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ORIGINAL ARTICLE

C-reactive protein concentration as a significant correlate for metabolic syndrome: a Chinese population-based study

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Abstract Increasing evidence suggests that chronic, low-grade inflammation may be a common soil involving the pathogenesis of metabolic syndrome (MetS) and cardiovascular disease. We examined the association between C-reactive protein (CRP) concentration, an extensively studied biomarker of low-grade inflammation, and the MetS in a representative sample of Chinese adults in Taiwan. We performed a cross-sectional analysis of data from 4234 subjects [mean (±SD) age, 47.1 (±18.2) years; 46.4 % males] who participated in a population-based survey on prevalences of hypertension, hyperglycemia, and hyperlipidemia in Taiwan. CRP levels were measured by the immunoturbidimetric CRP-latex high-sensitivity assay. The MetS was defined by an unified criteria set by several major organizations. Odds ratios (ORs) and 95 % confidence intervals (CIs) were calculated with logistic regression model. Overall, there were 938 subjects with MetS among 4,234 participants, resulting in a prevalence rate of 22.1 %. A significantly progressive increase in the prevalence of MetS across quartiles of CRP was observed (p for trend <0.001). Participants in the second, third, and upper quartiles of CRP had significantly higher risk of having MetS when compared with those in the lowest quartile [adjusted ORs (95 % CIs) were 2.18 (1.62–2.94), 4.39 (3.31–5.81), and 7.11 (5.39–9.38), respectively; p for trend <0.001]. Furthermore, there was a strong stepwise increase in CRP levels as the number of components of the MetS increased. The prevalence of MetS showed a graded increase according to CRP concentrations. The possible utility of CRP concentration as a marker for MetS risk awaits further evaluation in prospective studies.

Keywords Chinese · C-reactive protein · Cross-sectional study · Inflammation · Metabolic syndrome

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Introduction

The metabolic syndrome (MetS) comprises a constellation of various metabolic abnormalities, including hypertension, dyslipidemia, diabetes mellitus or impaired glucose tolerance, and obesity [1], and is closely related to cardiovascular morbidity and mortality in the general population [2, 3]. Increasing evidence suggests that chronic, low-grade inflammation may be a common soil involving the pathogenesis of MetS and cardiovascular disease [4]. Recent studies in the United States [5] and Mexico [6] have suggested that C-reactive protein (CRP), an extensively studied biomarker of low-grade inflammation, might have an effect on the development of MetS. Although underlying mechanisms linking elevated CRP to these disorders are not known, it is possible that their association is partly mediated by adipose tissue, an important source of circulating inflammatory cytokines [7]. Several epidemiological studies in Asian populations, including the Chinese population in Taiwan, have shown that Asians have higher amounts of body fat at lower body mass indexes (BMIs) than do Western populations [8, 9]. Furthermore, a strong association between CRP and overweight/obesity status have been found in epidemiological studies [7]. Given these circumstances, we investigated the epidemiological association of CRP concentration with the components of the MetS and their clustering in a representative sample of Chinese adults in Taiwan. We further evaluated the joint effect of CRP concentration and overweight status on the prevalence rate of MetS.

Materials and methods

Subjects

The study population consisted of subjects who participated in the second wave of the Taiwanese Survey on Prevalences of Hypertension, Hyperglycemia, and Hyperlipidemia (TwSHHH-II). The study design and study population of the original TwSHHH have been reported elsewhere [10, 11]. The initial TwSHHH was conducted in 2002 based on a multistage random sample of the civilian, non-institutionalized population in Taiwan. In total, 10,292 individuals were randomly selected for the TwSHHH. Of these 10,292 subjects, 7,578 (73.6 %) completed a questionnaire and 6,600 (64.1 %) permitted additional blood pressure measurement and blood examination for biomarkers. These 6,600 individuals who completed all examinations in the TwSHHH were eligible for the TwSHHH-II. Among them, 242 subjects had died and 581 persons could not be contacted. The remaining 5,777 individuals were invited to participate in the TwSHHH-II. Accordingly, a total of 4,682 persons were enrolled in the TwSHHH-II, resulting in a response rate of 81.0 %. Differences in sex and age distributions were not statistically significant between participants and nonparticipants in the TwSHHH-II. The protocols for the TwSHHH and TwSHHH-II were approved by the Institutional Review Board at the Bureau of Health Promotion, Department of Health, Executive Yuan in Taiwan. Written informed consent was obtained from all participants in the TwSHHH and the TwSHHH-II.

Measurements

At study entry, participants underwent baseline demographic data collection and anthropometric measurements, including body weight, height, and waist circumference, by well-trained nurses under a standardized protocol. BMI was calculated as weight divided by height squared (kg/m^2). Arterial blood pressure was also taken from each participant using an electric sphygmomanometer (BP3AC1-1, Microlife Cooperation, Berneck, Switzerland). The electric sphygmomanometer has been validated according to the international protocol published by the European Society of Hypertension [12]. In this study, well-trained nurses measured the systolic blood pressure (SBP) and diastolic blood pressure (DBP) two times in the left arm of seated participants according to a standardized protocol. A third blood pressure measurement was made if the first two blood pressure readings differed by more than 10 mmHg. The average of the two closest readings was calculated to determine the reported blood pressure for each participant.

A blood sample was collected into an EDTA anticoagulant tube for each participant after a 12-h overnight fast. Standard enzymatic methods were used to determine serum cholesterol and triglycerides. Electrophoresis was performed to measure high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C). Fasting plasma glucose (FPG) was measured by the hexokinase glucose-6-phosphate dehydrogenase procedure. In addition, CRP levels were assessed by the immunoturbidimetric CRP-latex high-sensitivity assay from Denka Seiken (Tokyo, Japan) performed according to the manufacturer's protocol. This assay has been validated against the Dade Behring method (Deerfield, Ill) [13]. The coefficients of variation of these measurements were $\sim$5 %. All biochemical tests were performed using automatic analyzers (TBA-200FR, Toshiba Corporation, Tokyo, Japan). All measurements were taken with blinded quality control specimens in the central laboratory.

Definitions

In this study, MetS was defined according to the criteria set by a joint statement of the International Diabetes Federation

Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; Word Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity [1]. Accordingly, three of the following five criteria were grounds for definition: (1) elevated blood pressure: blood pressure of at least 130/85 mmHg or use of antihypertensive medications, (2) hypertriglyceridemia: serum triglycerides of at least 150 mg/dL or use of drug treatment for elevated triglycerides, (3) reduced HDL-C: HDL-C $<$ 40 mg/dL in men and $<$50 mg/dL in women or use of drug treatment for reduced HDL-C, (4) hyperglycemia: FPG of 100 mg/dL or more or use of drug treatment of elevated glucose, and (5) central obesity: waist circumference $\geqq$90 cm in men and $\geqq$80 cm in women. In addition, a BMI-based categorization of adiposity modified for Chinese adults [14] was used in this study. Namely, normal weight was defined as 22.0 kg/m^2 $\leqq$ BMI $\leqq$ 23.9 kg/m^2 and overweight was indicated by BMI $\geqq$ 24.0 kg/m^2. In this study, habitual cigarette smoking was defined as smoking cigarettes at least once a week for more than 6 months. Similarly, regular alcohol intake was defined as consuming any alcoholic beverage at least once a week for more than 6 months.

Statistical analysis

In the study population, distributions of anthropometric and blood pressure measurements and levels of blood lipids, triglyceride, FPG, and CRP were not normally distributed; therefore, median values with interquartile range are presented. The difference in medians between the MetS and non-MetS groups was examined using the Wilcoxon rank sum test. Spearman correlation analyses were performed between CRP and individual components of the MetS. In the association analysis, CRP concentration was divided into quartiles and the lowest quartile was set as a reference group. Nevertheless, the CRP level was classified into tertiles in the analysis of the joint effect of CRP concentration and overweight status on the prevalence rate of MetS for the sake of sample size consideration. The associations between CRP concentration and individual components of the MetS and MetS itself were examined from the logistic regression model, with the calculation of odds ratios (ORs) and their 95 % confidence intervals (CIs). Given considerations that the National Cholesterol Education Program Adult Treatment Panel III (NCEP/ATP III) guidelines consider reducing LDL-C to be primary prevention for cardiometabolic disorders [15], cigarette smoking and alcohol consumption have been known to influence CRP levels [16, 17], and there are collinearity issues between studied covariates and components of the MetS (such as serum total cholesterol and HDL-C, BMI, and waist circumference), potential confounding variables included in the analysis were age, sex, status of habitual cigarette smoking and regular alcohol intake, and LDL-C concentration. In this study, likelihood ratio tests were used to examine statistical interactions between CRP and overweight status on the prevalence of MetS by comparing -2 log likelihood χ^2 between nested models with and without the cross-product terms. Data management and statistical analyses were performed with SAS version 9.2 (SAS Institute, Cary, NC, USA), and all of the statistical tests were 2-tailed with an α level of 0.05.

Results

In this study, participants without data on SBP, DBP, FPG, triglycerides, waist circumference, and BMI ($n = 311$), and those who had a CRP concentration above 10 mg/L ($n = 137$), a level signaling an acute-phase inflammation, were excluded from the analyses. The final analytic sample included 4,234 participants with a mean ($\pm$SD) age of 47.1 ($\pm$18.2) years, and 46.4 % were males. Overall, there were 938 subjects (435 males and 503 females) with MetS in the total population, resulting in a prevalence rate of 22.1 % (males: 22.1 %, females: 22.2 %). Table 1 shows the baseline characteristics for subjects with or without MetS. The MetS group was significantly older than the non-MetS group (median age was 56.4 and 44.5 years, respectively, $p < 0.001$). As expected, participants with the MetS had significantly higher levels of BMI, waist circumference, arterial blood pressure, lipid profile, triglycerides, FPG, and CRP than those without this syndrome ($p < 0.001$). In addition, the prevalences of habitual cigarette smoking and regular alcohol intake were significantly higher among people who had the MetS than those did not ($p = 0.004$ and $p < 0.001$, respectively). Furthermore, subjects with the MetS had significantly higher proportions of taking antihypertensive drugs, lipid-lowering medications, and hypoglycemic agents than those without the MetS ($p < 0.001$). In contrast, the level of HDL-C in the MetS group was significantly lower than that in the non-MetS group ($p < 0.001$).

Spearman's correlation coefficients between CRP concentration and the components of MetS and other cardiovascular risk factors are shown in Table 2. After adjustment for age, sex, cigarette smoking status, regular alcohol intake status, and level of LDL-C, there was a significantly positive correlation between CRP and each of SBP, DBP, triglycerides, fasting glucose, waist circumference, BMI, total cholesterol, and LDL-C ($p < 0.001$). However, there was a significantly negative correlation of CRP with HDL-C ($p < 0.001$). The strongest correlation was observed between CRP and adiposity measures, including BMI ($r = 0.31$) and waist circumference ($r = 0.30$).

Table 1 General characteristics of participants with or without MetS

Characteristics	MetS	Non-MetS	*p* value
Total (n = 4,234)	(n = 938, 22.2 %)	(n = 3,296, 77.8 %)	
Median (interquartile range)			
Age (years)	56.4 (20.9)	44.5 (22.0)	<0.001
BMI (kg/m^2)	27.0 (4.4)	22.9 (4.4)	<0.001
WC (cm)	92.0 (12.2)	79.0 (13.7)	<0.001
SBP (mmHg)	134.0 (20.0)	115.0 (19.0)	<0.001
DBP (mmHg)	82.0 (16.0)	73.0 (14.0)	<0.001
TC (mg/dL)	187.0 (55.0)	175.0 (46.0)	<0.001
LDL-C (mg/dL)	116.0 (46.0)	106.0 (41.0)	<0.001
HDL-C (mg/dL)	46.1 (10.7)	52.8 (13.5)	<0.001
TG (mg/dL)	169.0 (106.0)	90.0 (55.0)	<0.001
FPG (mg/dL)	98.0 (29.0)	84.0 (11.0)	<0.001
CRP (mg/L)	1.8 (2.4)	0.7 (1.3)	<0.001
No. (%)			
Male gender	435 (46.4)	1530 (46.4)	0.981
Habitual cigarette smoking	285 (30.5)	848 (25.7)	0.004
Regular alcohol drinking	172 (18.3)	406 (12.3)	<0.001
Anti-hypertensive drug use	348 (37.1)	268 (8.1)	<0.001
Lipid-lowering drug use	139 (14.8)	53 (1.6)	<0.001
Hypoglycemic agent use	189 (20.1)	58 (1.8)	<0.001

BMI body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *TC* total cholesterol, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triglyceride, *FPG* fasting plasma glucose, *CRP* C-reactive protein

Table 2 Spearman partial correlation coefficients (r_s) between CRP and components of MetS and other cardiovascular risk factors

Factor	Total (n = 4,234)	
	r_s	*p* value
Individual components of MetS		
SBP	0.17	<0.001
DBP	0.14	<0.001
HDL cholesterol	−0.17	<0.001
Triglyceride	0.15	<0.001
Glucose	0.12	<0.001
Waist circumference	0.30	<0.001
Other cardiovascular risk factors		
BMI	0.31	<0.001
Total cholesterol	0.14	<0.001
LDL cholesterol	0.13	<0.001

Spearman partial correlation coefficients were adjusted for age, sex, cigarette smoking status, regular alcohol intake status, and level of LDL cholesterol

HDL high-density lipoprotein, *LDL* low-density lipoprotein

Associations of CRP quartiles for MetS are presented in Table 3. After adjustment for age, sex, cigarette smoking status, regular alcohol intake status, and LDL-C concentration, a significantly stepwise increase in the prevalence of MetS across quartiles of CRP was observed in the study population (p for trend <0.001). Participants in the second, third, and upper quartiles of CRP had significantly higher risk of having MetS when compared with subjects in the lowest quartile [adjusted ORs (95 % CI) were 2.18 (1.62–2.94), 4.39 (3.31–5.81), and 7.11 (5.39–9.38), respectively.]. This positive gradient for prevalence of MetS across quartiles of CRP was also observed in both men and women.

Furthermore, the associations of CRP concentration with individual components of the MetS are shown in Table 4. Increased ORs for each of the five components of the MetS were observed from the 1st to the 4th CRP quartiles. Compared with participants in the lowest CRP quartile, those in the highest quartile had an OR (95 % CI) of 1.72 (1.37–2.16) for elevated blood pressure, 2.57 (2.04–3.25) for reduced HDL-C, 2.10 (1.64–2.70) for hypertriglyceridemia, 2.10 (1.56–2.84) for hyperglycemia, and 4.24 (3.41–5.27) for central obesity, respectively. As expected, there was a strong stepwise increase in CRP levels as the number of components of the MetS increased (p for trend <0.001) (Fig. 1). The CRP median for subjects with 0, 1, 2, 3, 4, or 5 components of the MetS were 0.48, 0.83, 1.30, 1.73, 2.20, and 3.21 mg/L, respectively.

We further evaluated the joint effect of CRP concentration and overweight status on the prevalence rate of MetS. As shown in Table 5, the risk of having MetS was more pronounced among overweight participants with increased CRP levels. In the normal weight group, participants with the highest CRP tertile had 90 % (95 % CI 1.11–3.27) higher risk for having MetS than those in the lowest CRP tertile after adjustment for potential confounders. Moreover, among the

Table 3 Association between quartiles of CRP and MetS

MetS	Quartile of CRP (mg/L)				
	*Q*1 <0.42	*Q*2 0.42–0.95	*Q*3 0.96–2.20	*Q*4 ≥2.21	p_{trend}
Total					
Absent	1,033	914	778	654	
Present	72	171	309	440	
OR (95 % CI)	1.00 (reference)	2.18 (1.62–2.94)	4.39 (3.31–5.81)	7.11 (5.39–9.38)	<0.001
Men					
Absent	401	440	398	335	
Present	37	84	150	186	
OR (95 % CI)	1.00 (reference)	1.85 (1.22–2.81)	3.57 (2.41–5.28)	5.04 (3.41–7.45)	<0.001
Women					
Absent	632	474	380	319	
Present	35	87	159	254	
OR (95 % CI)	1.00 (reference)	2.38 (1.55–3.66)	5.15 (3.42–7.75)	9.92 (6.64–14.82)	<0.001

ORs were adjusted for age, sex (for the total group), cigarette smoking status, regular alcohol drinking status, and LDL-C

OR odds ratio, *CI* confidence interval

Table 4 Association between quartile of CRP and individual components of the MetS

	Quartile of CRP (mg/L)				
	*Q*1 <0.42	*Q*2 0.42–0.95	*Q*3 0.96–2.20	*Q*4 ≥2.21	*p* for trend
Elevated blood pressure					
Absent	882	711	601	525	
Present	223	374	486	569	
OR (95 % CI)	1.00 (referent)	1.36 (1.09–1.70)	1.53 (1.23–1.91)	1.72 (1.37–2.16)	<0.001
Central obesity					
Absent	913	734	544	411	
Present	192	351	543	683	
OR (95 % CI)	1.00 (referent)	1.66 (1.34–2.06)	2.97 (2.40–3.67)	4.24 (3.41–5.27)	<0.001
Low HDL cholesterol					
Absent	922	847	806	709	
Present	183	238	281	385	
OR (95 % CI)	1.00 (referent)	1.58 (1.26–1.98)	1.82 (1.44–2.29)	2.57 (2.04–3.25)	<0.001
Hypertriglyceridemia					
Absent	983	839	739	675	
Present	122	246	348	419	
OR (95 % CI)	1.00 (referent)	1.59 (1.24–2.05)	1.98 (1.55–2.54)	2.10 (1.64–2.70)	<0.001
Hyperglycemia					
Absent	1,030	949	869	793	
Present	75	136	218	301	
OR (95 % CI)	1.00 (referent)	1.29 (0.94–1.76)	1.66 (1.23–2.25)	2.10 (1.56–2.84)	<0.001

ORs were adjusted for age, sex, cigarette smoking status, regular alcohol drinking status, LDL-C, and other components of the MetS shown in the table

OR odds ratio, *CI* confidence interval

overweight participants, those in the highest CRP tertile had a 3-fold OR for having MetS compared with those in the lowest CRP tertile [adjusted OR 13.99 (95 % CI, 8.75–22.37) for the third tertile vs. adjusted OR 4.41 (95 % CI 2.74–7.09) for the first tertile]. The likelihood ratio test for interaction between CRP concentration and overweight

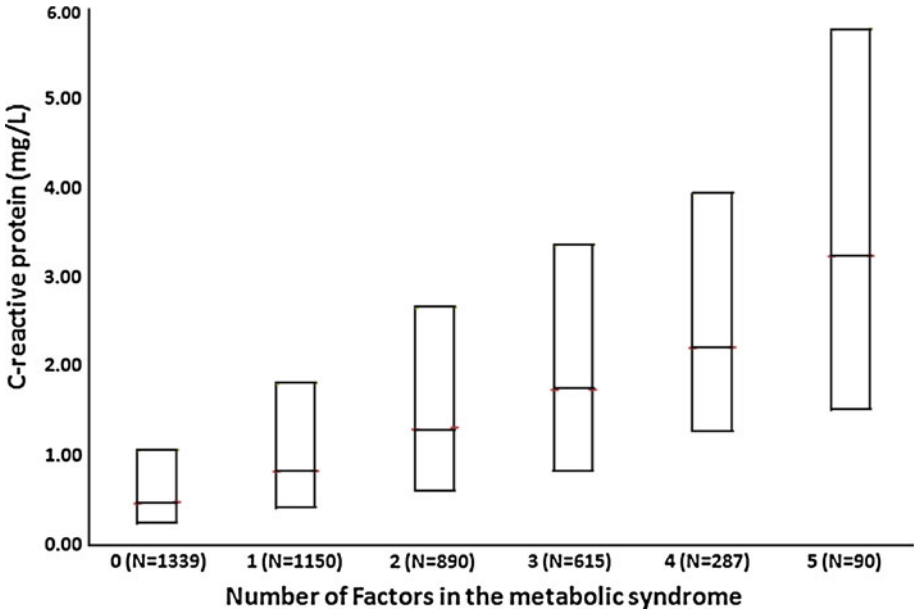

Fig. 1 Distribution of CRP levels among 4,371 Chinese adults according to the presence of the number of components in the MetS. *Box plots* display median, 25th, and 75th percentile values for CRP

Table 5 Adjusted ORs for MetS in relation to obesity status and serum CRP concentration

Obesity status	CRP concentration	No. of subjects	MetS	
			No. (%)	OR (95 % CI)
BMI 22.0–23.9 kg/m^2	T1 (<0.53 mg/L)	311	22 (7.1)	1.00 (reference)
	T2 (0.53–1.33 mg/L)	305	29 (9.5)	1.21 (0.67–2.19)
	T3 (>1.33 mg/L)	308	50 (16.2)	1.90 (1.11–3.27)
BMI ≧ 24.0 kg/m^2	T1 (<0.95 mg/L)	694	184 (26.5)	4.41 (2.74–7.09)
	T2 (0.95–2.30 mg/L)	689	293 (42.5)	8.95 (5.60–14.30)
	T3 (>2.30 mg/L)	699	371 (53.1)	13.99 (8.75–22.37)
p for trend				<0.001

ORs were adjusted for age, sex, cigarette smoking status, regular alcohol drinking status, and LDL-C

CI confidence interval, *BMI* body mass index

status in the risk of having MetS was statistically significant ($p = 0.038$).

Discussion

In this study, elevated CRP concentration had a significantly positive association with each MetS component and MetS itself in a representative sample of Chinese adult population. Of particular note, the risk of having MetS was more pronounced among overweight participants with increased CRP levels.

That CRP levels correspond with individual components of the MetS and MetS itself in this study is in line with the results of previous reports [5, 6, 18–22]. The underlying mechanisms for the apparent association between CRP and MetS are unclear, but several explanations are possible. Several lines of experimental evidence implicate CRP in inducing endothelial cell activation and dysfunction [23, 24], and it is well established that individual components of the MetS are related to endothelial dysfunction [25–27]. In addition, the proinflammatory effects of CRP have been documented in this study by the positive correlations between CRP concentration and serum levels of total cholesterol, LDL-C, and triglycerides as well as an inverse association between CRP and HDL-C. Furthermore, CRP has been shown to result in increased superoxide production as a result of enhanced NADPH oxidase activity in endothelial cells and in human peripheral blood monocytes [28, 29]. Coherently, oxidative stress, mainly superoxide, plays a critical role in the pathogenesis of MetS parameters [4]. Moreover, insulin resistance (IR) is thought to be the central feature of MetS [30] and IR is associated with a state of chronic, low-grade inflammation [31]. Indeed, previous studies have shown that individuals with a high level of CRP were at higher risk of IR than those with a low level of CRP [32]. Further, Karnchanasorn et al. [33] documented that IR was significantly associated with three components of MetS, namely, elevated triglycerides, low HDL-C, and abnormal glucose. Thus, CRP might be an

effective biomarker for the metabolic status and potential low-grade inflammatory state. Taken together, laboratory and epidemiological data have provided a strong relationship between CRP and MetS.

Of particular note, this study indicated a low overall CRP level in Chinese people; the median level (interquartile range) was 0.95 (1.80) mg/L and 75 % of our study population had a CRP level below 2.2 mg/L (upper threshold of the third quartile). Indeed, previous studies have documented that CRP levels were lower in Chinese compared with Caucasian populations [34, 35]. The reason why Chinese people have lower CRP levels than Western populations is unclear. Genetic diversity was reported to influence CRP levels [36]. In addition, several epidemiological studies have shown that Asians and Chinese have lower BMIs than Caucasians [8, 9]. Relatively low BMI may also modulate the CRP concentration. More importantly, participants with a low level of CRP in this study tended to have higher triglycerides and lower HDL-C (Table 4). This study provides additional evidence that even mildly elevated CRP from a low baseline level was associated with an increased risk for the presence of MetS.

Findings from this study revealed that the strongest correlation between CRP and individual components of the MetS was observed between CRP and waist circumference. Indeed, previous studies have shown a positive association between CRP and obesity [7, 19, 37]. It has been known that adipose tissue produces interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) [38, 39]. In addition, TNF-α has been reported to be a potent inducer of IL-6 [38], which is an important determinant of hepatic CRP synthesis [40]. These facts may explain the positive association between CRP and obesity. Moreover, BMI-based categorization of adiposity was found to be an important mediator for the association between CRP and the MetS in this Chinese population (Table 5). Accordingly, the inflammatory process may be more enhanced by obesity through adipose tissue-derived cytokine expression and may be more strongly related to the pathophysiological process leading to MetS. Further studies are needed to elucidate mechanisms responsible for the obesity-mediated association between CRP and MetS.

Recent studies have suggested that chronic inflammation, as indicated by elevated CRP concentration, might have a greater effect on the development of MetS in women than in men [5, 6]. However, no gender differences were observed for the relationship of CRP with the MetS in this study and a previous investigation of Chinese people in China [22]. The gender difference in the relationship of CRP with the MetS may be ethnicity specific. Future studies are warranted to clarify whether inflammation has a greater effect on MetS in women than in men among different populations, especially among Asians.

We acknowledge certain limitations to this study. Admittedly, with the cross-sectional nature of this study, it is not possible to establish a causal relationship between CRP and MetS. This study could not eliminate the potential effects of underlying diseases and medications used for these diseases among participants on the present findings. Further population-based prospective studies are needed to elucidate a cause–effect relationship between CRP concentration and MetS. In addition, IR has been considered to be a significant risk factor for MetS [30]. However, we did not have information on the degree of IR among subjects, which might have affected study results and is a study limitation. Furthermore, CRP levels were assessed from a single blood sample in this study and therefore intra-individual variation cannot be taken into account. However, such variation will likely result in an underestimation and previous work found that CRP levels are stable over long periods and have no diurnal variation [41, 42]. More positively, high-sensitivity CRP was measured in this study. Standard clinical assays for CRP typically lack sensitivity within the low-normal range and cannot be used effectively for cardiometabolic risk prediction [13]. In contrast, high-sensitivity CRP assays provide an adjunctive method for global assessment of cardiovascular risk [43].

In conclusion, previous studies have confirmed clustering of established MetS components and revealed additional associated cardiovascular risk factors in this Chinese population in Taiwan [44]. This study demonstrated that a positive gradient for prevalence of MetS was observed across quantitative levels of CRP. These findings suggest that MetS is associated with a systemic low-grade inflammation. Early identification and treatment of the modifiable risk factors for cardiometabolic disorders may be of utmost importance in prevention efforts.

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Conflict of interest The authors declare no conflict of interest.

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