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CARDIOVASCULAR DISEASE AND DIABETES

The metabolic syndrome and associated lifestyle factors among South Korean adults

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Introduction The importance of managing the metabolic syndrome has been emphasized in the prevention of cardiovascular disease. Although South Koreans have a low prevalence of obesity, little information is available about the prevalence of the metabolic syndrome and its associated risk factors.

Methods Data was obtained from individuals aged 20–79 years from the Korean National Health and Nutrition Examination Survey 1998, a cross-sectional health survey of a nationally representative sample of non-institutionalized civilian South Koreans. The prevalence of the metabolic syndrome, as defined by the Third Report of the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP III), was determined, and lifestyle factors associated with predisposition to the metabolic syndrome were analysed.

Results Among South Korean adults, the age-adjusted prevalence of the metabolic syndrome was 14.2% for men and 17.7% for women, whereas the age-adjusted prevalence of obesity (body mass index (BMI) $\geqslant$30 kg/m^2) was 1.7% and 3.0% for men and women, respectively. Age, unemployment, higher BMI, and current smoking were associated factors for the metabolic syndrome regardless of gender. Moderate exercise (2–3 sessions/week) in men and light alcohol drinking (<15 g/day) in women decreased the odds of the metabolic syndrome.

Conclusions Metabolic syndrome is present in more than 15% of South Koreans despite a low prevalence of obesity. Higher BMI and current smoking were identified as independent modifiable risk factors of the metabolic syndrome. Weight control and smoking cessation may therefore decrease the prevalence of the metabolic syndrome in South Korean adults.

Keywords Metabolic syndrome, risk factor, BMI, smoking, South Korea

Cardiovascular mortality is markedly increased in subjects with the metabolic syndrome.[1] In the US, the unadjusted and age-adjusted prevalence of the metabolic syndrome have been estimated to be 21.8% and 23.7%, respectively.[2] The Third Report of the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP III) has recommended appropriate measures to identify individuals with the metabolic syndrome and to manage their care prior to development of cardiovascular complications.[3] For example, abdominal obesity has been found to promote insulin resistance, which further predisposes individuals to type 2 diabetes[4–6] and contributes to an increase in cardiovascular risk factors.[7,8]

Over the past several decades, South Korea has experienced rapid socioeconomic growth, resulting in lifestyle changes that have promoted the development of components of the metabolic syndrome within the population. For example, consumption of a contemporary high-fat diet, consisting of meat, fast food, and processed foods, has increased, such that, between 1969 and 1998, the fat intake of South Koreans increased threefold, from 7.2% to 19.0% of total energy consumption.[9] In addition, industrialization and the growth of the technology

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sector have led to a decrease in physical activity. Cardiovascular disease is therefore becoming more prevalent in South Korea and is now a leading cause of death, while stroke is currently ranked second as a cause of mortality in Korean men and women.[10] Compared with Caucasians, Asians generally tend to have a higher prevalence of cardiovascular diseases at lower body mass index (BMI), which may be due to the propensity of Asians to have central obesity and increased incidence of diabetes.[8]

Using BMI as a measure of obesity, it has been estimated that 19.5% of men and 25.0% of women in Western countries are obese, and that there is a correlation between the prevalence of obesity and the metabolic syndrome.[11] The resident population of South Korea has a lower average BMI than do residents of Western countries, suggesting that the prevalence of the metabolic syndrome may also be lower. Although modification of lifestyle-associated risk factors is known to be essential for preventing and managing the metabolic syndrome, there is currently insufficient information available on the prevalence of the metabolic syndrome and associated factors in Asian countries. We therefore investigated the prevalence of the metabolic syndrome and lifestyle factors contributing to the development of this syndrome in South Korean adults.

Methods

Study population

The Korean Ministry of Health and Welfare conducted the Korean National Health and Nutrition Examination Survey in non-institutionalized Korean civilians in 1998. A stratified multistage probability sampling design was used, with selection made from sampling units based on geographical area, sex, and age groups using household registries. Weights indicating the probability of being sampled were assigned to each respondent, enabling the results to represent the entire Korean population. The staff conducted surveys in households, administering questionnaires to adults and children. Household surveys included the demographic, socioeconomic, dietary, and medical history of each respondent.

A total of 10 876 people were identified to take part in the survey. Of these, 1105 individuals either had missing anthropometric measurements or did not fast properly prior to the blood test. Complete data were obtained for the remaining 9771 participants (89.8%). The mean age of the 9771 individuals (37.1 years for men, 38.1 years for women) was similar to that of the initial 10 876 individuals recruited for the survey (36.4 years for men, 38.1 years for men). Data from subjects <20 (n = 1809) and >80 (n = 97) years of age were excluded, leaving data from 7865 respondents available for analysis. Since the survey utilized a stratified multistage probability sampling method, a weight variable was generated to back-calculate the original population distribution. Such use of weight allows unbiased point estimates of population parameters for the entire population and its subsets.[12] This weighting of 7865 observations resulted in 8650 subjects representative of the census population.

The characteristics of the study population, stratified by gender, are presented in Table 1. Of the study participants, 45.5% were men (n = 3937) and 54.5% were women (n = 4713). Accounting for all the participants, the mean age was 43 years, and the mean BMI was 23.2 kg/m^2. The percentage of total energy intake from fat was 15% in men and 16% in women. Almost half of all male and female respondents lived in large cities. More than 70% of subjects were married, and 17% of men and 32% of women had $\leqslant 6$ years of education. Additionally, 17% of men and 52.3% of women were unemployed. Approximately 10% of both men and women earn more than US$2000 per month, which is US$1000 more than the nationwide average monthly income.

Definition of the metabolic syndrome

We used the definition of the metabolic syndrome proposed by the NCEP ATP III[3] since it is easier to apply to the study population than the World Health Organization (WHO)[13] and other guidelines.[14–17] As detailed in the NCEP ATP III report,[3] participants having three or more of the five following criteria were defined as having the metabolic syndrome: high blood pressure ($\geqslant 130/\geqslant 85$ mmHg), elevated fasting blood glucose ($\geqslant 110$ mg/dl or $\geqslant 6.05$ mmol/l), hypertriglyceridaemia ($\geqslant 150$ mg/dl or $\geqslant 1.65$ mmol/l), low high density lipoprotein (HDL)-cholesterol (men, <40 mg/dl or <1.05 mmol/l; women, <50 mg/dl or <1.30 mmol/l), and abdominal obesity, as measured by a waist circumference of >102 cm for men and >88 cm for women. Participants receiving pharmacological treatment for hypertension (i.e. ACE inhibitors, angiotensin receptor blockers, α-blockers, β-blockers, calcium channel blockers, or diuretics) were included in the high blood pressure group (3.7% of participants), while participants receiving pharmacological treatment for diabetes (i.e. sulphonylurea, biguanide, α-glucosidase inhibitors, or insulin) were included in the high fasting blood glucose group (1.9% of participants).

Anthropometric measurements

Anthropometric measurements on individuals wearing light clothing and without shoes were conducted by well-trained examiners. Height was measured to the nearest 0.1 cm using the portable stadiometer (850–2060 mm; Seriter®). Weight was measured in the upright position to the nearest 0.1 kg using a calibrated balance beam scale (Giant-150N; HANA®). BMI was calculated by dividing weight (kg) by height squared (m^2). Waist circumference measurements were taken at the end of normal expiration to the nearest 0.1 cm, measuring from the narrowest point between the lower borders of the rib cage and the iliac crest.

Lifestyle factors

Dietary intake was assessed by the single 24-hour dietary recall method. All subjects were requested to maintain their usual diet before testing. Experienced interviewers instructed respondents to recall and describe the foods and beverages consumed over the previous 24 hours. Food models and measuring bowls, cups, and spoons were used to assist in estimating portion sizes. The record for each subject was coded, and standard reference tables were used to convert household portions to gram weights. Nutrient analysis of the records was quantified using a computerized program (CAN, Korean Nutrition Society, Seoul, Korea).[18] As one relevant measure of dietary composition,

Table 1 Characteristics of the study population, Korean National Health and Nutrition Examination Survey 1998

Variables		Men (n = 3937) Mean ± SD	Women (n = 4713) Mean ± SD
Age (year)		42.9 ± 14.7	43.2 ± 15.7
Body mass index (kg/m^2)		23.2 ± 3.1	23.2 ± 3.5
Waist circumference (cm)		82.9 ± 8.8	78.2 ± 10.2
Total calories (kcal)		2285 ± 1014	1816 ± 789
Carbohydrate (%)		63.7 ± 12.5	66.7 ± 10.7
Fat (%)		16.3 ± 8.6	15.5 ± 8.7
Protein (%)		15.1 ± 5.3	14.6 ± 5.5
		No (%)	No (%)
Residential area	Large city	1923 (48.8)	2295 (48.7)
	Medium-sized city	1201 (30.5)	1421 (30.2)
	Rural area	813 (20.7)	998 (21.2)
Marital status	Married	3082 (78.3)	3411 (72.4)
	Unmarried	728 (18.5)	578 (12.3)
	Others	127 (3.2)	725 (15.4)
Education level (years)	<7	668 (17.0)	1510 (32.0)
	7–9	526 (13.4)	687 (14.6)
	10–12	1564 (39.7)	1648 (35.0)
	>12	1179 (30.0)	869 (18.4)
Occupation	Professional, administrative	358 (9.1)	217 (4.6)
	Office worker	484 (12.3)	285 (6.0)
	Sales, service	681 (17.3)	738 (15.7)
	Agricultural, fishing	422 (10.7)	430 (9.1)
	Labourer	1121 (28.5)	474 (10.1)
	Others	202 (5.1)	106 (2.3)
	Unemployed	669 (17.0)	2463 (52.3)
Income (US$/month)	<500	797 (20.2)	1059 (22.5)
	500–1000	1216 (30.9)	1438 (30.5)
	1000–2000	1509 (38.3)	1708 (36.2)
	>2000	416 (10.6)	509 (10.8)
Alcohol (g/day)	None	894 (22.7)	2555 (54.2)
	<15	1335 (33.9)	1912 (40.6)
	15–30	616 (15.7)	155 (3.3)
	>30	1092 (27.7)	91 (1.9)
Smoking	None	659 (16.7)	4210 (89.3)
	Ex-smoker	620 (15.7)	113 (2.4)
	Current smoker	2659 (67.5)	391 (8.3)
Exercise (sessions/week)	None	2225 (58.9)	3417 (74.2)
	⩽1	952 (25.2)	689 (15.0)
	2–3	306 (8.1)	221 (4.8)
	⩾4	296 (7.8)	280 (6.1)

carbohydrate intake was selected[19,20] and expressed as a percentage of total energy. In this study, the percentage intake of carbohydrates was divided into quartiles (<45%, 45–60%, 60–70%, and ⩾70%).

Self-reported alcohol intake, smoking frequency, and physical exercise were estimated from the questionnaire. Alcohol consumption was estimated from the usual daily intake of alcoholic beverages. Drinkers were divided into four groups by the amount of alcohol consumed: none, <15 g/day, 15–30 g/day, and ⩾30 g/day. Regarding smoking, individuals were classified based on whether the respondent was a non-smoker, an ex-smoker, or a current smoker. Participants were also asked about the frequency and extent of physical activity on a weekly basis, including sports, physically active hobbies, and fitness exercises over the past 30 days. Physical exercise was defined as any activity causing light perspiration or a slight to moderate increase in breathing or heart rate for at least 30 minutes. Physical exercise was categorized into four groups: not at all, once per week, 2–3 sessions per week, or >4 sessions per week.

Cardiovascular risk factors

A mercury sphygmomanometer (Baumanometer®) was used to measure the blood pressure of each subject in the sitting position after a 10-minute rest period. During the 30 minutes preceding the measurement, subjects were required to refrain from smoking or consuming caffeine. The appearance of the first sound (phase 1 Korotkoff sound) was used to define systolic blood pressure and the disappearance of sound (phase 5 Korotkoff sound) was used to define diastolic blood pressure.[21] Two readings, each of systolic and diastolic blood pressure were recorded and the average of each measurement was used for data analysis. If the first two measurements differed by more than 5 mmHg, additional readings were obtained.

Blood samples were obtained from an antecubital vein into vacutainer tubes containing EDTA in the morning after a 12-hour, overnight fasting period. Samples were subsequently analysed at a central, certified laboratory. Plasma glucose, total cholesterol, triglyceride, and HDL-cholesterol were measured by an autoanalyser (Hitachi 747 auto-analyzer, Japan).

Statistical analysis

Sampling weights were used to take the complex sampling into account, and statistical analyses were conducted with the weighting using the statistical software, SAS version 8.1. Multiple logistic regression analysis was performed to examine the determinants of the metabolic syndrome among demographic, anthropometric, and lifestyle factors. Covariates included age, residential area, marital status, education level, occupation, economic status, BMI, carbohydrate intake, alcohol consumption, cigarette smoking, and physical exercise. The adjusted odds ratios (OR) are presented together with their 95% CI. Adjustments were carried out for all independent variables, including demographic, anthropometric, and lifestyle factors. The linear trend in odds was evaluated using the trend test. All analyses were two-tailed and a *P*-value < 0.05 was considered statistically significant.

Table 2 Age-adjusted prevalence of each component of the metabolic syndrome and clustering of components among Korean adults, Korean National Health and Nutrition Examination Survey 1998

	Men (n = 3937)	Women (n = 4713)
Variables	(%)	(%)
Abdominal obesity[a]	1.3	15.3
Blood pressure ⩾130/85 mmHg	44.7	29.9
Fasting blood glucose ⩾110 mg/dl	20.4	17.3
Triglycerides ⩾150 mg/dl	35.0	20.5
Low HDL-cholesterol[b]	24.5	45.8
⩾1 component	71.2	67.4
⩾2 components	37.7	35.9
⩾3 components	14.2	17.7
⩾4 components	2.6	6.5
5 components	0.1	1.4

[a] Abdominal obesity: waist circumference >102 cm for men, >88 cm for women.

[b] Low high density lipoprotein-cholesterol: <40 mg/dl for men, <50 mg/dl for women.

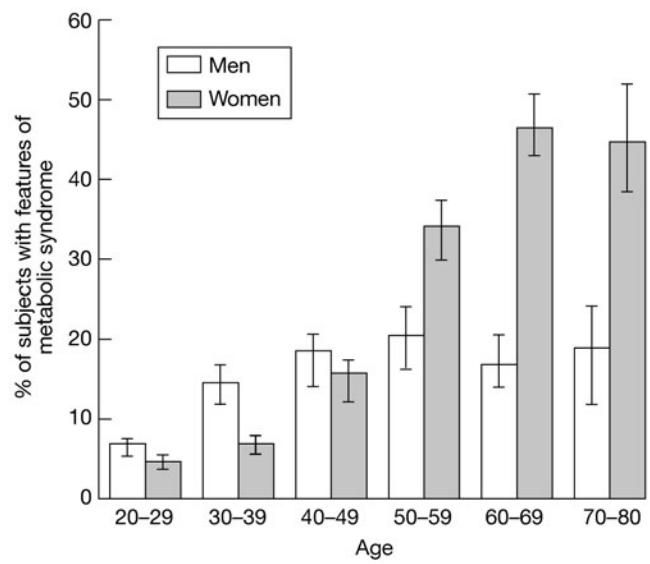

Figure 1 Age-specific prevalence of the metabolic syndrome among Korean adults by sex, Korean National Health and Nutrition Examination Survey 1998

Results

Prevalence of the metabolic syndrome

The unadjusted and age-adjusted prevalence of the metabolic syndrome were 15.2% and 14.2% in Korean men, and 19.1% and 17.7% in Korean women, respectively. The age-adjusted prevalence for each component of the metabolic syndrome and the clustering of metabolic components are shown in Table 2. Men had a higher prevalence of high blood pressure, high fasting blood glucose level, and high triglyceride content, whereas women had a higher prevalence of abdominal obesity, low HDL-cholesterol, and a clustering of three or more components of the metabolic syndrome.

Figure 1 shows the age-specific prevalence of the metabolic syndrome in the study population. Prevalence of the metabolic syndrome increased with age for both men and women. Under the age of 50, men had a higher prevalence than women, but this was reversed among the participants who were over 50. The prevalence of the metabolic syndrome in older women exceeded the prevalence in men by more than 100%.

Table 3 Adjusted odds ratios (OR) for the metabolic syndrome as a dependent variable and the associated factors as independent variables among Korean men (n = 3937)

Variables	Categories	% with metabolic syndrome	Adjusted OR (95% CI)	*P*-value
Age (years)[a]	20–29	6.7	1.0	
	30–39	14.4	2.1 (1.4, 3.2)	0.0003
	40–49	17.8	2.4 (1.6, 3.8)	<0.0001
	50–59	21.1	3.1 (1.9, 4.9)	<0.0001
	60–69	18.1	2.6 (1.6, 4.7)	0.0003
	⩾70	16.7	2.6 (1.4, 5.0)	0.0030
Residential area	Large city	15.4	1.0	
	Medium-sized city	13.8	0.9 (0.7, 1.2)	0.33
	Rural area	16.5	0.9 (0.7, 1.2)	0.61
Marital status	Married	16.9	1.0	
	Unmarried	8.1	0.9 (0.6, 1.4)	0.82
	Others	12.8	0.7 (0.4, 1.2)	0.16
Education level (years)	<7	18.8	1.0	
	7–9	18.0	0.9 (0.7, 1.3)	0.75
	10–12	14.5	0.8 (0.6, 1.1)	0.27
	>12	12.7	0.9 (0.6, 1.3)	0.49
Occupation[a]	Blue collar	12.7	1.0	
	White collar	14.6	1.2 (0.9, 1.6)	0.27
	Unemployed	20.3	2.0 (1.4, 2.8)	0.0002
Income (US$/month)	<500	17.9	1.0	
	500–1000	14.0	0.9 (0.7, 1.2)	0.38
	1000–2000	14.0	0.9 (0.7, 1.2)	0.49
	>2000	17.6	1.2 (0.8, 1.7)	0.46
Body mass index (kg/m^2)[a]	<18.5	8.6	1.0	
	18.5–22.9	7.8	1.0 (0.6, 1.8)	0.97
	23.0–24.9	17.4	2.6 (1.5, 4.6)	0.0012
	25.0–26.9	21.8	3.5 (1.9, 6.2)	<0.0001
	⩾27	35.9	7.5 (4.1, 13.6)	<0.0001
Carbohydrate (%)	<45	16.0	1.0	
	45–60	15.4	1.0 (0.7, 1.3)	0.92
	60–70	14.6	0.9 (0.7, 1.2)	0.51
	>70	14.4	0.9 (0.7, 1.1)	0.24
Alcohol (g/day)	None	16.3	1.0	
	<15	12.3	0.8 (0.7, 1.1)	0.21
	15–30	15.8	1.0 (0.7, 1.4)	0.95
	>30	17.3	1.0 (0.8, 1.3)	0.97
Smoking[a]	None	12.9	1.0	
	Ex-smoker	15.6	1.1 (0.8, 1.5)	0.55
	Current smoker	15.6	1.4 (1.1, 1.8)	0.0161
Exercise	None	15.8	1.0	
(sessions/week)	⩽1	14.6	1.0 (0.8, 1.3)	0.78
	2–3	10.9	0.6 (0.5, 0.9)	0.0332
	⩾4	17.5	0.9 (0.7, 1.3)	0.72

[a] $P < 0.05$ from trend test.

Associated factors of the metabolic syndrome

Tables 3 and 4 show the adjusted OR for associations between demographic, socioeconomic, anthropometric, and lifestyle factors and the risk of the metabolic syndrome in men and women, respectively. The relation with age remained after multivariate adjustment. The association between the metabolic syndrome and age increased gradually in men, whereas in women it increased abruptly in those over 50 years of age. The

Table 4 Adjusted odds ratios (OR) for the metabolic syndrome as a dependent variable and the associated factors as independent variables among Korean women (n = 4713)

Variables	Categories	% with metabolic syndrome	Adjusted OR (95% CI)	*P*-value
Age (years)[a]	20–29	4.5	1.0	
	30–39	6.7	1.5 (0.9, 2.4)	0.06
	40–49	15.5	3.4 (2.1, 5.3)	<0.0001
	50–59	33.6	7.8 (4.8, 12.6)	<0.0001
	60–69	47.2	15.3 (9.1, 25.7)	<0.0001
	⩾70	46.6	17.0 (9.6, 30.3)	<0.0001
Residential area	Large city	17.3	1.0	
	Medium sized city	16.1	0.8 (0.6, 1.1)	0.13
	Rural area	27.6	0.8 (0.6, 0.9)	0.03
Marital status	Married	17.5	1.0	
	Unmarried	6.2	2.0 (1.2, 3.3)	0.01
	Others	37.0	0.8 (0.7, 1.1)	0.14
Education level (years)	<7	36.1	1.0	
	7–9	20.4	0.9 (0.7, 1.2)	0.40
	10–12	9.6	0.9 (0.7, 1.3)	0.67
	>12	6.8	1.1 (0.7, 1.6)	0.79
Occupation[a]	Blue collar	5.5	1.0	
	White collar	19.3	1.3 (0.8, 2.2)	0.27
	Unemployed	21.8	1.7 (1.0, 2.7)	0.03
Income (US$/month)	<500	30.8	1.0	
	500–1000	17.1	0.9 (0.7, 1.1)	0.30
	1000–2000	15.1	1.0 (0.8, 1.3)	0.89
	>2000	14.3	1.0 (0.7, 1.4)	0.81
Body mass index (kg/m^2)[a]	<18.5	2.8	1.0	
	18.5–22.9	8.3	3.7 (1.7, 8.4)	0.0014
	23.0–24.9	19.7	8.0 (3.5, 18.0)	<0.0001
	25.0–26.9	30.5	14.0 (6.2, 31.6)	<0.0001
	⩾27	51.4	38.5 (17.0, 87.4)	<0.0001
Carbohydrate (%)	<45	22.1	1.0	
	45–60	11.6	0.7 (0.5, 1.0)	0.08
	60–70	16.0	1.1 (0.8, 1.4)	0.60
	>70	20.8	1.0 (0.8, 1.2)	0.64
Alcohol (g/day)	None	24.9	1.0	
	<15	11.5	0.8 (0.7, 0.9)	0.02
	15–30	15.7	0.9 (0.5, 1.4)	0.56
	>30	23.2	1.7 (0.9, 3.0)	0.10
Smoking[a]	None	18.0	1.0	
	Ex-smoker	22.4	0.9 (0.5, 1.6)	0.70
	Current smoker	30.0	1.6 (1.2, 2.1)	0.0015
Exercise	None	19.9	1.0	
(sessions/week)	⩽1	13.8	1.1 (0.8, 1.4)	0.58
	2–3	16.9	0.9 (0.6, 1.5)	0.79
	⩾4	20.1	0.8 (0.5, 1.1)	0.21

[a] $P < 0.05$ from trend test.

OR for metabolic syndrome in women residing in urban areas was higher than for women residing in rural areas, and was higher in unmarried than married women. The same findings were not observed in men. No significant associations were found between the metabolic syndrome and education level or economic status. However, the OR for the metabolic syndrome in unemployed men and women was significantly higher than in employed subjects, and the OR increased in the following order: blue collar workers, white collar workers, and the unemployed.

A prominent dose–response relationship across the BMI category was observed, and the linear trend was statistically significant for both men and women. The adjusted OR for the metabolic syndrome according to BMI increased abruptly with incremental increases in BMI for women relative to men. The OR for the metabolic syndrome were similar across the categories of carbohydrate intake. Additionally, light alcohol consumption (<15 g/day) lowered the OR for the metabolic syndrome in women. Smoking was found to be a significant risk factor in both men and women. Compared with non-smokers, the risk was significantly higher in current smokers but not in ex-smokers. A graded association across smoking status was noted with statistical significance in both genders. Moderate exercise (2–3 sessions/week) lowered the OR for the metabolic syndrome in men only.

Discussion

Using the NCEP ATP III definition of the metabolic syndrome, we have found that its age-adjusted prevalence was 14.2% in Korean men and 17.7% in Korean women, although only 2.3% of this population was obese, as defined by a BMI $\geqslant$30 kg/m^2. These results support earlier findings that central obesity and insulin resistance in Asians are associated with a high prevalence of cardiovascular risk.[8] Our findings may be explained by the thrifty genotype or phenotype theory,[22–25] in which the ability to store fuels in periods of food deprivation could lead to weight gain following rapid changes in socioeconomic status, increasingly sedentary lifestyles, or excessive energy consumption during a short period of time.

Despite the abundant research on the subject, definitions of the metabolic syndrome and the various cutoffs for its components have varied widely.[14–17] To aid in research, the WHO consultation for the classification of diabetes and its complications[13] and the NCEP ATP III[3] have recently published definitions of the metabolic syndrome. Using the WHO definition,[13] the prevalence of the metabolic syndrome among Korean adults might be lower than that reported here, in as much as the WHO criteria require the inclusion of glucose intolerance, impaired glucose tolerance or diabetes mellitus, and/or insulin resistance as components of the metabolic syndrome.

In Korean men, the prevalence of the metabolic syndrome reached a plateau during the middle years of life and beyond. In Korean women, however, the prevalence of the metabolic syndrome increased abruptly in women over 50 years of age. Menopause may be a contributing factor for this increase. Gender differences in the prevalence of the metabolic syndrome after age 50 may be related to the higher prevalence of abdominal obesity and prominent weight gain associated with ageing in women compared with men in Korea.[9]

Many studies have reported that low socioeconomic status is associated with a higher mortality rate from cardiovascular disease.[26–29] We have found that the OR for the metabolic syndrome among Korean adults was significantly increased in the unemployed, regardless of gender. We also found that the OR increased with BMI, regardless of gender, and that BMI was the most sensitive marker among associated factors for the metabolic syndrome. This finding is consistent with results for US adults, in which BMI was shown to be a strong predictor for the metabolic syndrome independent of ethnicity.[30] Asians have been shown to be more prone to obesity-related co-morbidities than Caucasians, even at lower BMI and/or smaller waist circumferences.[31] Since we found a greater preponderance of metabolic syndrome in overweight young men in this study, our results point to the need for measures to prevent and treat obesity in this and other high risk groups.

Current smoking was found to be a significant independent risk factor for the metabolic syndrome in both men and women, in accordance with previous cross-sectional studies.[30,32] Furthermore, smoking is known to impair insulin action and may lead to insulin resistance.[33] Smokers have been shown to be hyperinsulinaemic[34] and dyslipidaemic compared with a matched group of non-smokers.[35] Cigarette smoking may also induce an increase in abdominal obesity,[36] as well as causing high blood pressure by increasing sympathetic activity, and it may elevate triglyceride levels and lower HDL-cholesterol.[37] The clustering of metabolic abnormalities associated with insulin resistance has been shown to be sixfold higher in smokers than in non-smokers.[32]

In our study, mild alcohol consumption (<15 g/day) decreased the odds for the metabolic syndrome in women, but not in men. Slight and moderate alcohol consumption has been found to be associated with beneficial effects on HDL-cholesterol levels[38,39] and blood pressure.[40,41] In addition, casual drinking, but not binge drinking, has been reported to have significant cardioprotective effects.[42] Since our study did not distinguish between normal casual and binge drinking, however, associations between alcohol consumption and the metabolic syndrome should be interpreted cautiously.

A high intake of rapidly absorbed carbohydrates, characterized by a high glycaemic load, may increase the risk of coronary heart disease by aggravating glucose intolerance and dyslipidaemia (i.e. an increase in triglyceride and a decrease in HDL-cholesterol).[43] We did not find any significant association between carbohydrate intake and the metabolic syndrome, possibly because there was only a small variation in reported nutrient intake among subjects. In addition, there may be misclassifications of macronutrient intake from a single 24-hour dietary recall. These findings suggest that a potential modification of diet pattern among the subjects by their BMI or physical activity would be possible.

The OR for the metabolic syndrome was significantly decreased in men exercising moderately (2–3 sessions/week). Moderate exercise is beneficial to promote weight loss in obese individuals and favourably modifies components of the metabolic syndrome, including promoting loss of abdominal fat accumulation,[43] reduction of blood pressure,[44] improved insulin sensitivity,[45] lower triglyceride levels,[46] and increased HDL cholesterol levels.[47]

A major limitation of this study is its cross-sectional nature, suggesting that caution should be used in causal interpretation of these findings. Other limitations include the possible misclassification of risk status, recall bias, and confounding factors. Estimates from the single 24-hour recall method cannot be corrected for intra-individual daily variation in consumption. Although single recalls are useful for estimating population means, the variation of intake might in fact be under or overestimated.[48] The daily variations within a 24-hour dietary recall most probably lead to an attenuation of the effect size, thus explaining the null result for macronutrients in this study. We could not analyse other dietary factors, including those that might influence components of the metabolic syndrome, such as ingestion of fruits, vegetables, cereal fibre, and particular fatty acids, or the overall glycaemic index of the diet.

In conclusion, our study revealed that the metabolic syndrome is present in more than 15% of South Koreans, despite a low prevalence of obesity. The contributing risk factors for the metabolic syndrome were age, unemployment, higher BMI, and current smoking in both men and women. Moderate exercise in men and mild alcohol consumption in women were found to be protective factors for the metabolic syndrome. Controlling weight and smoking cessation need to be reinforced in order to effectively prevent and manage the metabolic syndrome in Korean adults.

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KEY MESSAGES

- The age-adjusted prevalence of the metabolic syndrome in South Korean adults was 14.2% for men and 17.7% for women, although only 1.7% of men and 3.0% of women were obese ($\geqslant$30 kg/m^2).
- Associated factors of the metabolic syndrome among South Koreans were age, unemployment, higher body mass index, and current smoking, regardless of gender.
- Weight control and smoking cessation need to be reinforced for the prevention and management of the metabolic syndrome in South Korean adults.

References

1 Isomaa B, Almgren P, Tuomi T *et al.* Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001;**24:**683–89.

2 Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 2002;**287:**356–59.

3 National Heart Lung and Blood Institute. Executive Summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and Treatment of high blood cholesterol in adults (Adult Treatment Panel III). *JAMA* 2001;**285:**2486–97.

4 Hauner H. Insulin resistance and the metabolic syndrome—a challenge of the new millennium. *Eur J Clin Nutr* 2002;**56(Suppl.1):**S25–29.

5 Hjermann I. The metabolic cardiovascular syndrome: syndrome X, Reaven's syndrome, insulin resistance syndrome, atherothrombogenic syndrome. *J Cardiovasc Pharmacol* 1992;**20(Suppl.8):**S5–10.

6 Ohlson LO, Larsson B, Svardsudd K *et al.* The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* 1985;**34:**1055–58.

7 Masuda T, Imai K, Komiya S. Relationship of anthropometric indices of body fat to cardiovascular risk in Japanese women. *Ann Physiol Anthropol* 1993;**12:**135–44.

8 McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991;**337:**382–86.

9 Korean Ministry of Health and Welfare. *The Korean National Health Nutrition Examination Survey 1998*. Seoul: Korean Ministry of Health and Welfare, 1999.

10 Korean National Statistical Office. *2000 Report of Statistics in Mortality in Korean*. Daejeon: Korean National Statistical Office, 2001.

11 National Heart Lung and Blood Institute. *Clinical guidelines in the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*. Bethesda, MD: NHLBI, 1998.

12 Brogan D. Software for sample survey data, misuse of standard packages. In: Armitage P, Colton T (eds). *Encyclopedia of Biostatistics.* Chichester, UK: John Wiley & Co., 1998, pp. 4167–74.

13 Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med* 1998;**15:**539–53.

14 Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes* 1988;**37:**1595–607.

15 Laaksonen DE, Lakka HM, Niskanen LK, Kaplan GA, Salonen JT, Lakka TA. Metabolic syndrome and development of diabetes mellitus: application and validation of recently suggested definitions of the metabolic syndrome in a prospective cohort study. *Am J Epidemiol* 2002;**156:**1070–77.

16 Haffner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP. Prospective analysis of the insulin-resistance syndrome (syndrome X). *Diabetes* 1992;**41:**715–22.

17 Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. *Am J Cardiol* 1999;**83:**25F–29F.

18 Korean Society of Nutrition. *Computerized Aided Nutritional Analysis Program*. Seoul: Apac Intelligence, 1998.

19 Willett WC. Will high-carbohydrate/low-fat diets reduce the risk of coronary heart disease? *Proc Soc Exp Biol Med* 2000;**225:**187–90.

20 Wirfalt E, Hedblad B, Gullberg B *et al.* Food patterns and components of the metabolic syndrome in men and women: a cross-sectional

study within the Malmo Diet and Cancer cohort. *Am J Epidemiol* 2001;**154:**1150–59.

[21] American Society of Hypertension. Recommendations for routine blood pressure measured by indirect cuff sphygmomanometry. *Am J Hypertens* 1992;**5:**207–09.

[22] Barker DJ, Hales CN, Fall CH, Osmond C, Phipps K, Clark PM. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. *Diabetologia* 1993;**36:**62–67.

[23] Fujimoto WY, Bergstrom RW, Boyko EJ, Leonetti DL, Newell-Morris LL, Wahl PW. Susceptibility to development of central adiposity among populations. *Obes Res* 1995;**3(Suppl.2):**179S–86S.

[24] Swinburn BA. The thrifty genotype hypothesis: how does it look after 30 years? *Diabet Med* 1996;**13:**695–99.

[25] Turner RC, Levy JC, Clark A. Complex genetics of type 2 diabetes: thrifty genes and previously neutral polymorphisms. *Q J Med* 1993;**86:**413–17.

[26] Brunner EJ, Marmot MG, Nanchahal K *et al.* Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia* 1997;**40:**1341–49.

[27] Choiniere R, Lafontaine P, Edwards AC. Distribution of cardiovascular disease risk factors by socioeconomic status among Canadian adults. *Can Med Assoc J* 2000;**162:**S13–24.

[28] Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* 1993;**88:**1973–98.

[29] Pekkanen J, Tuomilehto J, Uutela A, Vartiainen E, Nissinen A. Social class, health behaviour, and mortality among men and women in eastern Finland. *BMJ* 1995;**311:**589–93.

[30] Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988–1994. *Arch Intern Med* 2003;**163:**427–36.

[31] International Obesity Task Force. Asia-Pacific perspective: redefining obesity and its treatment. Western Pacific Region. Sydney: Health Communications Pty Ltd, 2000.

[32] Tahtinen TM, Vanhala MJ, Oikarinen JA, Keinanen-Kiukaanniemi SM. Effect of smoking on the prevalence of insulin resistance-associated cardiovascular risk factors among Finnish men in military service. *J Cardiovasc Risk* 1998;**5:**319–23.

[33] Attvall S, Fowelin J, Lager I, Von Schenck H, Smith U. Smoking induces insulin resistance—a potential link with the insulin resistance syndrome. *J Intern Med* 1993;**233:**327–32.

[34] Smith U. Smoking elicits the insulin resistance syndrome: new aspects of the harmful effect of smoking. *J Intern Med* 1995;**237:**435–37.

[35] Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM. Insulin resistance and cigarette smoking. *Lancet* 1992;**339:**1128–30.

[36] Shimokata H, Muller DC, Andres R. Studies in the distribution of body fat. III. Effects of cigarette smoking. *JAMA* 1989;**261:**1169–73.

[37] Kong C, Nimmo L, Elatrozy T *et al.* Smoking is associated with increased hepatic lipase activity, insulin resistance, dyslipidaemia and early atherosclerosis in Type 2 diabetes. *Atherosclerosis* 2001;**156:**373–78.

[38] Bleich S, Bleich K. Moderate alcohol consumption and the risk of cardiovascular disease. *Am J Clin Nutr* 2002;**75:**948.

[39] Lamon-Fava S. High-density lipoproteins: effects of alcohol, estrogen, and phytoestrogens. *Nutr Rev* 2002;**60:**1–7.

[40] Gillman MW, Cook NR, Evans DA, Rosner B, Hennekens CH. Relationship of alcohol intake with blood pressure in young adults. *Hypertension* 1995;**25:**1106–10.

[41] Gaziano JM, Buring JE, Breslow JL *et al.* Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. *N Engl J Med* 1993;**329:**1829–34.

[42] Murray RP, Connett JE, Tyas SL *et al.* Alcohol volume, drinking pattern, and cardiovascular disease morbidity and mortality: is there a U-shaped function? *Am J Epidemiol* 2002;**155:**242–48.

[43] Liu S, Manson JE. Dietary carbohydrates, physical inactivity, obesity, and the 'metabolic syndrome' as predictors of coronary heart disease. *Curr Opin Lipidol* 2001;**12:**395–404.

[44] Stewart KJ. Exercise training and the cardiovascular consequences of type 2 diabetes and hypertension: plausible mechanisms for improving cardiovascular health. *JAMA* 2002;**288:**1622–31.

[45] Pratley RE, Hagberg JM, Dengel DR, Rogus EM, Muller DC, Goldberg AP. Aerobic exercise training-induced reductions in abdominal fat and glucose-stimulated insulin responses in middle-aged and older men. *J Am Geriatr Soc* 2000;**48:**1055–61.

[46] Pescatello LS, Murphy D, Costanzo D. Low-intensity physical activity benefits blood lipids and lipoproteins in older adults living at home. *Age Ageing* 2000;**29:**433–39.

[47] Heim DL, Holcomb CA, Loughin TM. Exercise mitigates the association of abdominal obesity with high-density lipoprotein cholesterol in premenopausal women: results from the third National Health and Nutrition Examination Survey. *J Am Diet Assoc* 2000;**100:**1347–53.

[48] Lichtman SW, Pisarska K, Berman ER *et al.* Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Engl J Med* 1992;**327:**1893–98.