Association Between Cigarette Smoking and Metabolic Syndrome

The Korea National Health and Nutrition Examination Survey

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Metabolic syndrome is highly prevalent in the U.S. (age-adjusted prevalence of 23.7%) (1) and in Korea (20.8% for men and 26.9% for women) (2). This syndrome is well documented to increase the risk for developing type 2 diabetes and cardiovascular diseases (CVDs) and is associated with an all-cause mortality (3–5). Physical inactivity, excessive weight gain, high alcohol intake, and certain dietary factors have been identified as important modifiable risk factors for metabolic syndrome and its consequences (2,6,7).

Smoking is also a strong risk factor for atherosclerosis and CVD, with a dose-dependent relationship (8). Smokers have abnormalities in lipoprotein metabolism (9) and endothelial function (10). Moreover, there is some evidence that smokers are at greater risk than nonsmokers of becoming insulin resistant and hyperinsulinemic (11,12). Thus, based on these findings, smoking may be considered as an important modifiable risk factor for metabolic syndrome. However, this relationship has not been clarified, and reports on this issue are also scarce. The aim of this study was to search for associations between smoking and metabolic syndrome with a representative population-based sample in Korea.

RESEARCH DESIGN AND METHODS — This study was based on the 1998 Korea National Health and Nutrition Examination Survey. Selection methods for a nationwide representative sample of Koreans and other survey methods were detailed elsewhere (2). Complete data were obtained for 9,771 of 10,876 individuals (89.8%) who participated in the Health Examination Study. We restricted our analyses to men because the proportion of Korean women who smoked was too small (8.3%) to allow detailed analyses (2). Finally, data from 3,452 men ≥20 years of age formed the basis of this analysis.

The surveys included questions about age, education level, occupation, income, marital status, smoking habits, alcohol consumption, exercise, previous and current diseases, and family disease history, among others. The nutrition survey employed in this study assessed daily energy and nutrient intakes by using 24-h recall and a food-intake frequency method.

Past smokers were defined as those who had abstained from smoking for >3 months at the time of examination. Information on the daily smoking habits of current smokers was obtained from questions about the average number of cigarettes smoked daily during the month before the interview. Because participants often reported their current smoking amounts by the unit of pack (20 cigarettes), five categories were established: <10, 10–19, 20–29, 30–39, and ≥40 cigarettes/day. The total number of pack-years of cigarettes smoked was also calculated from the total number of years spent smoking multiplied by the number of packs smoked daily. Total pack-years were divided into quartiles for the analyses.

The 2001 definition of metabolic syndrome provided by the National Cholesterol Education Program Adult Treatment Panel III was used (13). However, because Asians have greater risk of fitting the metabolic profile at lower waist circumferences than Caucasians (14), we used the abdominal obesity guidelines for waist circumference (>90 cm for men and >80 cm for women) suggested by the 1998 World Health Organization Asian Pacific Guideline (14). Multiple logistic regression was used to calculate odds ratios (ORs). After several models were tested using a hierarchical approach, a model adjusted for age, education level, exercise, alcohol consumption, weight change during the past year, and amount of dietary fat and fiber consumed was constructed. The linear trend in the OR was evaluated using the trend test.

RESULTS — Table 1 shows that current smoking amounts had a statistically significant dose-dependent association with metabolic syndrome ($P = 0.023$ for trend). In the analyses of the individual components of metabolic syndrome, high triglycerides and low HDL cholesterol showed statistically significant dose-dependent associations. When the analyses were confined to current smokers, there was a positive dose-dependent asso-

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Abbreviations: CVD, cardiovascular disease.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Adjusted ORs of metabolic syndrome and its components according to current smoking amounts*

	Nil	Ex-smoker	Cigarettes/day					*P* for trend
			1–9	10–19	20–29	30–39	≥40	
Abdominal obesity	1	1.01 (0.75–1.37)	0.70 (0.43–1.14)	0.71 (0.52–0.96)	1.03 (0.78–1.36)	1.25 (0.76–2.07)	1.54 (0.92–2.56)	0.465
High blood pressure	1	1.13 (0.88–1.47)	0.92 (0.63–1.35)	1.02 (0.80–1.30)	1.02 (0.81–1.29)	1.25 (0.81–1.94)	1.33 (0.83–2.12)	0.605
High triglycerides	1	1.37 (1.06–1.78)	1.05 (0.72–1.54)	1.00 (0.78–1.29)	1.46 (1.15–1.85)	1.42 (0.91–2.19)	1.62 (1.01–2.58)	0.014
Low HDL cholesterol	1	1.00 (0.75–1.33)	1.50 (1.01–2.23)	0.99 (0.75–1.30)	1.44 (1.11–1.86)	2.29 (1.44–3.65)	1.28 (0.74–2.20)	0.001
High fasting glucose	1	1.09 (0.81–1.47)	1.03 (0.66–1.61)	1.11 (0.83–1.48)	1.17 (0.89–1.54)	1.02 (0.60–1.73)	1.25 (0.73–2.14)	0.290
Metabolic syndrome	1	1.09 (0.81–1.48)	1.07 (0.68–1.68)	0.91 (0.67–1.23)	1.24 (0.94–1.64)	1.79 (1.10–2.91)	1.77 (1.06–2.96)	0.023

Data are ORs (95% CI) unless otherwise indicated. *P* values were calculated after adjustment for age (continuous), education level (none, elementary, middle or high school, and college or higher), exercise (none, sometimes, regularly), alcohol consumption (none, <15, 15–29, ≥30 g/day), weight change during the past year (continuous), and amount of dietary fat and fiber (continuous).

ciation between current smoking amount and abdominal obesity ($P = 0.002$ for trend, not shown in Table 1).

Total pack-years of cigarettes smoked during a subject's lifetime also showed significant dose-dependent associations with metabolic syndrome ($P = 0.040$ for trend, not shown in Table 1). Similar to the results of current smoking amounts, high triglycerides and low HDL cholesterol had a significant dose-dependent association with total pack-years. We could also find significant dose-dependent associations across the quartiles of total pack-years after exclusion of never smokers ($P = 0.003$ for trend, not shown in Table 1).

CONCLUSIONS

— Of the components of metabolic syndrome, high triglycerides, low HDL cholesterol, and abdominal obesity are thought to be the main contributors to the association. In contrast to our expectations, we could not find associations of smoking with high fasting glucose and high blood pressure. There have been inconsistent epidemiologic findings from previous studies on this issue. However, recent well-established evidence supports the associations of smoking with hypertension (15,16) and type 2 diabetes (17,18). We suppose that the cross-sectional design of this study limited its ability to detect these associations.

When the analyses were restricted to smokers, we found that an increased amount of smoking tended to be positively associated with abdominal obesity. This finding is somewhat contrary to the general concept that smokers have lower anthropometric indexes than never-smokers (19). However, results from other recent studies showed that increased smoking amounts can contribute to abdominal obesity (20–22); this is consistent with our findings. Although a precise mechanism explaining the association remains to be elucidated, we did find that increased smoking amounts are associated with abdominal obesity and can aggravate metabolic syndrome in smokers.

The strengths of this study were that the study subjects were a representative population-based sample of Koreans and that the analyses were conducted with adjustments for various possible confounders. However, our study has some limitations. First, we could not demonstrate this association for women because of their lower smoking rate. Second, the cross-sectional design of this study prohibited us from concluding causal relationships and may have included some biases.

In conclusion, these population-based data support the hypothesis that cigarette smoking is independently associated with metabolic syndrome. However, further studies are needed to elucidate the underlying mechanisms and the causal effects of smoking on metabolic syndrome.

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