

Cigarette Smoking, Blood Pressure and Serum Lipids and Lipoproteins in Middle-Aged Women

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Abstract The relationship of cigarette smoking with blood pressure and serum lipids and lipoproteins was studied in the 3934 middle-aged women aged 40 to 59 years. After adjusting age, body mass index (BMI), alcohol intake and physical activity scores, the mean systolic and diastolic blood pressures (SBP and DBP, respectively) did not indicate dose-dependent relationships. The largest significant mean differences in SBP (4.6 mmHg), DBP (3.9 mmHg), high density lipoprotein cholesterol (HDL-C) (9.6 mg/dL), ratio of total cholesterol to HDL-C (TC/HDL-C) (0.8), triglycerides (TG) (22.9 mg/dL) and the logarithmic transformation of TG (Log TG) (0.26) were found between the non-smokers and smokers. When age, BMI, alcohol intake and physical activity scores were included in the forward stepwise multiple regression analyses, there were negative relationships found for cigarette smoking and SBP, DBP and HDL-C and positive relationships for cigarette smoking and TC/HDL-C, TG, Log TG and low density lipoprotein cholesterol. Although the results are somewhat variable, the present study shows cigarette smoking is negatively associated with SBP and DBP and unfavorably associated with serum lipids and lipoproteins in middle-aged women. *J Physiol Anthropol*, 20 (1): 1–6, 2001 <http://www.jstage.jst.go.jp/en/>

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Introduction

The unfavorable effects of cigarette smoking on serum or plasma lipids and lipoproteins have been reported in a metaanalysis (Craig et al., 1989). The relationship between cigarette smoking and blood pressure (BP) has also been reported by Green et al. (1986) who examined

the results of 15 published studies and stated that a negative association between smoking and BP was consistent over a wide range of cross-sectional epidemiological studies. However, limited number of studies have specifically examined the dose-dependent relationship between cigarette smoking and BP (Brischetto et al., 1983; Gofin et al., 1982; Green et al., 1986; Handa et al., 1990; Imamura et al., 1996; Savdie et al., 1984). Among these, a dose-dependent relationship between cigarette smoking and BP has been demonstrated in men (Handa et al., 1990; Imamura et al., 1996). However, a dose-dependent relationship was not clearly demonstrated in women (Brischetto et al., 1983; Gofin et al., 1982; Green et al., 1986), although cigarette smokers, as a group, had lower BP than non-smokers in the 2 studies (Gofin et al., 1982; Green et al., 1986).

In a recent study from our laboratory (Imamura et al., 2000), using young women, we have reported that the mean high-density lipoprotein cholesterol (HDL-C) and the ratio of total cholesterol (TC) to HDL-C (TC/HDL-C) showed dose-dependent relationships with cigarette smoking, but the mean systolic and diastolic BP (SBP and DBP, respectively) did not indicate such relationships with cigarette smoking after adjusting for age, body mass index (BMI) alcohol intake and physical activity scores. The purpose of the present study was to examine the dose-dependent relationship between cigarette smoking with BP and serum lipids and lipoproteins in middle-aged women, while taking into account several potential confounding factors such as age, BMI, alcohol intake and physical activity habits.

Methods

Subjects

The subjects of the present study were 4,422 Japanese

women, ranging from 40 to 59 years of age. The subjects were self-referred to take physical examinations at the Minami Osaka Total Health Screening Center between August 1990 and March 1993. They were predominantly house-wives, whose socio-economic status was very similar. Of these 4,422 subjects, 488 were excluded because they were taking antihypertensive or lipid-lowering drugs. Of the remaining 3,934 subjects, 3,384 (86.0%) were non-smokers, 104 (2.7%) were ex-smokers, and 446 (11.3%) were smokers.

Subject information and measurements

The detail of methods in the present study have been reported elsewhere (Imamura et al., 1996). Briefly, information on smoking, drinking, and physical activity habits was obtained via a computer-administered questionnaire. The BMI was expressed as weight/height (kg/m^2). Subjects were seen after an overnight fast and were asked not to smoke on the morning of the examination. The SBP and 5th phase DBP were measured once in the morning by a trained nurse using a cuff and mercury sphygmomanometer after the subject had been sitting for more than 5 min. After BP was measured, blood was drawn from the antecubital vein. Serum lipids and lipoproteins measured were TC, HDL-C and triglycerides (TG). The blood analyzers were calibrated every morning with standard samples (Nihon Shoji, Osaka, Japan). Low-density lipoprotein cholesterol (LDL-C) was calculated using the following formula (Friedewald et al., 1972): $\text{LDL-C} = \text{TC} - \text{HDL-C} - \text{TG}/5$

Statistical analysis

Forward stepwise multiple regression analysis and analysis of covariance were performed using the general linear model of the Statistical Analysis System. Logarithmic transformation of TG (Log TG) was used to normalize a grossly skewed distribution of TG. The subjects were divided into 5 groups by their smoking habits: subjects who never-smoke, ex-smokers, and those who smoke 1–9 (light smokers), 10–19 (moderate smokers), and more than 20 (heavy smokers) cigarettes/day. Scores of 0–4 were given to the 5 levels of cigarette smoking. Scores of 0–2 were given to the 3 levels of physical activity (sedentary/ walk only/ play sports), and scores of 0–3 were given to the 4 levels of alcohol intake (non-drinker/ up to 1 drink/ up to 2 drinks/ more than 2 drinks per day). Forward stepwise multiple regression analysis was performed with SBP, DBP, TC, HDL-C, TC/HDL-C, TG, Log TG, or LDL-C as the dependent variables, and cigarette smoking scores, age, BMI, physical activity scores, and alcohol intake scores as independent variables.

Differences in proportions among groups were tested using the Chi-squared test. The unadjusted mean differences among the 5 groups determined on the basis

of smoking habits were determined by analysis of variance techniques. The Scheffe's method was used to identify specific significant differences when significant F-values were identified. Analysis of covariance was performed to adjust mean values for possible influences of confounding factors. A 2-sided p value of less than 0.05 was considered to be statistically significant.

Results

The distribution of subjects in the different smoking categories over the 4 seasons and physical activity did not differ significantly. However, smokers tended to have a higher alcohol intake than non-smokers (Table 1). Also, cigarette smoking had a significant positive correlation with alcohol intake ($r=0.220$; $P<0.001$).

Table 2 presents the unadjusted mean characteristics of subjects according to smoking status. The significant mean differences among different smoking categories were observed for SBP, DBP, HDL-C, TC/HDL-C, TG, Log TG and LDL-C.

Table 3 presents the adjusted mean BP and serum lipids and lipoproteins according to smoking status. The unadjusted mean characteristics shown in the Table 2 increased after these variables were adjusted for age, BMI, alcohol intake and physical activity scores. Ex-smokers and subjects in all smoking categories had significantly lower mean SBP and DBP than non-smokers. The largest mean differences in SBP (4.6 mmHg; 3.9%) and DBP (3.9 mmHg; 5.4%) were found between the non-smokers and smokers. Dose-dependent relationships in both SBP and DBP were not observed after possible confounding factors were adjusted for.

The significant mean differences among different smoking categories were observed in serum lipids and lipoproteins, except TC. When non-smokers and current smokers were compared, the largest mean differences in HDL-C (9.6 mg/dL; 15.5%), TC/HDL-C (0.8; 23.7%), TG (22.9 mg/dL; 27.4%) and Log TG (0.26; 6.0%) were found between the non-smokers and heavy smokers, and in LDL-C (7.7 mg/dL; 6.5%) was found between the non-smokers and moderate smokers. The relationships between cigarette smoking and HDL-C, TC/HDL-C, TG, and Log TG appeared to be dose-dependent.

Table 4 presents results of the forward stepwise multiple regression analyses. There were negative relationships between cigarette smoking and SBP, DBP and HDL-C and positive relationships between cigarette smoking and TC/HDL-C, TG, Log TG and LDL-C.

Discussion

Confounding factors and sampling bias

The results of our study may not be able to be extrapolated to the entire population, because the

Table 1 Season, Physical activity and alcohol intake

	No. of cigarettes/day				
	0	Ex-smoker	1-9	10-19	≥20
Season*					
spring	718 (21.2)	21 (20.2)	48 (23.2)	33 (19.8)	14 (19.4)
summer	980 (29.0)	27 (26.0)	62 (30.0)	57 (34.1)	28 (38.8)
fall	965 (28.5)	31 (29.8)	61 (29.5)	43 (25.7)	16 (22.2)
winter	721 (21.3)	25 (24.0)	36 (17.4)	34 (20.4)	14 (19.4)
Physical activity**					
Sedentary	1785 (52.7)	43 (41.3)	112 (54.1)	103 (61.7)	47 (65.3)
Walk only	971 (28.7)	34 (32.7)	56 (27.1)	43 (25.7)	14 (19.4)
Play sports	628 (18.6)	27 (26.0)	39 (18.8)	21 (12.6)	11 (15.3)
Alcohol intake***					
Non-drinker	2722 (80.4)	51 (49.0)	89 (43.0)	94 (56.3)	42 (58.3)
< 1 cup /day	583 (17.2)	43 (41.3)	91 (44.0)	37 (22.2)	10 (13.9)
< 2 cup /day	64 (1.9)	5 (4.8)	22 (10.6)	22 (13.2)	8 (11.1)
≥2 cup / day	15 (0.4)	5 (4.8)	5 (2.4)	14 (8.4)	12 (16.7)

Data show the number of subjects, with percentages given in parentheses. $\chi^2=9.12$, p=NS; ** $\chi^2=21.66$, p=NS; *** $\chi^2=563.03$, p<0.01.

Table 2 Subject characteristics

	No. of cigarettes/day					F value
	0 (n=3384)	Ex-smoker (n=104)	1-9 (n=207)	10-19 (n=167)	≥20 (n=72)	
Age (years)	47.8 ± 5.1	46.6 ± 5.5	47.5 ± 5.0	48.2 ± 5.6	47.1 ± 5.4	2.3
Height (cm)	154.7 ± 5.3	155.9 ± 4.7	154.9 ± 4.9	155.6 ± 4.8	156.0 ± 4.8	3.1
Weight (kg)	54.0 ± 7.1	55.6 ± 7.6	53.0 ± 6.5	53.8 ± 7.3	54.7 ± 8.7	2.6
BMI (kg/m ²)	22.5 ± 2.8	22.9 ± 2.8	22.1 ± 2.6	22.2 ± 2.8	22.5 ± 3.6	2.1
SBP (mmHg)	116.5 ± 14.4	113.1 ± 14.0	113.0 ± 15.3*	112.6 ± 14.3*	113.4 ± 14.6	7.4
DBP (mmHg)	72.6 ± 10.5	70.5 ± 9.7	69.6 ± 11.0*	69.2 ± 10.5*	69.9 ± 9.7	9.1
TC (mg/dL)	198.0 ± 33.1	199.5 ± 33.9	192.7 ± 33.6	198.5 ± 41.3	194.6 ± 39.7	1.4
HDL-C (mg/dL)	61.8 ± 16.1	59.6 ± 16.6	62.2 ± 16.5	55.5 ± 16.9*†	55.2 ± 18.5*†	9.0
TC/HDL-C	3.41 ± 1.05	3.57 ± 1.12	3.30 ± 1.02	3.90 ± 1.44*†	3.95 ± 1.66*†	13.56
TG (mg/dL)	83.9 ± 47.0	91.1 ± 41.1	90.7 ± 64.0	100.1 ± 67.2*	105.7 ± 50.8*	8.6
LogTG	4.32 ± 0.45	4.42 ± 0.42	4.39 ± 0.45	4.48 ± 0.47*	4.56 ± 0.44*	11.17
LDL-C (mg/dL)	119.4 ± 31.4	121.7 ± 32.0	112.4 ± 31.8	123.0 ± 39.3†	118.2 ± 40.5	3.2

Values are the mean ± SD. The unadjusted mean differences among the 5 groups divided on the basis of smoking habits were determined by analysis of variance techniques. *p<0.05 compared with non-smokers; †p<0.05 compared with ex-smokers; ‡p<0.05 compared with smokers smoking between one and 9 cigarettes/day. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; TG, triglycerides; logTG, logarithmic transformation of triglycerides; LDL-C, low-density lipoprotein-cholesterol.

subjects were self-referred, not a random sample of the general population. They were predominantly housewives of which only 446 (11.3%) subjects were smokers. However, any bias with respect to age, BMI, physical activity, and alcohol intake have been controlled by the forward stepwise multiple regression analysis. Furthermore, the mean values of BP and serum lipids and lipoproteins were adjusted for these confounding factors by analysis of covariance. Some other confounding factors such as coffee consumption, family history of hypertension, diet, oral contraceptive use, menopause and duration of cigarette smoking may possibly influence

the relationships of cigarette smoking to BP and serum lipids and lipoproteins (Green et al., 1986; Willett et al., 1983). However, no information on these factors was available in the present study.

Cigarette smoking and blood pressure

Brischetto et al. (1983), using 236 women and 214 men aged 16 to 69 years, reported that the results were similar in both sexes and did not find any relationship between cigarette smoking and BP. Gofin et al. (1982) reported that both SBP and DBP were significantly lower in smokers than in non-smokers, with greater differences in

Table 3 Adjusted mean (\pm SEM) of subjug characteristics

	No. of cigarettes/day				
	0 (n=3384)	Ex-smoker (n=104)	1-9 (n=207)	10-19 (n=167)	≥ 20 (n=72)
SBP (mmHg)	116.6 \pm 0.2	112.2 \pm 1.4**	112.8 \pm 1.0***	112.0 \pm 1.1***	112.4 \pm 1.7*
DBP (mmHg)	72.7 \pm 0.2	69.8 \pm 1.0**	69.5 \pm 0.7***	68.8 \pm 0.8***	69.1 \pm 1.2**
TC (mg/dL)	197.5 \pm 0.6	203.2 \pm 3.1	195.7 \pm 2.2	199.8 \pm 2.5	198.7 \pm 3.8
HDL-C (mg/dL)	62.1 \pm 0.3	58.5 \pm 1.5*	59.8 \pm 1.1*	53.3 \pm 1.2***††††	52.5 \pm 1.8***†††
TC/HDL-C	3.38 \pm 0.02	3.70 \pm 0.10**	3.50 \pm 0.07	4.06 \pm 0.08***††††	4.18 \pm 0.12***††††
TG (mg/dL)	83.7 \pm 0.8	91.1 \pm 4.7	92.7 \pm 3.3**	100.8 \pm 3.7***	106.6 \pm 5.6***††
LogTG	4.32 \pm 0.01	4.43 \pm 0.04*	4.41 \pm 0.03**	4.49 \pm 0.03***	4.58 \pm 0.05***††††
LDL-C (mg/dL)	118.7 \pm 0.5	126.5 \pm 2.9**	117.4 \pm 2.1††	126.4 \pm 2.3***††††	124.9 \pm 3.5

Data are the mean \pm SEM. Analysis of covariance was performed to adjust mean values for possible influences of age, body mass index, alcohol intake and physical activity. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ compared with non-smokers; † $p < 0.05$, †† $p < 0.01$, ††† $p < 0.001$ compared with ex-smokers; ‡ $p < 0.05$, ‡‡ $p < 0.01$, ‡‡‡ $p < 0.001$ compared with smokers smoking between one and 9 cigarettes/day. SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; TG, triglycerides; logTG, logarithmic transformation of triglycerides; LDL-C, low-density lipoprotein-cholesterol.

Table 4 Standardized partial regression coefficients

	SBP	DBP	TC	HDL-C	TC/HDL-C	TG	LogTG	LDL-C
Cigarette	-0.091 (0.250)***	-0.106 (0.182)***		-0.129 (0.275)***	0.147 (0.018)***	0.096 (0.818)***	0.108 (0.007)***	0.045 (0.532)**
Age	0.116 (0.043)***	0.116 (0.031)***	0.301 (0.098)***		0.163 (0.003)***	0.121 (0.147)***	0.167 (0.001)***	0.275 (0.092)***
BMI	0.242 (0.079)***	0.247 (0.057)***	0.126 (0.180)***	-0.305 (0.086)***	0.352 (0.006)***	0.226 (0.269)***	0.269 (0.002)***	0.217 (0.168)***
Alcohol	0.072 (0.369)***	0.083 (0.268)***	-0.076 (0.809)***	0.152 (0.407)***	-0.179 (0.026)***			-0.158 (0.785)***
PA			0.032 (0.652)*	0.053 (0.314)**	-0.031 (0.02)*	-0.036 (0.975)*	-0.036 (0.009)*	
R ²	0.086	0.092	0.120	0.125	0.198	0.078	0.117	0.155

Data show coefficients with the standard error given in parenthesis. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. BMI, body mass index; PA, physical activity; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; HDL-C, high-density lipoprotein-cholesterol; TG, triglycerides; logTG, logarithmic transformation of triglycerides; LDL-C, low-density lipoprotein-cholesterol.

middle-aged women than in men after adjusting for the effects of age, body mass, ethnic origin and season. Savdie et al. (1984) demonstrated negative dose-dependent relationships between cigarette smoking and both SBP and DBP independent of drinking status and gender. However, men and women were not reported separately in this study. Ribeiro (1983) examined the joint effects of biological and social dimensions on BP by means of identifying internally homogenous subgroup of female workers and reported that lean and young smokers had significantly lower DBP than the lean and young non-smokers. However, this study did not specifically examined the dose-dependent relationships. Green et al. (1986) reported that both SBP and DBP were significantly lower in smokers than in non-smokers in the older women, but not in younger women. Among male subjects, the mean DBP was significantly lower in smokers than in non-smokers in younger men, but not in older men. In contrast, in the present study using middle aged-women, the ex-smokers and smokers had significantly lower mean SBP and DBP than in non-smokers after adjusting age, BMI, physical activity and

alcohol intake scores. The present study as well as the previous study reported from our laboratory using young women (Imamura et al., 2000) and the study by Green et al. (1986) examined dose-dependent relationships, but such relationships were not observed.

In the present study, smokers as well as ex-smokers had significantly lower mean SBP (3.8 to 4.6 mmHg) and DBP (2.9 to 3.9 mmHg) than non-smokers. Although it has been suggested that a 2 mmHg reduction in DBP would result in a 17% decrease in the prevalence of hypertension as well as a 6% decrease in the risk of coronary heart disease (Cook et al., 1995), these results need to be treated with caution because, as noted by Gofin et al. (1982), it does not offset the substantial increase in coronary risk associated with smoking. However, as Green et al. (1986) stated, the role of smoking as a risk factor for hypertension is not supported by the results of this study.

The mechanisms by which chronic cigarette smoking decreases BP are unclear and need to be further elucidated. Three explanations have been proposed: (1) the alleviation of stress by cigarette smoking may

contribute to lowering BP (Handa et al., 1990); (2) regular smokers who do not smoke before and during their examination may experience a downward rebound of BP due to the short-term absence of a nicotine stimulus (Havlik et al., 1980); (3) there may be substances in cigarette smoke other than nicotine which have a hypotensive action (Savdie et al., 1984); (4) a reduced stroke volume may contribute to lowering BP because smokers tend to have higher heart rate than non-smokers (Green et al., 1986).

Cigarette smoking and serum lipids

Brischetto et al. (1983) demonstrated positive dose-dependent relationships between cigarette smoking and TC, TC/HDL-C and TG and a negative dose-dependent relationship between cigarette smoking and HDL-C after adjusting for age and weight. The results were similar in both sexes in this study. Willett et al. (1983), used women aged 20 to 40 years, reported that smokers had significantly higher TC/HDL-C and TG than non-smokers. These results did not change, even after age, weight, height, blood glucose, resting pulse, and oral contraceptive use were adjusted for. However, dose-dependent relationships were not observed in this study. In a recent study from our laboratory using women aged 20 to 39 years (Imamura et al., 2000), we have reported that the mean HDL-C and TC/HDL-C showed dose-dependent relationships with cigarette smoking. In contrast, in the present study, we have observed dose-dependent relationships not only between cigarette smoking and HDL-C and TC/HDL-C but also between cigarette smoking and TG and Log TG after adjusting possible confounding factors. Because the experimental methods and adjusted confounding factors in the present study were the same as in the previous study (Imamura et al., 2000), the disparities in findings between these two studies may reflect differences in the subjects characteristics (i.e., middle-aged v.s. young women) and/or in the duration of cigarette smoking.

Although the mechanisms by which cigarette smoking changes serum lipid and lipoprotein levels are not fully understood, possible explanations have been proposed by Brischetto et al. (1983). In brief, the nicotine stimulates the secretion of catecholamines as well as other hormones such as cortisol and growth hormone, leading to an increased serum concentration of free fatty acids which stimulates hepatic secretion of very low density lipoprotein and TG.

Some studies found that the mean SBP, DBP, TC, HDL-C, and/or TG (Brischetto et al., 1983; Criqui et al., 1980; Gofin et al., 1982; Green et al., 1986; Muscat et al., 1991; Savdie et al., 1984; Willett et al., 1983) in ex-smokers being similar to non-smokers. In the present study, there were no significant mean differences between the two groups in TC and TG. However, the ex-smokers showed

significantly lower mean SBP, DBP and HDL-C and higher Log TG, TC/HDL-C and LDL-C than non-smokers. The disparities in findings may reflect differences in the selection of subject populations and adjustments regarding confounding factors. However, with respect to BP, body weight changes may possibly affect the results. Seltzer (1974) reported that ex-smokers exhibited higher DBP over the 5-year interval when associated with weight gain, but there was no change in DBP for the ex-smokers who lost substantial amounts of body weight. Unfortunately, no information on body weight changes in different smoking categories was available in the present study.

In conclusion, the present study revealed that cigarette smoking was negatively associated with SBP and DBP, but dose-dependent relationships were not observed. The relationship of cigarette smoking to HDL-C, TC/HDL-C, TG, and Log TG appeared to be dose-dependent in the middle-aged women.

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