were performed using complex survey methods.

Results There were significant trends of increasing prevalence of hypertension with increasing years ($P = 0.05$) and pack-years ($P = 0.03$) of smoking after adjusting for age, BMI, and alcohol intake. Relative to never-smokers, the risk of hypertension for those who had smoked for 30 years or more and those who had smoked 20 pack-years or more were 1.52 (95% CI 0.95–2.44) and 1.34 (95% CI 0.94–1.91), respectively. Overall, however, current smokers were not at higher risk of hypertension than never-smokers (prevalence ratio = 1.08, 95% CI 0.70–1.68), and ex-smokers were more likely to be hypertensive than either never-smokers (prevalence ratio = 1.81, 95% CI 1.07–3.06) or current smokers (prevalence ratio = 1.67, 95% CI 1.25–2.23), similarly adjusted.

Conclusion In this population-based sample, hypertension was associated with smoking in a dose–response manner when characterized as number of years of smoking and lifetime cigarette consumption, but was not associated with current smoking status. _J Hypertens_ 28:245–250 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins.

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Abbreviations: CI, confidence interval; CVD, cardiovascular disease; STEPS, The STEPwise approach to surveillance of noncommunicable disease

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Introduction

Hypertension is an important contributor to cardiovascular disease (CVD) mortality worldwide [1], and identifying its risk factors is critical for better prevention efforts. Even though tobacco smoking is a well documented risk factor for CVD, its association with hypertension remains a paradox [2]. Smoking is associated with chronic low-grade inflammation [3] and arterial stiffness [4,5], which are associated with hypertension [6]. Carefully controlled experiments in healthy humans have shown that smoking causes an acute increase of blood pressure [4,7], and that smoking cessation reduces blood pressure, heart rate, and plasma epinephrine and norepinephrine concentrations among smokers [8]. Nevertheless, whereas some population studies have shown an association between tobacco smoking status and elevated blood pressure [6,9–11], others have failed to provide any evidence or even demonstrated a negative association [12–15]. In these epidemiological studies, the assessment of smoking has focused on smoking status (current, former, never). Very few studies have explored the dose–response relationship between hypertension and smoking. When dosage has been considered, it has been based on current consumption [9,16,17] or number of cigarettes smoked per day when smoking [18] rather than measures of lifetime dose, such as smoking duration or pack-years, which may be more relevant to hypertension risk.

Despite a rapid increase in prevalence of hypertension in developing nations of Asia in the last few decades, information on risk factors for hypertension in these populations are not available [19]. Apart from a study from Indonesia [14], little is known about the association between smoking and hypertension in these populations. Although the smoking prevalence in Vietnamese men has been among the highest in the world [20,21], the effects of smoking on hypertension has not been investigated in the Vietnamese population. A better understanding on this link will assist health authorities in making health promotion and intervention policies.

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We had the opportunity to investigate the association between tobacco smoking and hypertension in a population-based sample of Vietnamese men.

Method

Participants

Participants were 25–64-year-old men ($n = 910$) who participated in a population-based survey of risk factors of cardiovascular disease and diabetes in Can Tho, Vietnam. Participants included in this analysis were all males because the number of female smokers ($n = 19$) in the survey was too small to allow meaningful analyses. The survey was conducted using the World Health Organization (WHO) STEPwise approach to surveillance of noncommunicable disease (STEPS). Eligible participants for this survey were selected by multistage sampling with age, sex, and urban/rural stratification. Details about the survey have been published elsewhere [22]. Informed consent was obtained from participants. The study was approved by the Ethics Committee of Can Tho University of Medicine and Pharmacy. Data collection was carried out from July to November 2005.

Measurements

STEPS survey measures included demographic characteristics, behavioural risk factors (smoking, alcohol consumption, fruit and vegetable consumption, and physical activity), physical characteristics (weight, height, waist and hip ratio, and blood pressure), fasting blood glucose, and total cholesterol. All measurements were performed in accordance with the WHO STEPS protocols [23].

Tobacco smoking was assessed as a component of the STEPS questionnaire. Current smokers were those who reported that they currently smoked any tobacco products such as cigars, cigarettes or pipes either on a daily or nondaily basis. Current daily smokers were asked to provide information on the age they commenced smoking and the quantities of tobacco they smoked. Ex-smokers were those who previously smoked daily. Ex-smokers were asked to recall the ages at which they started and stopped smoking. Smokeless tobacco use and passive smoking were also recorded. The questionnaire was translated into Vietnamese and back-translated to ensure the appropriate meaning of each item was retained.

After participants had rested for at least 5 min, blood pressure was measured at the mid-point of the right arm using an Omron T9P digital automatic blood pressure monitor by trained staff. Two blood pressure readings were obtained for all participants. In accordance with the STEPS protocols in 2005, a third reading was taken if there was a difference of more than 25 mmHg for systolic blood pressure (SBP) or 15 mmHg for diastolic blood pressure (DBP) between the first two readings (this was required for only 24 men). The means of all measures were used. Substituting the mean of the two closest measures for the 24 men with three readings had a negligible impact on our results.

Statistical methods

Data were coded and presented according to the WHO guidelines [23]. Smoking duration in years was calculated as current age (current smokers) or age at quitting (ex-smokers) less the age commenced smoking. Pack-years of smoking was calculated by multiplying the number of years spent smoking by the number of cigarettes smoked daily, divided by 20 [24]. Hypertension was defined as SBP at least 140 mmHg and/or DBP at least 90 mmHg, or taking medication for hypertension. Only 29 men were taking medication for hypertension, and of them 19 had SBP at least 140 mmHg and/or DBP at least 90 mmHg. BMI was calculated as body weight divided by the square of height (kg/m^2). Poisson regression was used to estimate prevalence ratios and 95% confidence intervals (CIs). In the multivariable analyses, associations between smoking and hypertension were adjusted for age, BMI, and alcohol intake. All analyses were performed using complex survey methods provided by STATA software version 10.

Results

Table 1 presents the characteristics of the study participants. Hypertensive men were older, consumed more alcohol, and had higher BMI than their normotensive counterparts.

The prevalence of smoking in this sample has been presented elsewhere [22]. The proportions of current smokers, ex-smokers, and never-smokers were 67.8 (631/910), 13.0 (130/910), and 19.2% (149/910), respectively. On average, men in this sample smoked for 15.5 years and their lifetime cigarette consumption was 10.2 pack-years.

Table 2 presents the associations between smoking and hypertension. Current smokers as a group had only a marginally higher prevalence of hypertension than never-smokers. Ex-smokers had a higher prevalence of hypertension than either never-smokers ($P = 0.03$ adjusted for

Table 1 Characteristics of study participants ($n = 910$)

	Normotensive ($n = 590$) Mean ± 95% CI[a]	Hypertensive ($n = 320$) Mean ± 95% CI[a]	P
Age	38.1 ± 0.7	44.5 ± 1.3	<0.001
Years at school	7.5 ± 1.1	7.1 ± 1.2	0.367
Alcohol (standard drinks)[b]	7.1 ± 1.2	11.6 ± 2.9	0.002
Fruit + vegetable intake[c]	4.1 ± 0.5	3.7 ± 0.3	0.097
Physical activity[d]	106.4 ± 29.3	106.4 ± 29.3	0.999
Weight (kg)	55.4 ± 3.3	58.3 ± 2.6	0.097
BMI (kg/m^2)	21.0 ± 0.8	22.0 ± 0.7	0.044
Waist	73.9 ± 2.2	78.1 ± 2.5	0.005
Waist–hip ratio	0.84 ± 0.1	0.87 ± 0.1	<0.001

[a] 95% confidence interval. [b] Number of standard drinks consumed in the last 7 days. [c] Serving per day. [d] Moderate and vigorous physical activity in Metabolic Equivalent Task unit (MET)-hour per week.

Table 2 Association between measures of smoking history and current hypertension among Vietnamese men, 2005

	Years[a]	*n*/*N*	Model 1[b] PR[e] (95% CI)[f]	Model 2[c] PR[e] (95% CI)[f]	Model 3[d] PR[e] (95% CI)[f]
Smoking status					
Never smokers	0	47/149	1.00	1.00	1.00
Ex-smokers	16.5	65/130	1.80 (1.08–2.98)	1.93 (1.14–3.27)	1.81 (1.07–3.06)
Current smokers	20.0	207/630	1.06 (0.64–1.74)	1.17 (0.73–1.88)	1.08 (0.70–1.68)
Smoking duration					
Never-smokers	0	47/149	1.00	1.00	1.00
≤15 years	9.9	34/168	0.97 (0.59–1.59)	1.07 (0.64–1.79)	1.01 (0.62–1.65)
15–30 years	21.7	111/323	1.29 (0.72–2.31)	1.45 (0.85–2.48)	1.37 (0.83–2.24)
30+ years	35.7	120/240	1.48 (0.86–2.57)	1.70 (1.04–2.77)	1.52 (0.95–2.44)
P for trend			0.13	0.03	0.05
Lifetime cigarettes[g]					
Never smokers	0	47/149	1.00	1.00	1.00
<10 pack-years	13.8	43/176	0.90 (0.52–1.57)	1.02 (0.60–1.73)	0.96 (0.59–1.56)
10–20 pack-years	22.0	68/218	1.09 (0.61–1.92)	1.27 (0.74–2.18)	1.17 (0.72–1.91)
20+ pack-years	29.6	86/196	1.30 (0.84–2.01)	1.50 (1.00–2.26)	1.34 (0.94–1.91)
P for trend			0.09	0.02	0.03

[a] Mean years of smoking. [b] Adjusted for age. [c] Adjusted for age and BMI. [d] Adjusted for age and BMI, and alcohol consumption. [e] Prevalence ratio. [f] 95% confidence interval. [g] Excludes ex-smokers, who did not provide information on numbers of cigarettes smoked per day.

age, BMI and alcohol consumption) or current smokers (prevalence ratio = 1.67, 95% CI 1.25–2.23, $P < 0.01$ similarly adjusted). However, when smokers were categorized by their years of smoking or by the lifetime number of cigarettes smoked, dose–response relationships with hypertension were revealed. Men who smoked for longer duration or had higher lifetime cigarette consumption were at significantly higher risk of hypertension. These trends persisted after adjusting for BMI and alcohol intake. Adjusting for waist circumference instead of BMI resulted in only minor changes to these estimates (data not shown).

Associations between smoking and blood pressure were similar to those between smoking and hypertension. Ex-smokers had higher SBP and DBP than never-smokers (see Table 3). The mean SBP of participants by duration of smoking and lifetime cigarette consumption are depicted in Fig. 1. There was a trend of increasing SBP with increasing years of smoking ($P = 0.02$ adjusted for age, BMI and alcohol intake). For lifetime cigarettes, the trend was not significant ($P = 0.25$ similarly adjusted).

To investigate possible reasons for the higher blood pressure among ex-smokers, Table 3 presents means of possible explanatory factors for participants categorized by smoking status. Ex-smokers were older, less physically active, and spent more time sitting than the other two groups. In additional analyses, sitting time was found to be associated with hypertension ($P = 0.04$), whereas physical activity was not ($P = 0.54$). However, adjusting for physical activity and sitting time did not alter the relationship of smoking status and hypertension with the mean SBP for those who were not on medication for hypertension (this excluded 29 men who were taking antihypertensive medication) and prevalence of hypertension remaining higher for ex-smokers (data not shown). Adjusting for moderate-intensity activity instead of total physical activity did not change the results either (data not shown).

Discussion

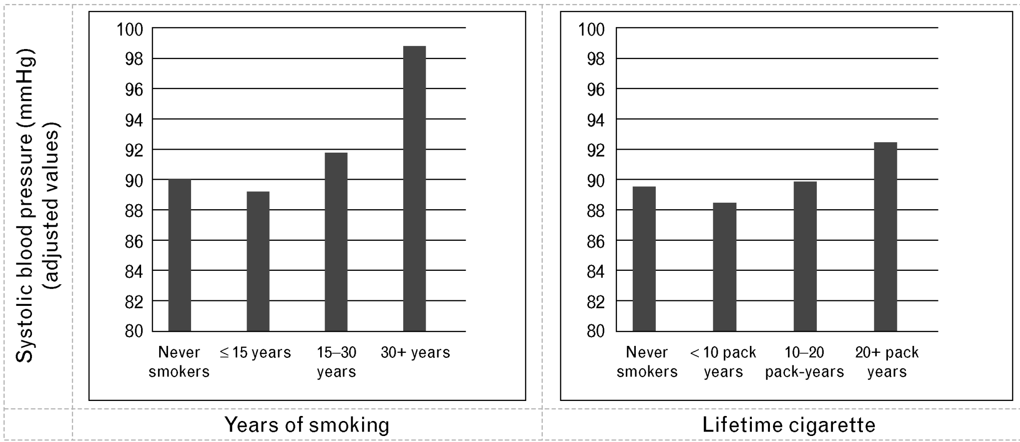
The study examined the association between smoking and hypertension in a population-based sample of Vietnamese men. We found that smoking duration and

Table 3 Characteristics of study participants categorized by smoking status

	Never smokers ($n = 149$) Mean ± 95% CI[a]	Ex-smokers ($n = 130$) Mean ± 95% CI[a]	Current smokers ($n = 631$) Mean ± 95% CI[a]
Age	37.2 ± 1.9	42.6 ± 1.8	40.0 ± 0.7
Household income (US$/year)	2332 ± 1139	1812 ± 628	1568 ± 239
Years at school	9.5 ± 1.9	7.6 ± 1.2	6.9 ± 0.8
Alcohol (standard drinks)[b]	3.6 ± 1.3	7.8 ± 4.3	9.7 ± 2.5
Fruit + vegetable intake[c]	4.4 ± 0.3	4.0 ± 0.6	3.9 ± 0.3
Physical activity[d]	115.9 ± 45.1	87.6 ± 42.8	106.3 ± 25.6
Sitting time (h/day)	3.8 ± 0.9	4.0 ± 0.5	3.8 ± 0.8
Weight (kg)	58.3 ± 2.4	57.0 ± 2.6	55.4 ± 1.9
BMI (kg/m^2)	22.1 ± 1.1	21.3 ± 0.7	21.0 ± 0.7
Waist (cm)	76.7 ± 2.7	76.8 ± 2.5	74.2 ± 2.0
Waist–hip ratio	0.85 ± 0.01	0.86 ± 0.01	0.84 ± 0.02
SBP (mmHg)	126.9 ± 2.6	135.4 ± 5.0	127.5 ± 2.2
DBP (mmHg)	78.4 ± 2.0	84.8 ± 3.6	79.1 ± 2.2

[a] 95% confidence interval. [b] Number of standard drinks consumed in the last 7 days. [c] Serving per day. [d] Moderate and vigorous activity in Metabolic Equivalent Task unit (MET)-hour per week.

Fig. 1



Mean of SBP by smoking duration and lifetime cigarette consumption after adjusting for age, BMI, and alcohol intake. Excluded from analyses were 29 men who were taking medication for hypertension.

higher lifetime cigarette consumption were each associated with a higher risk of hypertension in a dose–response fashion in this male population with high smoking prevalence and independent of age, BMI, and alcohol consumption. However, current smokers were not at higher risk of hypertension than never-smokers; rather, ex-smokers were more likely to be hypertensive than either never-smokers or current smokers.

A link between tobacco smoking and blood pressure is biologically plausible and cigarette smoking has been observed to cause acute increases in blood pressure in experimental settings [7]. Cigarette smoking increases sympathetic outflow [25], possibly through an increased release and/or reduced clearance of catecholamines at the neuroeffector junctions [26]. In addition, smoking is associated with chronic low-grade inflammation [3] and arterial stiffness [4,5], which are associated with hypertension [6].

In the context of lung cancer, Doll and Peto [27] have emphasized the importance of quantifying the dose of tobacco exposure in terms of years of smoking and total number of cigarettes smoked. This is in accordance with the principle of toxicology that the dose of exposure (determined by frequency of exposure, length of exposure, and other factors) determines the response of the organism [28]. Experimental studies have shown that the number of cigarettes consumed is associated with acute increases in blood pressure following a dose–response pattern [8]. Furthermore, smoking high-nicotine cigarettes has been shown to induce a higher and more sustained elevation of blood pressure than smoking low-nicotine cigarettes [29]. In epidemiological studies, when the chronic effects of past smoking are investigated, cigarette smoking has been associated with dose-related impairment of endothelium-dependent arterial dilation [30], and more pack-years of smoking is associated with progression of atherosclerosis [31]. In this study, a clear association between smoking and blood pressure emerged once dosage was taken into account. Men who smoked for longer duration or consumed more pack-years of cigarettes had a higher risk of hypertension after adjusting for age, BMI, and alcohol consumption. It is possible that the different results observed for smoking status and duration or pack-years may explain some of the equivocal findings of previous epidemiological studies which focused on smoking status as the measure of tobacco exposure and did not take into account the duration of smoking and lifetime cigarette consumption [6,9–15,32–34].

A higher prevalence of hypertension among ex-smokers has been reported in previous studies [35,36]. In each study, weight gain following smoking cessation was ruled out as a potential explanation. Likewise, adjusting for BMI did not change the association observed in this study. Whereas ex-smokers did less physical activity and tended to spend more time sitting, adjusting for physical activity and sitting time did not alter the results. The possibility of reverse causality, whereby ex-smokers may have quit because they were told they had hypertension, cannot be ruled out as a potential explanation for the higher prevalence of hypertension observed among ex-smokers in this study.

A key strength of this study was its use of a representative sample, recruited with a high response proportion. The use of WHO standardized protocols, intensive training of data collection staff, prestudy testing of procedures, and the close supervision of staff during data collection, all highlight the attention that was paid to minimizing avoidable sources of measurement error. The availability of various measures of smoking and covariates including demographic characteristics, alcohol use, fruit and vegetable consumption, physical activity, and body composition allowed us to investigate the association between smoking and hypertension thoroughly. The low proportions of treated hypertensive men may have enabled us to better estimate the association of blood pressure with smoking.

The study has some limitations. Firstly, we did not measure quantity of tobacco use for ex-smokers, and their pack-years of smoking could not be calculated. However, ex-smokers comprised only a small proportion (17%) of ever-smokers. Secondly, we also did not have an objective measure of dietary sodium. The indirect indicators of salt intake measured by the STEPS questionnaire did not show any association with blood pressure, but measurement of salt intake by questionnaire is problematic [37]. Failure to adjust for an accurate estimate of sodium intake may have influenced the findings of this study. Lastly, the analyses of this study were restricted to men because very few of the women in our sample were smokers. Smoking has deleterious effects for women [38], and plausibly the dose–response effects observed for men would occur for women as well, but this remains to be investigated in a population with a higher prevalence of female smokers.

In summary, lifetime smoking duration and intensity (pack-years) were each associated with hypertension in a dose–response manner in this population-based sample of Vietnamese men.

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There are no conflicts of interest.

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