Effects of cigarette smoking on blood pressure stratified by BMI in Mongolian population, China

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Abstract

Background. The relationship between smoking and hypertension is still unclear and controversial; we examine effects of smoking on blood pressure stratified by body mass index (BMI) in the Mongolian population.

Methods. A total of 2589 Mongolians aged 20 years or more were recruited as study subjects. Demographic data, lifestyle factors, family history of hypertension, blood pressure measurements, physical examination and blood sample were obtained and analyzed for all subjects.

Results. Among subjects with BMI ≤ 25 kg/m², adjusted mean diastolic blood pressure in all smokers (82, 83 and 82 mmHg for subjects who smoke 1–9, 10–19 and ≥20 cigarettes/day, respectively) were lower than that in non-smokers (84 mmHg), all p-values < 0.05; among subjects with BMI ≥ 25 kg/m², mean systolic blood pressure (137 mmHg for non-smokers, 141, 135 and 132 mmHg for subjects who smoke 1–9, 10–19 and ≥20 cigarettes/day, respectively) decreased with amount of smoking and linear trend was statistically significant, p<0.05. Multivariate adjusted odds ratios of hypertension for three smoking groups were all not statistically significant.

Conclusions. This study did not provide support that smoking was a risk factor of hypertension and elevated blood pressure.

Key Words: Blood pressure, body mass index, hypertension, smoking

Introduction

Although smoking has been shown to raise blood pressure (BP) by vasoconstriction and accelerate heart beat as an acute effect in some studies (1–3), and smoking is a strong risk factor of cardiovascular disease (CVD) (4), its chronic effect on BP is much less certain. Niskanen and colleagues reported that current cigarette smoking was independently associated with increased risk of hypertension among middle-aged men in a population-based study (5). A positive association between current smoking and hypertension was reported among women who smoked at least 15 cigarettes per day in a prospective cohort study (6). However, some studies (7,8) showed that smokers had lower BP compared with non-smokers, and BP rose after smoking cessation (9,10). Halimi and colleagues (11) reported that a positive association of smoking with hypertension disappeared after adjustment for body mass index (BMI) among volunteers aged 20–69 years. Therefore, the relationship between smoking and BP and hypertension is still unclear and controversial (12).

The findings of our pre-study (13,14) suggested that prevalence of hypertension and rates of smoking were higher among Mongolian in a rural and animal husbandry area, Inner Mongolia, China, but the relationship between smoking and BP and hypertension had not been studied in the Mongolian population. The purpose of the present study was to examine effect of smoking on BP and hypertension stratified by BMI in the Mongolian population.

Methods

Study participants

A cross-sectional survey was conducted between 2002 and 2003. In the survey, two townships including 32 villages in Kezuohou Banner (county) and Naiman Banner in Inner Mongolia, China, were
selected for the study. The two adjacent townships are 100 km from Tongliao, a prefecture-level city in eastern Inner Mongolia, China. Most of the residents in the investigation field were Mongolian who had lived there for a long time from generation to generation; they have maintained traditional manners and customs of Mongolian ethnicity, and their professions were both farmers and herdsmen, and their diets were high in fat and salt. There were a total of 3475 Mongolian people aged 20 or over in the 32 villages, among them 2589 Mongolian people who signed informed consent in the field were included in the present analysis. A total of 886 did not sign informed consent and were not investigated because they were out of the field or refused to respond at the time of investigation. This study was approved by Soochow University School of Radiation Medicine and Public Health Ethics Committee.

Data collection and examination
Data on demographic characteristics, lifestyle risk factors, family history of hypertension and medical history were obtained using a standard questionnaire administered by trained staff. Cigarette smoker was defined as having smoked at least 1 cigarette per day (CPD) for 1 year or more. The amount and type of alcohol consumed during the past year was collected; alcohol drinking was defined as consuming at least 1 unit (50 g) alcohol per day for 1 year or more.

Three BP measurements were taken after taking rest for 30 min while the study participant was in the sitting position using a standard mercury sphygmomanometer according to a standard protocol. The first and fifth Korotkoff sounds were recorded as systolic BP (SBP) and diastolic BP (DBP), respectively. The mean of three BP measurements was used in all analyses. Hypertension was defined as SBP ≥140 mmHg and/or DBP ≥90 mmHg and/or use of antihypertensive medication in the recent 2 weeks. Normal BP was defined as SBP <140 mmHg and DBP <90 mmHg and no use of antihypertensive medication.

Body weight, height and waist circumference (WC) were measured by trained staff. The BMI was calculated as weight in kilograms divided by the square of the height in meters. Overweight was defined as BMI ≥25 kg/m², and central obesity was defined as WC ≥85 cm for males and ≥80 for females (15,16).

Fasting blood samples were collected in the morning after at least 8 h of fasting for all subjects; serum was subsequently isolated from the whole blood, and all serum samples were frozen at −80°C for test. Fasting blood glucose (FBG) was examined using a glucose meter (Roche, Basel, Switzerland) in the field; hyperglycemia was defined as FBG ≥ 6.1 mmol/l. Total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C) and triglycerides (TG) were analyzed enzymatically on a Beckman Synchrony CX5 Delta Clinical System (Beckman Coulter, Inc., Fullerton, CA, USA) using commercial reagents, and low-density lipoprotein-cholesterol (LDL-C) level was calculated by use of the Friedewald equation for the subjects. Hypercholesterolemia was defined as TC ≥5.18 mmol/l, hypertriglyceridemia TG ≥1.70 mmol/l, hyper LDL-cholesterolemia LDL-C ≥3.37 mmol/l, hypo HDL-cholesterolemia HDL-C <1.04 mmol/l (17).

Statistical analysis
The subjects were categorized into four groups based upon numbers of CPD: non-smoker, subjects who smoke 1–9 CPD, 10–19 CPD and ≥20 CPD. Unadjusted and age–gender-adjusted means of continuous variables and proportions of categorical variables were calculated for the four groups, respectively. Analysis of variance (ANOVA) was used in the intergroup comparison for continuous variables and Student–Newman–Keuls (SNK) used for comparison between each two groups. Age–gender-adjusted means were calculated and compared using a general linear model (GLM). Comparisons of categorical variables between/among groups were performed by χ² tests, and age–gender-adjusted prevalence was computed using a direct standardized method and compared using weighted χ² tests.

Multivariate adjusted means of SBP and DBP were calculated and compared using GLM. Associations between hypertension and CPD were analyzed using a logistic regression model. Linear and quadratic trends for means were tested using a contrast statement under GLM, and trend tests for odds ratio (OR) were conducted by using logistic model. All p-values were based on two-side test and a significance level of 0.05. Statistical analyses were conducted using SAS statistical software (version 9.1).

Results
There were 968 hypertensives and 1621 normotensives among 2589 subjects aged 20–84 years. There were 1437 non-smokers, 295 smokers who smoked 1–9 CPD, 478 smokers who smoked 10–19 CPD and 379 smokers who smoked ≥20 CPD.

Table I presents unadjusted and age–gender-adjusted baseline characteristics according to cigarette smoking status. After adjusted for age and gender, all current smokers were more likely to have higher rate of alcohol drinking and lower rates of overweight and central obesity compared with non-smokers, whereas smokers who smoked 10–19 and ≥20 CPD were more likely to have higher rate of alcohol drinking compared with smokers with 1–9 CPD, and smokers who smoked ≥20 CPD were
Table I. Unadjusted and age–gender-adjusted baseline characteristics according to cigarette smoking status.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-smoker</th>
<th>1–9 CPD</th>
<th>10–19 CPD</th>
<th>≥20CPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crude</td>
<td>Adjusted</td>
<td>Crude</td>
<td>Adjusted</td>
</tr>
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<td></td>
<td>Crude</td>
<td>Adjusted</td>
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<td></td>
<td>Crude</td>
<td>Adjusted</td>
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<td>Adjusted</td>
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<td></td>
<td>Crude</td>
<td>Adjusted</td>
<td>Crude</td>
<td>Adjusted</td>
</tr>
<tr>
<td>-family history of hypertension, % (95% CI)</td>
<td>11.0 (9.4–12.7)</td>
<td>13.7 (11.9–15.6)</td>
<td>11.9 (8.4–16.1)</td>
<td>11.7 (9.2–14.1)</td>
</tr>
<tr>
<td>Alcohol drinker, % (95% CI)</td>
<td>17.5 (15.5–19.5)</td>
<td>24.1 (22.1–26.0)</td>
<td>41.4a (35.7–47.2)</td>
<td>34.8a (31.1–38.5)</td>
</tr>
<tr>
<td>Central obesity, % (95% CI)</td>
<td>25.1 (22.9–27.4)</td>
<td>24.2 (22.1–26.4)</td>
<td>14.3a (10.5–18.8)</td>
<td>12.8a (9.3–16.2)</td>
</tr>
<tr>
<td>FBG, mmol/l, % (95% CI)</td>
<td>9.7 (8.2–11.4)</td>
<td>9.9 (8.3–11.4)</td>
<td>8.2 (5.3–12.0)</td>
<td>6.9 (4.9–8.8)</td>
</tr>
<tr>
<td>TC, mmol/l, % (95% CI)</td>
<td>14.6 (12.8–16.5)</td>
<td>17.2 (15.3–19.2)</td>
<td>16.8 (12.7–21.6)</td>
<td>16.1 (12.7–19.6)</td>
</tr>
<tr>
<td>Hypertensive, % (95% CI)</td>
<td>34.9 (32.4–37.4)</td>
<td>39.7 (37.3–42.1)</td>
<td>34.9 (29.7–40.5)</td>
<td>29.6a (26.2–33.1)</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>129±25</td>
<td>131±25</td>
<td>130±25</td>
<td>128±21</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>84±13</td>
<td>85±12</td>
<td>84±11</td>
<td>83±12a</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>77±11</td>
<td>77±12</td>
<td>76±12</td>
<td>76±11</td>
</tr>
</tbody>
</table>

*aCompared with non-smokers, p<0.05. bCompared with light smokers, p<0.05. cCompared with moderate smokers, p<0.05. CPD, cigarettes per day; CI, confidence interval; BMI, body mass index; FBG, fasting blood glucose; TG, triglycerides; TC, total cholesterol; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure.
more likely to have higher rates of overweight and central obesity compared with smokers with 10−19 CPD. Smokers who smoked 10–19 CPD had a lower rate of hyperglycemia compared with non-smokers and smokers who smoked ≥20 CPD; smokers with 10–19 CPD were more likely to have a rate of hyperLDL-cholesterolemia compared with non-smokers. Both smokers with 1–9 and 10–19 CPD were more likely to have lower prevalence of hypertension compared with non-smokers, but there was no significant difference between smokers who smoked ≥20 and non-smokers.

Means of DBP were lower in smokers who smoked 1–9 and 10–19 CPD than those in non-smokers, and means of SBP were lower in smokers who smoked 10–19 CPD than in non-smokers and the smokers who smoked ≥20, whereas there was no statistically significant difference between the smokers who smoked ≥20 and non-smokers. There was no significant difference in heart rate between non-smokers and current smokers; however, the smokers who smoked ≥20 had higher heart rates than the smokers who smoked 1–9 and 10–19 CPD.

Multivariate adjusted mean levels of SBP and DBP by the amounts of smoking are presented in Table II. The results appeared to show that multivariate adjusted mean levels of DBP increased with amounts of smoking, but the linear trends were not statistically significant. The ORs of hypertension associated with amounts of smoking and the linear trends were also not statistically significant.

More likely to have higher rates of overweight and central obesity compared with smokers with 10–19 CPD. Smokers who smoked 10–19 CPD had a lower rate of hyperglycemia compared with non-smokers and smokers who smoked ≥20 CPD; smokers with 10–19 CPD were more likely to have a rate of hyperLDL-cholesterolemia compared with non-smokers. Both smokers with 1–9 and 10–19 CPD were more likely to have lower prevalence of hypertension compared with non-smokers, but there was no significant difference between smokers who smoked ≥20 and non-smokers.

Multivariate adjusted means of SBP and DBP stratified by BMI according to smoking status are presented in Table III. In subjects with BMI<25, adjusted mean SBP levels appeared to have a curvilinear relation with amount of smoking; it was lower in subjects who smoked 10–19 CPD. The adjusted means of DBP were significantly lower in all smokers with various CPD than that in non-smokers, and the linear trend of DBP decreasing with amount of smoking was statistically significant. In subjects with BMI≥25, the mean of SBP was lower in the smokers who smoked ≥20 CPD than that in non-smokers, the mean of SBP was decreased with amount of smoking and the linear trend was statistically significant; however, the linear trend of DBP decreasing with amount of smoking was not statistically significant.

Multivariate adjusted ORs of hypertension associated with smoking status by BMI are presented in Table IV. In subjects with BMI<25, the multivariate adjusted ORs of hypertension were not statistically significant for the smokers with various amounts of smoking and the linear trend was also not statistically significant. In subjects with BMI≥25, the multivariate adjusted ORs of hypertension appeared to be decreased with amount of smoking, but the linear trend was not statistically significant.

Discussion
In this cross-sectional study among Mongolian population, we did not find that smoking was positively and
Table IV. Multivariate adjusted odds ratios (ORs) and 95% confidence intervals (CIs) of hypertension for smoking stratified by body mass index (BMI).

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>BMI&lt;25</th>
<th></th>
<th>BMI≥25</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>OR</td>
<td>95% CI</td>
<td>p</td>
</tr>
<tr>
<td>Non-smoker</td>
<td>1076</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1–9 CPD</td>
<td>252</td>
<td>0.72</td>
<td>0.51–1.02</td>
<td>0.066</td>
</tr>
<tr>
<td>10–19 CPD</td>
<td>421</td>
<td>0.87</td>
<td>0.65–1.17</td>
<td>0.361</td>
</tr>
<tr>
<td>≥20 CPD</td>
<td>315</td>
<td>0.79</td>
<td>0.57–1.10</td>
<td>0.163</td>
</tr>
<tr>
<td>p-value for linear test</td>
<td>0.36</td>
<td></td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>p-value for quadratic test</td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Multivariable adjustment included age, gender, family history of hypertension, alcohol drinking, waist circumference, fasting blood glucose, triglycerides, total cholesterol, low-density lipoprotein-cholesterol, heart rate.

significantly associated with SBP and DBP levels after adjusted for co-variables. However, we found that smokers who smoked 10–19 and ≥20 CPD had lower multivariate adjusted BMI among subjects with BMI≥25, and all smokers had lower multivariate adjusted DBP among subjects with BMI<25, compared with non-smokers. In addition, adjusted OR of hypertension associated with higher levels of smoking were not significant. These data did not provide support that smoking was a risk factor of hypertension. In fact, many early epidemiological studies have reported that BP is lower in smokers than that in non-smokers (7,8,18–20). A study reported that BP rose after smoking cessation (21), whereas others reported that smoking cessation had no obvious effect on BP (22,23). In 1990, Okubo and colleagues (24) conducted a cross-sectional study regarding relationship between smoking habits and BP level among 2781 Japanese men aged 40–50 years; their results showed that there was no significant difference in the adjusted SBP and DBP between non-smokers and ex-smokers, and the adjusted SBP and DBP in light, moderate and heavy smokers were significantly lower than in non- and ex-smokers. Recently, Gu et al. (25) published their findings regarding the relation of smoking with deaths in China; they conducted a large prospective cohort study in a nationally representative sample of 169,871 Chinese adults who were 40 years or older from 1991 to 2000. Their baseline data showed that male smokers had lower prevalence rate of hypertension than male non-smokers. There were also some prospective studies (8,26,27) showing that there is no significant positive association or there is a negative association between smoking and BP. On basis of a cross-sectional study, Okubo et al. (8) followed the male steel workers for 5 years, and analyzed the relationship between changes in smoking and BP; their study showed that chronic smoking reduced changes in BP and in 5-year cumulative incidence of hypertension after adjusting for some confounding factors. After following 4489 normotensives for 3 years, Nagahama and colleagues (26) found that habitual smoking had a significant negative effect on the incidence of hypertension in men after adjustment for some risk factors. Wang et al. (27) follow 4549 American men and women aged 45–74 years; their findings showed that smoking was significantly and negatively related to both SBP and DBP.

However, some studies (6,28–29) suggested that smoking was positively associated with hypertension. Bowman et al. (6) found that multivariate adjusted hazard ratio (HR) of hypertension among women who smoked more than 15 and 25 CPD were 1.11 (95% confidence interval, CI 1.03–1.21) and 2.95 (95% CI 1.06–1.39), respectively, after 9.8 years of follow-up. In a large worker cohort study in Taiwan (28), overall age-adjusted relative risk (RR) of hypertension for smokers was 1.23 (1.14–1.32). Halperin et al. (29) reported that adjusted RRs of hypertension were 1.08 and 1.15 for past smokers and current smokers, respectively, compared with never smokers, in a cohort of 15,529 male participants. Recently, Doshi and colleagues (30) also reported a positive association of smoking with BP in a cohort including 8251 male Japanese workers in a steel company. These findings implied that the relation of BP with smoking was a complex issue, because smoking itself was a social behavior associated with many other factors. Hence, we believed that a special design was needed to explore the association of smoking with hypertension fully excluding the confounding of other factors. Our findings did not support the association of smoking with hypertension or elevated BP. Although it was a cross-sectional study, we believed that our findings could show an objective association of hypertension with smoking, since the study was conducted in a Mongolian population with stable living conditions.

The fact that smokers in our study had lower BP compared with non-smokers could be related to the fact that smoking reduced body weight. For example, Primatesta et al.’s study (31) showed that light smokers (1–9 cigarettes/day) tended to have lower BP than never smokers among women, and current smokers had significantly lower BMI than never smokers. Moreover, some studies showed that body weight increased after giving up smoking (32–34), and the extent for increase of BMI with years was lower in smokers than that in non-smokers (35). In our study, age–gender-adjusted prevalence of overweight and central obesity was lower in smokers than that in non-smokers.
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Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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