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Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan

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Abstract

Insulin resistance is a common feature of metabolic syndrome. Smokers are at great risk of developing insulin resistance. Theoretically, smoking status should be associated with metabolic syndrome. This study aimed to explore the association among cigarette smoking, metabolic syndrome, and its individual components. Information of participants regarding previous and current diseases, family history of disease, smoking habits, alcohol consumption, betel nut chewing, and physical activity status were gathered from self-reported nutrition and lifestyle questionnaires. The fasting plasma glucose, triglyceride level, high-density lipoprotein cholesterol (HDL-C) level, blood pressure, and anthropometric indices in each patient were measured. Data of 1146 male subjects were analyzed. Individuals who currently smoked had a higher prevalence of metabolic syndrome than those who had never smoked and those who had quit smoking. The adjusted odds ratios of current smoking amount showed a statistically significant dose-dependent association with metabolic syndrome, high triglyceride level, and low HDL-C level. Current smokers who smoke ≥20 pack-years have a significantly increased risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level. The higher risk of development of metabolic syndrome, high triglyceride level, and low HDL-C level was insignificant in former smokers. In conclusion, this community-based study supports the view that smoking is associated with metabolic syndrome and its individual components. Smoking cessation is beneficial to metabolic syndrome and its individual components.

1. Introduction

Smoking is an important modifiable risk factor for atherosclerosis and cardiovascular disease [1]. Smokers are at greater risk of developing insulin resistance [2,3] and subsequently diabetes than nonsmokers [4-6]. Metabolic syndrome has been documented to increase the risk of type 2 diabetes mellitus [7] and cardiovascular disease [8]. Subjects with metabolic syndrome are associated with increased all-cause and cardiovascular mortality [8,9]. Although the pathophysiology of metabolic syndrome is unclear, evidence [10,11] has shown that insulin resistance is a common feature in patients with this syndrome. Thus, smoking is theoretically an important risk factor for metabolic syndrome. The aim of this study was to investigate the association between smoking and metabolic syndrome. We also explored the dose-dependent relationship between smoking and metabolic syndrome and the relationship among its individual components and the time needed to eliminate the metabolic effects of smoking in former smokers who had smoked ≥20 pack-years.

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2. Materials and methods

This study was based on the results of the Metabolic Syndrome Study (unpublished), a community-based study conducted from October 2004 to September 2005 designed to estimate the prevalence of metabolic syndrome in a metropolitan city in Central Taiwan. The study population and sampling method in that study were as follows: The target population consisted of residents of Taichung, Taiwan, aged 40 years and older in October 2004. There were a total of 363 543 residents in that age group in the area at that time, which represented about 4.09% of the national population of the same age. A 2-stage sampling design was used to choose residents, with sampling rate proportional to size within each stage. A total of 4280 individuals were chosen. Seven hundred fifty individuals were not eligible and therefore were excluded from the study sample. Among the 3530 individuals, 2359 agreed to participate in the Metabolic Syndrome Study. In the current study, we restricted our analyses to men because the proportion of Taiwanese women who smoked was too small (4.7% for former and current smokers). Informed consent was obtained from each participant, and the study was approved by the Human Research Committee of the China Medical University Hospital.

Data were collected from self-reported nutrition and lifestyle questionnaires. Items in the questionnaire explored basic data (sex, age, education level, marital status, income), previous and current diseases, family history of disease, smoking habits, alcohol consumption, betel nut chewing, and physical activity status. The nutrition survey used food-intake frequency method to assess daily energy and nutrition intakes. Participants were queried about age at onset of smoking, number of cigarettes smoked per day, number of smoking days per week, and age when they quit smoking. Participants were grouped according to whether they had never smoked, formerly smoked, or currently smoked. *Former smokers* were defined as those who had abstained from smoking for ≥1 year at the time of the study. Individuals who were currently smoking were labeled as *current smokers*. Assuming 20 cigarettes per pack, pack-years were estimated using the following formula: cigarettes per day/20 × years smoked. Level of education was categorized into <ninth grade, between ninth and 12th grade, and >12th grade education. Household income was classified as <US $1250, between US $1250 and US $4000, and >US $4000 per month. Alcohol consumption was assessed by types of beverage drank, age at onset of drinking or age when they began to abstain, frequency of drinking, and average amount of alcohol per drink. Physical activity was measured by frequency, duration, and intensity of walking, jogging, running, bicycle riding, swimming, aerobes, aerobic dancing, other types of dancing, tennis, table tennis, golf, basketball, and badminton. An activity metabolic equivalent (MET) was calculated by assigning a MET index to each activity and by calculating the product of intensity × duration × frequency of activity [12].

All participating subjects presented to the outpatient clinic of the department of family medicine subsequent to an overnight fasting. They were weighed in light clothing, and their heights were measured. Waist circumference was measured midway between the inferior margin of the last rib and the crest of the ileum in a horizontal plane. Circumference was measured to the nearest 1 mm. Blood pressure was measured from the right arm subsequent to the participant sitting at rest for a period of 20 minutes. The mean of 2 blood pressure recordings was used for statistical analysis. Fasting blood samples were drawn between 8:00 AM and 10:00 AM.

Metabolic syndrome was diagnosed if a subject met 3 or more of the following 5 criteria: serum triglyceride level ≥1.69 mmol/L (150 mg/dL) or on hypolipidemic agent; serum high-density lipoprotein cholesterol (HDL-C) level <1.03 mmol/L (40 mg/dL) in men or <1.29 mmol (50 mg/dL) in women or on hypolipidemic agent; average blood pressure ≥130/85 mm Hg or currently taking antihypertensive medication; fasting plasma glucose ≥6.1 mmol/L (110 mg/dL) or on oral antidiabetic medication. Because Asians have greater risk of fitting the metabolic profiles at lower waist circumference than white persons [13], we used waist circumference >90 cm for men and >80 cm for women as a diagnostic criterion of abdominal obesity.

2.1. Laboratory analysis

Plasma glucose level was determined by a glucose oxidase method (Synchron LX-20; Beckman Coulter, Fullerton, CA). Plasma triglyceride level was determined by an enzymatic colorimetric method (Synchron LX-20, Beckman Coulter). The HDL-C level was measured by a direct HDL-C method (Synchron LX-20).

2.2. Statistical analysis

All data are presented as mean ± standard deviation. Using smoking status as a factor, continuous variables were analyzed by 1-way analysis of variance and nominal variables were analyzed by χ^2 test to determine if there were significant differences between the groupings of smoking on categorical covariates. Bonferroni test was used for post hoc analysis. Multiple logistic regression analysis was used to calculate odds ratios (ORs), and the linear trend was evaluated using the trend test. A P value of less than .05 represented a statistically significant difference between compared data sets. All analyses were performed with the SAS statistical package 8.1 (SAS, Cary, NC).

3. Results

Among the 1146 men, 553 (48%) subjects had never smoked, whereas 259 (23%) subjects were former smokers and 334 (29%) subjects currently smoked. Former smokers were older than the other 2 groups. Never smokers had greater percentage of education level >12th grade than the

Table 1
Characteristics of the study subjects categorized by smoking status

Characteristic	Never (1) (n = 553)	Former (2) (n = 259)	Current (3) (n = 334)	*P*	Bonferroni test
Age (y)	59.58 ± 11.95	61.78 ± 13.28	54.29 ± 10.99	<.001	(2) > (1) > (3)
Education level				<.001	(1) > (2), (3)
<9	133 (35.14)	87 (33.72)	109 (32.73)		
9-12	241 (43.74)	123 (47.67)	178 (53.45)		
>12	177 (32.12)	48 (18.60)	46 (13.81)		
Household income (US$/mo)				.081	
<1250	244 (44.77)	127 (49.42)	170 (51.67)		
1250-4000	223 (40.92)	104 (40.47)	130 (39.51)		
>4000	78 (14.31)	26 (10.12)	29 (8.81)		
Alcohol consumption (g/d)					
Current drinker	11.41 ± 20.24	20.50 ± 32.42	23.66 ± 31.15	.001	(3) > (1)
Former drinker	20.12 ± 41.06	40.23 ± 95.30	32.36 ± 53.19	.627	
Physical activity (MET-h/wk)	89.98 ± 75.11	89.24 ± 78.88	107.99 ± 93.33	.002	(3) > (1), (2)
Daily energy intake					
Total calorie (kcal)	2204.36 ± 797.89	2154.21 ± 788.32	2170.43 ± 758.70	.658	
Carbohydrate	412.56 ± 141.18	399.87 ± 140.16	405.18 ± 134.43	.451	
Fat	32.16 ± 16.26	32.33 ± 16.18	32.57 ± 16.47	.936	
Protein	72.84 ± 32.53	71.81 ± 31.13	70.77 ± 31.13	.644	
Fiber	23.49 ± 15.74	21.62 ± 13.97	25.29 ± 18.69	.025	(3) > (2)
Anthropometric measure					
Body mass index (kg/m^2)	24.60 ± 3.16	25.04 ± 3.15	24.86 ± 3.21	.155	
Metabolic syndrome parameters					
Waist circumference >90 cm	149 (26.94)	83 (32.05)	107 (32.07)	.168	
Triglyceride ≥150 mg/dL or on medication	158 (28.57)	86 (33.20)	138 (41.32)	<.001	(3) > (2) > (1)
HDL-C <40 mg/dL or on medication	283 (51.18)	126 (48.65)	197 (58.98)	.023	(3) > (1), (2)
Blood pressure ≥130/85 mm Hg or on medication	373 (67.45)	197 (76.06)	226 (67.66)	.032	(2) > (1), (3)
Fasting plasma glucose ≥110 mg/dL or on medication	122 (22.06)	76 (29.34)	74 (22.16)	.054	
Prevalence of metabolic syndrome	189 (34.18)	100 (38.61)	143 (42.81)	.034	

Data are presented as mean ± SD or n (percentage).

other 2 groups, but household income was similar among 3 groups. Current smokers had the highest amount of current alcohol consumption. They also had the greatest amount of physical activity and fiber intake. The amount of physical activity between never smokers and former smokers was similar. They also had similar amount of fiber intake. There were no significant differences among the 3 groups in body mass index and waist circumference. Current smokers had a higher percentage of high triglyceride level and low HDL-C level than the other 2 groups. Former smokers had a higher percentage of high triglyceride level than never smokers, but the percentage of low HDL-C level between these 2 groups was similar. The percentage of high blood pressure was greatest in former smokers. Both never smokers and current smokers had similar percentage of high blood pressure. Metabolic syndrome was more prevalent in current smokers than in former smokers and those who had never smoked (P = .034) (Table 1). However, Bonferroni test could not determine the nature of differences among these 3 groups. There was a statistically significant dose-dependent relationship among smoking amount and metabolic syndrome (P = .003 for trend), high triglyceride level (P = .002 for trend), and low HDL-C level (P < .001 for trend) (Table 2). After controlling for other covariates, current smokers who smoke ≥20 pack-years had a significantly higher risk of developing metabolic syndrome (ORs, 1.82; 95% confidence interval

Table 2
Adjusted ORs of metabolic syndrome and its components according to smoking amount in current smokers

	Never	Former smoker	Current smoker (cigarettes/d)				*P* for trend
			1-9	10-19	20-39	≥40	
Metabolic syndrome	1.00	1.09 (0.78-1.54)	1.49 (0.87-2.57)	1.44 (0.96-2.16)	1.91 (1.14-3.20)	2.99 (0.63-14.20)	.003
Abdominal obesity	1.00	1.16 (0.81-1.65)	1.35 (0.77-2.38)	0.99 (0.64-1.54)	1.63 (0.95-2.78)	4.16 (0.88-19.70)	.113
High triglyceride	1.00	1.20 (0.84-1.71)	1.34 (0.76-2.35)	1.47 (0.97-2.22)	2.11 (1.26-3.54)	3.10 (0.65-14.84)	.002
Low HDL-C	1.00	1.03 (0.74-1.43)	1.52 (0.89-2.59)	1.86 (1.24-2.78)	2.18 (1.28-3.70)	2.83 (0.52-15.30)	<.001
High fasting glucose	1.00	1.21 (0.83-1.76)	1.08 (0.58-2.00)	0.94 (0.58-1.51)	0.77 (0.39-1.50)	3.00 (0.63-14.36)	.748
High blood pressure	1.00	1.23 (0.84-1.81)	0.74 (0.41-1.33)	0.96 (0.62-1.48)	1.17 (0.66-2.08)	0.75 (0.16-3.55)	.905

Adjusted for age, education level, household income, alcohol consumption, physical activity, mean daily energy intake, fiber intake, and TV watching.

Table 3
Adjusted ORs of metabolic syndrome, high triglyceride level, and low HDL-C level stratified by pack-years

Smoking status	n	Adjusted ORs (95% CI) metabolic syndrome	Adjusted ORs (95% CI) high triglyceride	Adjusted ORs (95% CI) low HDL-C
Never	553	1.00	1.00	1.00
Former				
<20 pack-y	150	1.02 (0.68-1.55)	1.08 (0.71-1.66)	0.92 (0.62-1.36)
≥20 pack-y	109	1.24 (0.78-1.96)	1.42 (0.88-2.28)	1.25 (0.80-1.97)
Current				
<20 pack-y	145	1.14 (0.74-1.74)	1.29 (0.84-1.99)	1.47 (0.98-2.21)
≥20 pack-y	189	1.82 (1.26-2.65)	1.86 (1.28-2.72)	2.17 (1.49-3.18)

Adjusted for age, education level, household income, alcohol consumption, physical activity, mean daily energy intake, fiber intake, and TV watching.

[CI], 1.26-2.65), high triglyceride level (ORs, 1.86; 95% CI, 1.28-2.72), and low HDL-C level (ORs, 2.17; 95% CI, 1.49-3.18) than those who had never smoked (Table 3). The higher risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level disappeared in former smokers (Table 3). After stratification by smoking cessation years in former smokers who had smoked ≥20 pack-years, the risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level was insignificant since stopping smoking for 1 to 4 years (Table 4). Although it was not statistically significant, cessation of smoking had a trend to lower the risk of developing metabolic syndrome in a year-dependent manner (Table 4).

4. Discussion

This study showed that current smokers demonstrated a significantly higher risk for development of metabolic syndrome that was associated with abnormality in triglyceride level and HDL-C level but not significantly related to the presence of high blood pressure, abnormal fasting glucose concentrations, or increased waist circumference. Among this group, there was a dose-dependent association among smoking amount and development of metabolic syndrome, high triglyceride level, and low HDL-C level. Metabolic syndrome and its individual components, high triglyceride level and low HDL-C level, were significantly higher in current smokers with a smoking amount ≥20 pack-years. Smoking cessation lowered the risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level.

Insulin resistance is the key pathophysiology of metabolic syndrome [14]; previous studies have shown that many long-term cigarette smokers are insulin resistant [2] and hyperinsulinemic [3]. Our study supports the findings of previous studies [15-18] that metabolic syndrome is more frequent in current smokers than in those who have never smoked. Weitzman et al [15] demonstrated that exposure to environmental tobacco smoke had a dose-response relationship with metabolic syndrome. Our study also revealed that cigarette smoking had a dose-dependent association with metabolic syndrome. This finding was consistent with previous studies [16-18]. After analysis of the components of metabolic syndrome, we found that there is also a dose-dependent association among smoking, high triglyceride level, and low HDL-C level, a finding that was also reported by other studies [2,16,17]. Ishizaka et al [16] found a higher prevalence of carotid plaque among both former and current smokers compared with those who had never smoked. Epidemiologic studies have shown that smoking raises the risk of cardiovascular disease [19,20]. Although there is still lack of strong evidence to support that high triglyceride level alone is an independent cardiovascular risk factor, high triglyceride level combined with low-density lipoprotein cholesterol–HDL-C ratio >5 or combined with low HDL-C level was a powerful risk factor for nonfatal myocardial infarction or coronary artery disease [21]. Previous large population studies have shown that low HDL-C level was inversely correlated with cardiovascular disease [22,23]. In the Framingham Heart Study, HDL-C level was found to be the strongest predictor of clinical events [23]. The increase in high triglyceride level and decrease in HDL-C level caused by smoking may explain in part the atherogenic change of blood vessels and the subsequent cardiovascular events seen in smokers. Our study revealed that long-term smoking is not associated with abdominal obesity, a finding consistent with a Korean study [17] but one that is inconsistent with studies on white persons [4,24-26]. This discrepancy may be due to ethnic differences. Although previous studies support the association between smoking and diabetes [4-6], our study did not find an association between smoking and high fasting plasma glucose. The cross-sectional survey of this study may

Table 4
Adjusted ORs of metabolic syndrome, high triglyceride level, and low HDL-C level stratified by smoking cessation years

Smoking status	n	Adjusted OR (95% CI) metabolic syndrome	*P* for trend	Adjusted OR (95% CI) high triglyceride	*P* for trend	Adjusted OR (95% CI) low HDL-C	*P* for trend
Never	553	1.00		1.00		1.00	
Former smoker							
≥20 pack-y			.743		.201		.421
Cessation 1-4 y	36	1.30 (0.62-2.74)		1.46 (0.68-3.11)		1.20 (0.58-2.49)	
4-12 y	35	1.18 (0.56-2.49)		1.35 (0.62-2.96)		1.06 (0.51-2.21)	
≥12 y	38	1.01 (0.46-2.20)		1.52 (0.67-3.46)		1.40 (0.65-3.02)	

Adjusted for age, education level, household income, alcohol consumption, physical activity, mean daily energy intake, fiber intake, and TV watching.

have limited our ability to detect the association. The relationship between long-term smoking and hypertension is still unclear and controversial [27]; and therefore, smoking is not considered a risk factor for the development of hypertension. In this study, no association between smoking and high blood pressure was found.

Compared with people who have never smoked, current smokers who smoke ≥20 pack-years have a higher risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level. This finding is consistent with a previous report [28]. The higher risk of developing metabolic syndrome, high triglyceride level, and low HDL-C level disappeared in former smokers, which reinforces the beneficial effects of smoking cessation on metabolic syndrome and its individual components. However, this study had some limitations. For example, the smoking status was based on the results of self-reported questionnaires; some of the individuals may be misclassified. Furthermore, we defined *former smokers* as having been abstinent from tobacco for 1 year or longer. Thus, individuals who were not currently smoking but had smoked within the past 1 year were classified as *current smokers*. This classification may influence some of the results of this study.

In conclusion, this community-based study supports the view that the presence of metabolic syndrome appears to be greater in current smokers than in never smokers or former smokers and that this increased risk of developing metabolic syndrome appears to be related to abnormalities in triglyceride level and HDL-C level rather than the presence of high blood pressure, abnormal fasting plasma glucose, or increased waist circumference. Smoking cessation is beneficial to metabolic syndrome and its individual components. The molecular mechanism(s) of nicotine on metabolic syndrome needs further investigation.

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