Obesity and Smoking: Relationship with Waist Circumference and Obesity-Related Disorders in Men Undergoing a Health Screening

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This study investigated whether smoking habits had a differential influence on waist circumference and obesity-related disorders in nonobese and obese men. We investigated 359 men with smoking habits confirmed by their spouses, including 172 nonobese men (BMI < 25) and 187 obese men (BMI ≥ 25). There were 113 nonobese smokers and 129 obese smokers. Obesity-related disorders were defined as hypertension, dyslipidemia, hyperglycemia, hyperuricemia, or treatment for one or more of these disorders. Nonobese subjects showed no differences of age, BMI, and waist circumference between smokers and nonsmokers, but smokers had a higher incidence of obesity-related disorders. Obese smokers were younger than obese nonsmokers and had a larger waist circumference, but a similar prevalence of obesity-related disorders. The prevalence of obesity-related disorders was similar between obese nonsmokers and smokers, but the smokers were younger. In nonobese subjects, smoking may increase obesity-related disorders by a mechanism other than visceral fat accumulation. In obese subjects, however, smoking may promote visceral fat accumulation. Further investigations will be necessary to better elucidate the relationship between the promotion of visceral fat accumulation in obese subjects by smoking and obesity-related disorders. J Atheroscler Thromb, 2005; 12: 199–204.

Key words: Smoking habits, Metabolic disorders, Hypertension, Visceral fat accumulation

Introduction

It is well recognized that obesity is complicated by metabolic disorders such as hyperlipidemia or diabetes mellitus as well as by hypertension, and complications are reported to increase along with the accumulation of visceral fat in the abdomen (1). Although it has been reported that smoking influences body mass index (BMI) (2) and arteriosclerotic diseases (3), as well as blood pressure (4, 5), lipid metabolism (5, 6) and glucose metabolism (7, 8), it has not been clarified whether there is a difference in the prevalence of hypertension or metabolic disorders between nonobese and obese persons based on the presence or absence of smoking.

Various methods, such as inquiry or the measurement of cotinine (a metabolite of nicotine), have been used to assess smoking habits in investigations about smoking, but each method has some problems (9). In the present study, we investigated the relationship between the presence or absence of smoking and abnormalities of BMI, waist circumference, blood pressure, lipid metabolism, glucose metabolism, and uric acid metabolism in men who underwent a comprehensive health screening and whose smoking habits were confirmed by their spouses.

Subjects and Methods

Subjects
The subjects were 117 nonsmoking men, who were confirmed by themselves and their spouses to never have...
smoked and 242 smoking men whose smoking habits were confirmed by themselves and their spouses. None of them had a history of malignancy, gastrointestinal disorders, or endocrinological disorders and they all underwent a health screening together with their spouses. They were enrolled after being given an explanation concerning the objectives of this study, especially the importance of providing accurate information about smoking habits, and all of them gave informed consent.

**Methods**

During the health screening, height and weight were measured with digital equipment (Tanita, Tokyo, Japan) and BMI was calculated as weight/height² (kg/m²). There were 172 nonobese men with a BMI < 25 and 187 obese men with a BMI \( \geq 25 \). The waist circumference was measured at the level of the umbilicus while standing in expiration. Systolic and diastolic blood pressures were measured at rest in the sitting position with an automatic electronic sphygmomanometer (Nihon Seimitsusokki Co., Gunma, Japan). A blood sample was collected after fasting from the previous evening and the total cholesterol (TC), triglycerides (TG), HDL-cholesterol (HDL-C), fasting plasma glucose (FPG), hemoglobin A₁c (HbA₁c), and uric acid (UA) levels were measured.

The subjects were defined as having obesity-related disorders if they met one or more of the following criteria: systolic blood pressure \( \geq 140 \) mmHg and/or diastolic blood pressure \( \geq 90 \) mmHg, TC \( \geq 220 \) mg/dl, TG \( \geq 150 \) mg/dl, HDL-C < 40 mg/dl, FPG \( \geq 110 \) mg/dl, HbA₁c \( \geq 5.8 \% \), UA \( \geq 7.0 \) mg/dl, or current treatment for one or more of hypertension, dyslipidemia, diabetes mellitus, and hyperuricemia.

**Assay methods**

Using blood samples collected after an overnight fast, TC, TG, HDL-C, and UA levels were measured by conducting enzymatic assays (Olympus autoanalyzer, Tokyo, Japan). The blood glucose concentration was measured by the glucose oxidase method (Arkray, Kyoto, Japan) and HbA₁c was measured by high performance liquid chromatography (Arkay, Kyoto, Japan).

**Statistical analysis**

Numerical values are shown as the mean ± SD. The t-test was used to assess differences between mean values and the chi-square test was used for percentages. In both cases, \( p < 0.05 \) was considered to indicate statistical significance.

**Results**

Smokers accounted for 113/172 nonobese subjects and 129/187 obese subjects, and there was no difference in the prevalence of smoking between the nonobese and obese groups (65.7% vs. 69.0%). Table 1 shows the presence or absence of smoking and obesity-related disor-
Table 2. Age, body mass index, waist circumference, and incidence of obesity-related disorders stratified by the presence or absence of smoking in nonobese and obese subjects.

<table>
<thead>
<tr>
<th></th>
<th>Nonobese</th>
<th></th>
<th>Obese</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nonsmokers</td>
<td>Smokers</td>
<td>Nonsmokers</td>
<td>Smokers</td>
</tr>
<tr>
<td></td>
<td>(59)</td>
<td>(113)</td>
<td>(58)</td>
<td>(129)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>49.7 ± 10.3</td>
<td>50.0 ± 7.8</td>
<td>51.9 ± 8.2</td>
<td>47.8 ± 8.3</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>22.3 ± 1.5</td>
<td>22.5 ± 1.8</td>
<td>26.8 ± 1.6</td>
<td>27.4 ± 2.1</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>78.8 ± 5.4</td>
<td>79.9 ± 5.8</td>
<td>89.9 ± 4.8</td>
<td>91.7 ± 6.0</td>
</tr>
<tr>
<td>Incidence of obesity-related disorders (%)</td>
<td>49.2</td>
<td>75.2</td>
<td>77.6</td>
<td>87.6</td>
</tr>
</tbody>
</table>

Nonobese subjects: body mass index < 25, obese subjects: body mass index ≥ 25
Values are the mean ± SD
The numbers in parentheses indicate the number of subjects
*: p < 0.05, **: p < 0.01, †: p < 0.025, ††: p < 0.005

Fig. 1. Age distribution of the nonobese and obese subjects classified by the presence or absence of smoking and the prevalence of obesity-related disorders.
Nonobese subjects: body mass index < 25, obese subjects: body mass index ≥ 25
NS: nonsmokers, S: smokers
Mean ± SD
*: p < 0.05, **: p < 0.01, ††: p < 0.001
Smoking is known to influence blood pressure, lipid metabolism, and glucose metabolism. It has been reported that the daytime blood pressure and average 24 hour blood pressure are higher on smoking days than on nonsmoking days (4). After the same fat load was given to smoking and nonsmoking subjects with normal fasting TG levels, serum TG increased significantly, while HDL-C decreased significantly in the smoking subjects (6). Following a glucose load, the blood glucose level increased significantly after smoking in smoking subjects, and insulin and C-peptide levels also increased significantly, while the blood glucose level increased only in nonsmokers.

### Discussion

The subjects were classified into 8 groups based on the presence or absence of obesity, smoking, and obesity-related disorders. The ages of these 8 groups are shown in Fig. 1. Obese smokers had obesity-related disorders at a younger age compared with nonobese smokers (48.3 ± 8.0 vs. 50.2 ± 7.8 years; \( p < 0.001 \)). In addition, the obese smokers with obesity-related disorders were younger than the obese nonsmokers with obesity-related disorders (48.3 ± 8.0 vs. 52.3 ± 8.3 years; \( p < 0.01 \)), while among obese subjects without obesity-related disorders, there was no difference in age between smokers and nonsmokers.

As shown in Table 3, there were no differences in BMI among the nonobese subjects when they were classified into 4 groups. When the obese subjects were classified similarly, again there were no differences in BMI among the groups. When each group of nonobese subjects was compared with each group of obese subjects (Table 3), the obese subjects always showed a higher BMI (\( p < 0.01 \)).

The waist circumferences of 8 groups classified by the presence or absence of obesity, smoking, and obesity-related disorders are shown in Fig. 2. When the waist circumference was compared between nonobese subjects with or without obesity-related disorders, it was found to be larger in smokers with obesity-related disorders (80.1 ± 5.8 cm) than in nonsmokers without obesity-related disorders (76.5 ± 4.8 cm) (\( p < 0.01 \)), whereas it was larger in nonsmokers with obesity-related disorders (81.2 ± 5.1 cm) than in smokers without obesity-related disorders (77.7 ± 5.6 cm) (\( p < 0.02 \)). Among obese subjects, the waist circumference of smokers with obesity-related disorders (92.1 ± 6.1 cm) was larger than that of nonsmokers without obesity-related disorders (87.5 ± 3.7 cm) (\( p < 0.01 \)). In addition, the waist circumference for each group of obese subjects was larger than that for each group of nonobese subjects (\( p < 0.01 \)).

### Table 3. Body mass index of the nonobese and obese subjects classified by the presence or absence of smoking and the prevalence of obesity-related disorders.

<table>
<thead>
<tr>
<th></th>
<th>Obesity-related disorders (−)</th>
<th>Obesity-related disorders (+)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Nonobese (172)</td>
<td>Obese (187)</td>
</tr>
<tr>
<td></td>
<td>Nonsmokers</td>
<td>Smokers</td>
</tr>
<tr>
<td></td>
<td>21.8 ± 1.6 (30)</td>
<td>22.8 ± 1.2 (29)</td>
</tr>
<tr>
<td></td>
<td>21.6 ± 1.9 (28)</td>
<td>22.8 ± 1.6 (85)</td>
</tr>
</tbody>
</table>

Nonobese subjects: body mass index < 25, obese subjects: body mass index ≥ 25
Values are the mean ± SD
The numbers in parentheses indicate the number of subjects
The body mass index of each group of nonobese subjects and each group of obese subjects was significantly different (\( p < 0.01 \)).
Fig. 2. Waist circumference of the nonobese and obese subjects classified by the presence or absence of smoking and the prevalence of obesity-related disorders. Nonobese subjects: body mass index < 25, obese subjects: body mass index ≥ 25
NS: nonsmokers, S: smokers
Mean ± SD
**: p < 0.02, ***: p < 0.01

The waist circumference of each group of nonobese subjects was significantly smaller than that of each group of obese subjects (p < 0.01).

slightly after smoking in nonsmoking subjects (8).
These investigations were performed in a setting where the smoking status could be confirmed. However, investigations concerning smoking are associated with some problems, e.g., subjects sometimes do not answer correctly about their smoking habits (9, 10). Although measurements of cotinine, a metabolite of nicotine, can be used (9), cotinine is rarely assessed in health screening. We attempted to increase the accuracy of the information obtained by inquiry by selecting men who attended the screening with their spouses if the answers of the couple coincided about the man’s smoking habits.

In the nonobese subjects, there was no difference of age, BMI, and waist circumference irrespective of smoking habits, but obesity-related disorders were more common in the smokers than the nonsmokers. The nonobese smokers showed a lower BMI and smaller waist circumference than the obese nonsmokers, but there was no difference in the incidence of obesity-related disorders. When nonobese subjects with obesity-related disorders were assessed, the waist circumference of nonsmokers was found to be larger than that of smokers.

Although there were no differences in BMI and the incidence of obesity-related disorders between the obese nonsmokers and smokers, the waist circumference was larger at a younger age in the smokers. Accordingly, the waist circumference of obese smokers in their late forties exceeded that of nonsmokers in their early fifties.

Waist circumference was selected as a useful index to estimate visceral fat volume, since it has been reported that there is a strong correlation between the area of visceral fat and the waist circumference (11). There was no constant relationship between the BMI or waist circumference and obesity-related disorders in nonobese subjects regardless of whether or not they were smokers, suggesting that smoking increases the incidence of obesity-related disorders by a mechanism other than visceral fat accumulation. In our obese subjects, obesity already existed to promote obesity-related disorders, and smoking seemed to accelerate visceral fat accumulation. A strong correlationship is reported to exist between the waist circumference or visceral fat area and the incidence of obesity-related disorders (11, 12). It is necessary to further investigate the relationship between obesity-related disorders and the promotion of visceral fat accumulation in obese subjects by smoking that was suggested in this study.

There is a possibility that smoking is involved in pro-
moting obesity-related disorders via different mechanisms in nonobese and obese men. It is also possible that smoking has a relationship with other factors, such as drinking alcohol, exercise, diet, and interest in health. In the future, it will be important to accurately assess the nature of the influence of smoking on health in nonobese and obese men and to further investigate factors other than smoking in more detail.

References


