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Association between Cigarette Smoking and Metabolic Syndrome in Thais

Abstract

Purpose: To investigate the relationship between metabolic syndrome as defined by using the modified NCEP/ATP III criteria [modified the National Cholesterol Education Program (NCEP)/Adult Treatment Panel III (ATP III) criteria] and cigarette smoking in Thai subjects.

Methods: This study was carried out among 254 smokers and 144 nonsmokers from suburban and urban residential areas in Bangkok, Thailand. All anthropometric variables, blood pressures, resting heart rate and biochemical parameters in each subject were measured.

Results: The anthropometric variables, biochemical parameters, blood pressures and resting heart rate were not significantly different between smokers and nonsmokers, except for white blood cell count (WBC). Cigarette smoking was associated with increased risk for metabolic syndrome (OR =1.97; 95% CI=1.11-3.42) and the percentages of metabolic syndrome in smoker and nonsmoker Thais were 22.8% and 13.2%, respectively. Moreover, the number of cigarette smoking per day showed significant association with metabolic syndrome (p=0.047). Logistic regression analysis revealed that cigarette pack-years, resting heart rate, body mass index (BMI) and total cholesterol were significantly increased risk factors for metabolic syndrome.

Conclusion: The current findings suggest that cigarette smoking is associated with the increased risk of metabolic syndrome by using the modified NCEP/ATP III criteria in Thais.

Keywords: Smoker; Cigarette pack-years; Modified NCEP/ATP III criteria; Biochemical parameters; Anthropometric variables.

Kanjana Suriyaprom[1]*

Pisit Namjuntra[1]

Kittisak Thawnasom[1]

Yaowaluk Pimainok[1]

Rungsunn Tungtrongchitr[2]

[1]Faculty of Medical Technology, Rangsit University, Paholyothin Road, Pathumthani 12000, Thailand.

[2]Department of Tropical Nutrition and Food Science, Faculty of Tropical Medicine, Mahidol University, 420/6 Rajvithi Road, Rajthevee, Bangkok 10400, Thailand.

***For correspondence:**

Tel: 66-2-9972222 ext. 1437, 1451
66-8-98975337

Fax: 66-2-9972222 ext 1451

E-mail: ksuriyaprom@yahoo.com

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Introduction

Nowadays, the prevalence of metabolic syndrome is increasing in the world and reports from many countries had similar prevalence rates ranging between 10-20% [1-3]. The prevalence in Nongkhai province, Thailand is about 17% [4]. Metabolic syndrome is a cluster of risk factors predictive of future cardiovascular diseases and type 2 diabetes mellitus [5-6]. Many factors,

including physical inactivity, obesity, and an unhealthy diet, appeared to promote the development of the disease [7], but the mechanisms causing the onset are not fully understood.

Smoking may be considered as an important modifiable risk factor for metabolic syndrome. Cigarette smoking is an important health problem in Thailand, and the proportion of smokers is approximately 21.2 percent [8]. It is considered to increase morbidity and the mortality risk of many chronic diseases such as atherosclerosis, cardiovascular diseases, type 2 diabetes, emphysema, and various types of malignancies [9-10]. Chemical components of cigarette smoke, other than nicotine, contribute to cardiovascular injury by causing production of carboxyhaemoglobin, increasing platelet aggregation, adversely affecting the lipid profile and producing oxidant injury [11,12]. Smokers have abnormalities in lipoprotein metabolism and endothelial function [13]. Several studies reported that smoking also induced insulin resistance and conducted to type 2 diabetes [6,14]. However, the link between smoking and metabolic syndrome has not been properly elucidated. Although some earlier studies have not found the relationship between smoking and metabolic syndrome [15-17], none of these studies was done in Thai subjects. Thus, the objective of this study was to investigate the relationship between metabolic syndrome and cigarette smoking in Thai subjects.

Methods

Subjects

Following approval by the Ethics Committee of Faculty of Tropical Medicine, Mahidol University, Bangkok, a convenient sample size of 398 Thai volunteers (aged 21 to 62 years) were recruited from suburban and urban residential areas in Bangkok, Thailand. These excluded subjects with a history of diabetes mellitus, liver, kidney and cardiovascular diseases as confirmed by physical appropriate examinations and laboratory tests. Of these subjects, 254 were smokers, while the rest (144) were nonsmokers. Using an interview format, all the volunteers were interviewed to obtain information on their lifestyle pattern, and medical history. Smoking characteristics such as age at onset of smoking, the number of cigarettes smoked and duration of smoking (years) were recorded in details. Cigarette pack-years were computed as duration of smoking (years) multiplied by the number of smoked cigarettes and divided by 20.

Overnight fasting venous blood (5 ml) was taken from each subject. Serum was used to assay biochemical variables e.g. total cholesterol, triglycerides, HDL-cholesterol, aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP). NaF blood was used to assay glucose and EDTA blood was used to assay hematological variables e.g. hemoglobin, hematocrit, and white blood cell count (WBC).

Anthropometric measurements, comprising weight, height, triceps skinfold thickness (TSF), waist circumference, and hip circumference for each subject were determined. For each subject, the waist and hip circumferences were measured and the waist and hip circumference ratio calculated. Body mass index (BMI) was determined and expressed as weight (kg) / height (m^2). Resting heart rate and blood pressure (BP) were measured by a nurse after 5 to 10 minutes of rest in the sitting position.

Laboratory techniques

For each subject, glucose, total cholesterol, triglycerides, HDL-cholesterol, AST, ALT and ALP were measured using enzymatic methods by DADE Dimension ®AR. Hemoglobin as well as hematocrit concentrations and WBC count were determined by Coulter Counter.

Criteria for metabolic syndrome

The metabolic syndrome was defined by using the modified NCEP/ATP III criteria [18]. The new cut-off on waist circumference in the Asia pacific region was used instead of original cut-off for waist circumference in ATP III criteria. The modified NCEP/ ATP III definition required at least three of the followings: (1) raised waist circumference: >90 cm in men and >80 cm in

women for Asians, (2) raised triglyceride level which is ≥ 150 mg/dL, (3) reduced high-density lipoprotein (HDL)-cholesterol which is < 40mg/dL in men and <50 mg/dL in women, (4) raised blood pressure: systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥ 85 mmHg or current antihypertensive medication, and (5) raised fasting plasma glucose which is ≥ 100 mg/dL.

Statistical analysis

Statistical calculations were performed using SPSS for Windows version 17.0 (SPSS, Chicago, IL). The median and 95% confidence interval (C.I.) were calculated. Difference between two groups was determined using the Mann-Whitney U-Wilcoxon Rank Sum W test. Relationship between metabolic syndrome and cigarette smoking was tested using Chi-square test and odds ratio (OR) determined as appropriate. To assess the association between metabolic syndrome as dependent variable and potential factors, logistic regression was applied. At 95% confidence interval, p values <0.05 were considered statistically significant. The goodness of fit of the logistic regression models was tested with the Hosmer Lemeshow test.

Results

The median and 95% confidence intervals of the age, blood pressures, resting heart rate, biochemical and hematological measurements as well as anthropometric variables in smokers and nonsmokers are shown in Table 1. All anthropometric variables, biochemical parameters, blood pressures and resting heart rate among the smokers were not statistically significantly different from those of the nonsmokers. Significantly higher WBC counts were observed among the smokers when compared with the nonsmokers.

The percentage of metabolic syndrome in this study is presented in Table 2. As defined by the modified NCEP / ATP III criteria, 77 (19.3%) of

Table 1: Age, anthropometric variables, biochemical and hematological parameters of volunteers

Parameters	Smokers (n= 254)		Nonsmokers (n=144)		p-value
	Median	95%C.I.	Median	95%C.I.	
Age (years)	37	35-40	36	34-39	0.289
BMI (kg/m^2)	23.66	23.00-24.02	23.36	22.84-24.32	0.462
Waist (cm)	83.00	80.80-85.00	83	80.79-85.00	0.467
Hip (cm)	96	95-97	96	94-97	0.959
Waist/Hip ratio	0.86	0.85-0.87	0.87	0.85-0.88	0.227
TSF (mm)	12.00	10.50-13.00	12.05	10.59-13.40	0.946
Systolic BP (mmHg)	120	120-122	120	116-123	0.781
Diastolic BP (mmHg)	80	78-80	78	76-80	0.434
Resting heart rate (beats/minute)	73	72-75	72	69-73	0.061
Glucose (mg/dL)	85	83-87	82	79-84	0.093
Total cholesterol (mg/dL)	204	197-210	203	198-210	0.902
Triglycerides (mg/dL)	124	114-138	121	112-129	0.122
HDL-C (mg/dL)	49	48-51	50	48-52	0.925
AST (U/L)	30	29-33	29	27-32	0.348
ALT (U/L)	30	28-33	31	28-33	0.397
ALP (U/L)	74	70-76	69	66-72	0.076
Hemoglobin (g/dL)	14.8	14.7-15.0	14.6	14.4-15.0	0.371
Hematocrit %	44.6	44.1-45.1	44.3	44.0-44.9	0.413
WBC (10^9/L)	7,100	6,800-7,500	5,900	5,700-6,300	0.000*

**p < 0.05*

Table 2: Prevalence of metabolic syndrome in smokers and nonsmokers (percentages in parentheses)

	Prevalence	
	Metabolic syndrome n=77	Non metabolic syndrome n=321
Smokers	58 (22.8)	196 (77.2)
Nonsmokers	19 (13.2)	125 (86.8)

the subjects had the metabolic syndrome. Smoking was found associated with increased risk for metabolic syndrome (OR=1.97; 95% C.I.=1.11-3.42, p=0.019) (Table 2). The prevalence of metabolic syndrome increased proportionally with increase in the number of cigarettes smoked per day (Table 3). BMI (OR =1.48, p<0.01), resting heart rate (OR =1.07, p=0.011), total cholesterol (OR =1.01, p=0.025) and cigarette pack-years (OR =1.08, p=0.009) had significant impact on the metabolic syndrome.

Table 3: Prevalence of metabolic syndrome according to the number of cigarette smoking per day.

Cigarettes per day	Prevalence of metabolic syndrome (n= 58)
1-5	17.5% (n=7)
6-10	19.8% (n=20)
11-20	24.3% (n=18)
>20	33.3% (n=13)

**p < 0.05 (Pearson Chi-square)*

Discussion

In this study, we found a good relationship between cigarette smoking and metabolic syndrome in Thai subjects. This finding is consistent with results from previous epidemiologic studies [19-20]. In Japanese, smoking was positively related to metabolic syndrome (OR=1.4) and the prevalence of metabolic syndrome was 19% in male smokers who did not drink [19]. In Korean, the prevalence of metabolic syndrome in sustained smokers was 14% [20]. Studies in Taiwan and in Korea showed a higher prevalence of metabolic syndrome in smokers than in never smokers and current smoking had a significant dose-dependent association with metabolic syndrome [21-22]. Although many pharmacological actions of cigarette smoking and nicotine have been demonstrated [23-24], the mechanism of how cigarette smoking increases the risk of the metabolic syndrome is still not clear. Insulin resistance is the key pathophysiology of metabolic syndrome [25]; previous study has shown that long-term cigarette smokers are insulin resistance [26]. A negative effect of cigarette smoking on insulin-mediated glucose uptake has been documented in studies [26-27]. Cigarette smoking elevates the circulating concentrations of insulin-antagonistic hormones [28] and chemical components of cigarette smoke may have direct toxic effects on the pancreas as well as on insulin receptor sensitivity [24]. Thus, cigarette smoking may contribute to the metabolic disturbances and may increase the risk of the metabolic syndrome. On the other hand, some studies had not found the relationship between smoking and the metabolic syndrome [29-31]. Gharipour et al reported that the percentage of nonsmokers with three components of metabolic syndrome was higher than in smokers [29] and in Iranian middle aged women, the odds ratio showed no significant association

Table 4: Multivariate logistic regression analysis when metabolic syndrome was used as dependent variable and BMI, total cholesterol, resting heart rate, WBC as well as cigarette pack-years were taken as independent variables

Variables	β	Odds ratios Exp (β)	(95% C.I.)	p-value
BMI	0.39	1.48	1.28-1.70	<0.001*
Resting heart rate	0.07	1.07	1.01-1.12	0.011*
Total cholesterol	0.01	1.01	1.00-1.02	0.025*
WBC	0.00	1.00	1.00-1.00	0.727
Cigarette pack-years	0.08	1.08	1.02-1.15	0.009*

**p<0.05*

between metabolic syndrome and smoking [28]. Moreover, our findings support that cigarette smoking has adverse affect on health. Multivariate logistic regression in this study showed that BMI, resting heart rate, total cholesterol and cigarette pack-years had significant impact on the metabolic syndrome, which is consistent with previous findings [32-33]. That elevated resting heart rate is associated with metabolic syndrome in both men and women has been reported by Rogowski et al [32]. BMI and total cholesterol were significantly higher in present metabolic syndrome group than non metabolic syndrome group [34]. Gnacnska et al also showed that increased BMI was the biggest factors stimulating manifestation of metabolic syndrome [33] and BMI was the positive associated factor to the diagnosis of metabolic syndrome in logistic regression model [35].

Conclusion

Our study found that cigarette smoking is associated with metabolic syndrome. Smokers had higher circulating WBC count than nonsmokers and the trend of resting heart rate increases in smokers. These observations may provide the data to promote antismoking campaign. Further investigation should clarify the mechanisms between cigarette smoking and the metabolic syndrome in large population studies.

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Conflict of Interest

We declare that we have no conflict of interest associated with this work.

Contribution of Authors

We declare that this work was done by the authors named in this article and all liabilities pertaining to claims relating to the content of this article will be borne by the authors**.** Kanjana Suriyaprom collected specimens and carried out laboratory analysis as well as preparation of the manuscript and contributed to the design of the study. Pisit Namjuntra carried out laboratory analysis and was involved in preparation of the manuscript. Rungsunn Tungtrongchitr and Kittisak Thawnasom collected specimens and analyzed the data. Yaowaluk Pimainog was involved in the preparation of manuscript. All authors approved the manuscript.

References

1. Gu D, Reynolds K, Wu X, Chen J, Duan X, Reynolds RF, Whelton PK, He J, InterASIA Collaborative Group. Prevalence of the metabolic syndrome and overweight among adults in China. Lancet 2005; 365: 1398-1405.
2. Ko GT, Cockram CS, Chow CC, Yeung V, Chan WB, So WY, Chan NN, Chan JC. High prevalence of metabolic syndrome in Hong Kong Chinese--comparison of three diagnostic criteria. Diabet Res Clin Pract 2005; 69: 160-168.
3. Yoon YS, Oh SW, Baik HW, Park HS, Kim WY. Alcohol consumption and the metabolic syndrome in Korean adults: the 1998 Korean National Health and Lamthong Kaewtrakulpong. Metabolic Syndrome: Prevalence in Si Chiang Mai District, Nong Khai Province, Thailand. J Trop Med Parasitol 2008; 31: 41-47.
4. Nutrition Examination Survey. Am J Clin Nutr 2004; 80: 217-224.
5. Malik S, Wong ND, Franklin SS, Kamath TV, L'Italien GJ, Pio JR, Williams GR. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. Circulation 2004; 110: 1245-1250.
6. 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: 1070-1077.
7. Grundy SM, Hansen B, Smith Jr SC, Cleeman JI, Kahn RA. Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association conference on scientific issues related to management, Circulation 2004; 109: 551-556.

8. Rimm EB, Chan J, Stampfer MJ, Colditz GA, Willett WC. Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. BMJ 1995; 310: 555-559.
9. Diana JN. Tobacco smoking and nutrition. Ann N Y Acad Sci 1993; 686: 1-11.
10. Suriyaprom K, Tungtrongchitr R. Cigarette: risk factors of cardiovascular diseases. J Tobacco Control 2007; 1: 77-85.
11. Suriyaprom K, Tungtrongchitr R, Namjuntra P. Free radicals and antioxidants in cigarette smokers. J Med Tech Assoc Thailand 2006; 34: 1349-1360.
12. Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto FJ, Tell GS. Cigarette smoking and progression of atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study. JAMA 1998; 279:119-124.
13. Kong C, Nimmo L, Elatrozy T, Anyaoku V, Hughes C, Robinson S, Richmond W, Elkeles RS. Smoking is associated with increased hepatic lipase activity, insulin resistance, dyslipidaemia and early atherosclerosis in type 2 diabetes. Atherosclerosis 2001; 156: 373-378.
14. Nakanishi N, Nakamura K, Matsuo Y, Suzuki K, Tatara K. Cigarette smoking and risk for impaired fasting glucose and type 2 diabetes in middle-aged Japanese men. Ann Intern Med 2000; 133: 183-191.
15. Gharipour M, Kelishadi R, Sarrafzadegan N, Baghaei A, Yazdani M, Anaraki J, Eshrati B, Tavassoli AA. The association of smoking with components of the metabolic syndrome in non-diabetic patients. Ann Acad Med Singapore 2008; 37: 919-923.
16. Delavar MA, Lye MS, Khor GL, Hanachi P, Hassan ST. Prevalence of metabolic syndrome among middle aged women in Babol, Iran. Southeast Asian J Trop Med Public Health 2009; 40: 612-628.
17. Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K. Coronary Artery Risk Development in Young Adults study: Risk factors for the metabolic syndrome: the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985-2001. Diabet Care 2004; 27: 2707-2715.
18. Rahim MA, Azad Khan AK, Sayeed MA, Akhtar B, Nahar Q, Ali SMK, Hussain A. Metabolic syndrome in rural Bangladesh: Comparison of newly proposed IDF, modified ATP III and WHO criteria and their agreements. Clin Res Rev 2007; 1: 251-257.
19. Takeuchi T, Nakao M, Nomura K, Yano E. Association of metabolic syndrome with smoking and alcohol intake in Japanese men. Nicotine Tob Res 2009; 11: 1093-1098.
20. Kim BJ, Kim BS, Sung KC, Kang JH, Lee MH, Park JR. Association of smoking status, weight change, and incident metabolic syndrome in men: a 3-year follow-up study. Diabet Care 2009; 32: 1314-1316.
21. Chen CC, Li TC, Chang PC, Liu CS, Lin WY, Wu MT, Li CI, Lai MM, Lin CC. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. Metabol 2008; 57: 544-548.
22. Oh SW, Yoon YS, Lee ES, Kim WK, Park C, Lee S, Jeong EK, Yoo T, Korea National Health and Nutrition Examination Survey. Association between cigarette smoking and metabolic syndrome: the Korea National Health and Nutrition Examination Survey. Diabet Care 2005; 28: 2064-2066.
23. Benowitz NL. Pharmacologic aspects of cigarette smoking and nicotine addition. N Engl J Med 1988; 319: 1318-30.
24. Pasupathi P, Bakthavathsalam G, Rao YY, Farook J. Cigarette smoking—Effect of metabolic health risk: A review. Clin Res Rev 2009; 3: 120–127.
25. 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.
26. Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM. Insulin resistance and cigarette smoking. Lancet 1992; 339: 1128-1130.
27. Rönnemaa T, Rönnemaa EM, Puukka P, Pyörälä K, Laakso M. Smoking is independently associated with high plasma insulin levels in nondiabetic men. Diabet Care 1996; 19: 1229-1232.
28. Kirschbaum C, Wust S, Strasburger CJ. 'Normal' cigarette smoking increases free cortisol in habitual smokers. Life Sci 1992; 50: 435-442.
29. Gharipour M, Kelishadi R, Sarrafzadegan N, Baghaei A, Yazdani M, Anaraki J, Eshrati B, Tavassoli AA. The association of smoking with components of the metabolic syndrome in non-diabetic patients. Ann Acad Med Singapore 2008; 37: 919-923.
30. Delavar MA, Lye MS, Khor GL, Hanachi P, Hassan ST. Prevalence of metabolic syndrome among middle aged women in Babol, Iran. Southeast Asian J Trop Med Public Health 2009; 40: 612-628.
31. Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K. Coronary Artery Risk Development in Young Adults study. Risk factors for the metabolic syndrome: the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985-2001. Diabet Care 2004; 27: 2707-2715.
32. Rogowski O, Steinvil A, Berliner S, Cohen M, Saar N, Ben-Bassat OK, Shapira I. Elevated resting heart rate is associated with the metabolic syndrome. Cardiovasc Diabetol 2009; 8: 55.
33. Gnacińska M, Małgorzewicz S, Lysiak-Szydłowska W, Sworczak K. The serum profile of adipokines in overweight patients with metabolic syndrome. Endokrynol Pol 2010; 61: 36-41.
34. Tonstad S, Svendsen M. Premature coronary heart disease, cigarette smoking, and the metabolic syndrome. Am J Cardiol 2005; 96: 1681-1685.
35. Limpawattana P, Sawanyawisuth K, Busaracome P, Foocharoen C, Phitsanuwong C, Chumjan S, Chotmongkol R, Chaisuksant S. The best criteria to diagnose metabolic syndrome in hypertensive Thai patients. J Med Assoc Thai. 2008; 91(4): 485-490.