Higher serum carotenoid concentrations associated with a lower prevalence of the metabolic syndrome in middle-aged and elderly Chinese adults

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Abstract

The association between serum carotenoids and the metabolic syndrome (MetS) remains uncertain, and little is known about this relationship in the Chinese population. The present study examined the association between serum carotenoid concentrations and the MetS in Chinese adults. We conducted a community-based cross-sectional study in which 2148 subjects (1547 women and 601 men) aged 50–75 years were recruited in urban Guangzhou, China. Dietary data and other covariates were collected during face-to-face interviews. Blood pressure, waist circumference, blood lipids, glucose and serum carotenoids (α-, β-carotene, β-cryptoxanthin, lycopene and lutein/zeaxanthin) were examined. We found dose–response inverse relationships between individual serum carotenoid concentrations and total carotenoids and the prevalence of the MetS after adjusting for potential confounders (*P* for trend < 0·001). The OR of the MetS for the highest (*v.* lowest) quartile were 0·31 (95 % CI 0·20, 0·47) for α-carotene, 0·23 (95 % CI 0·15, 0·36) for β-carotene, 0·44 (95 % CI 0·29, 0·67) for β-cryptoxanthin, 0·39 (95 % CI 0·26, 0·58) for lycopene, 0·28 (95 % CI 0·18, 0·44) for lutein + zeaxanthin and 0·19 (95 % CI 0·12, 0·30) for total carotenoids. Higher concentrations of each individual carotenoid and total carotenoids were significantly associated with a decrease in the number of abnormal MetS components (*P* for trend < 0·001–0·023). Higher serum carotenoid levels were associated with a lower prevalence of the MetS and fewer abnormal MetS components in middle-aged and elderly Chinese adults.

Key words: Carotenoids: Serum: Metabolic syndrome: Cross-sectional studies

The metabolic syndrome (MetS) is a cluster of independent risk factors that increase the risk of CVD and type 2 diabetes[1,2]. Existing data indicate that the incidence of the MetS is rising at an alarming rate globally[3]. In China, the MetS has become an increasing public health problem due to the changes in lifestyle brought about by economic development[4,5]. Although the exact mechanism of the MetS is still being elucidated, a growing body of evidence suggests that oxidative stress plays a considerable role in its pathogenesis[6–8]. Carotenoids are well known for antioxidant activities including quenching free radicals, reducing damage from reactive oxygen species and inhibiting lipid peroxidation[9–11]. The Coronary Artery Risk Development in Young Adults study suggested that carotenoids could prevent obesity by decreasing oxidative stress generated by adipose tissue[12]. Furthermore, carotenoids might inhibit pro-inflammatory cytokine and chemokine production, and then decrease the risk of obesity-associated pathologies, such as insulin resistance[13,14]. Thus, it was assumed that carotenoids might protect against the MetS.

An inverse association of serum carotenoids with the prevalence of the MetS has been shown in three cross-sectional studies conducted in the USA[15–17]. However, in a cross-sectional study with 1073 Japanese adults, Sugiura *et al.*[18] found the inverse relationship only in current smokers. Such a favourable association was not found in another cross-sectional study conducted among current male Japanese smokers[19] and in an Australian study[20]. A randomised controlled trial (RCT) has suggested that dietary supplementation with β-carotene had no significant effect on the incidence of the MetS[21]. The discrepancies in the results found among these studies might be attributable to the differences in study populations and designs, exposure makers and covariate adjustments. Moreover, significant ethnic differences in the associations between serum carotenoids and obesity have been reported, in which much weaker associations have been observed in black than in white participants[12]. The prevalence of the MetS was much lower in the Chinese (13·7 %) than in the Caucasian (> 25 %) population[22,23]. Apart from genetic factors[24], many environmental factors, such as diet, and other lifestyle factors might

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contribute to these differences[25,26]. Therefore, ethnic differences in the associations between carotenoids and the MetS could not be excluded, and the results obtained in other populations might not be well generalised to the Chinese population.

In the present cross-sectional study, we investigated the relationships between the major serum carotenoids (α-, β-carotene, β-cryptoxanthin, lycopene and lutein/zeaxanthin) and the prevalence of the MetS in middle-aged and elderly Chinese adults.

Methods

The present community-based cross-sectional study enrolled 3169 subjects aged 50–75 years in Guangzhou, Guangdong Province, China, from October 2008 to June 2010. Participants were required to have lived in Guangzhou for at least 5 years, and were recruited from communities in Guangzhou through advertisements, health talks and referrals. We excluded the participants who reported having confirmed cancer, CHD, stroke, Alzheimer's disease or dementia before the start of the study, or who had used medication (e.g. statin, anti-hypertension or anti-diabetic drugs) known to affect plasma lipids in the previous 3 months (*n* 201). We further excluded the participants who had missing or incomplete dietary data, had implausibly high ($\geq 16\,736$ kJ; ≥ 4000 kcal) or low (≤ 1674 kJ; ≤ 400 kcal) total daily energy intakes (*n* 14) and had missing carotenoid data (*n* 806). Finally, 2148 participants were included in the analysis. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the Ethics Committee of the School of Public Health of Sun Yat-sen University. Written informed consent was obtained from all the study participants.

Data collection

Questionnaire interview

A face-to-face interview was conducted by trained interviewers using a structured questionnaire. We collected information on the participants' sociodemographic characteristics, medication, lifestyle factors, history of disease, menopausal status (for women only), dietary intake and physical activity. Daily physical activity was estimated using a physical activity questionnaire containing nineteen items, and the metabolic equivalent intensity (excluding sleeping and sitting) was calculated[27]. Usual dietary intake was assessed by using a validated quantitative FFQ including seventy-nine items. The correlation coefficient for β-carotene was 0·55 for the short-term reproducibility, and that between the second FFQ and the multiple 24 h dietary records was 0·32[28]. Daily energy intake and other nutrients were calculated on the basis of the Chinese Food Composition Table 2004[29].

Measurements of anthropometric indices and blood pressure

Body weight and height were measured with participants wearing no shoes and light clothing. BMI was calculated as weight (kg)/height^2 (m^2). Waist circumference was measured at the mid-line between the costal margin and the iliac crest, while hip circumference was measured at the point of the maximum girth around the buttocks. Measurements of waist and hip circumferences were made twice and the mean of the two measurements was used for further analysis. Overall, two consecutive blood pressure (BP) measurements were taken from the right arm after the participants were seated for at least 10 min. BP (systolic and diastolic) was recorded to the nearest 2 mmHg. A third measurement was taken when the discrepancy between the two measured values was larger than 4 mmHg. The mean measurements were used for subsequent analyses.

Laboratory assay

Blood samples were obtained in the morning after an overnight fast. Serum was separated after centrifugation at 1500 *g* for 15 min at 4°C within 2 h and stored at −80°C until routine chemical analysis by standard methods. Serum total cholesterol, TAG, LDL-cholesterol, HDL-cholesterol (HDL-C) and fasting blood glucose concentrations were measured by colorimetric methods using commercial kits (Biosino Biotechnology Company Limited) on a Hitachi 7600-010 automated analyser. The CV was 2·17 % (5·03 mmol total cholesterol/l), 2·86 % (1·14 mmol TAG/l), 3·47 % (1·70 mmol HDL-C/l), 4·67 % (2·65 mmol LDL-cholesterol/l) and 2·52 % (4·45 mmol/l fasting blood glucose).

We used reversed-phase HPLC to assay simultaneously the levels of α-tocopherol, retinol, α-carotene, β-carotene, β-cryptoxanthin, lycopene and lutein/zeaxanthin, according to the method described by Burri *et al.*[30] with some modifications. In brief, 200 μl of serum sample were mixed with 500 μl of ethanol–butylated hydroxytoluene solution containing α-tocopherol acetate as the internal standard, and then mixed for 1 min on an orbital shaker. The carotenoids were extracted twice using 2 ml of hexane–butylated hydroxytoluene solution. The hexane layer was separated and evaporated to dry under N$_2$ at room temperature, and the residue was dissolved in 200 μl of the mobile phase B (acetonitrile–methanol–tetrahydrofuran–ammonium acetate 55:35:5:5, v/v). Then, 50 μl of samples were injected into a C$_{18}$ analytical column (Shiseido) at room temperature. Peaks were detected at a wavelength of 450 nm for the carotenoids by a Waters 2998 diode-array detector (Waters).

As these methods do not discriminate lutein from zeaxanthin, we used the combined concentration of lutein + zeaxanthin in the analyses. A pooled plasma sample was analysed with batches of study samples to monitor the analytic precision, with a day-to-day CV of approximately 7·8 % for α-carotene, 8·6 % for β-carotene, 9·7 % for β-cryptoxanthin, 10·6 % for lycopene and 8·0 % for lutein + zeaxanthin, respectively.

Definition of the metabolic syndrome

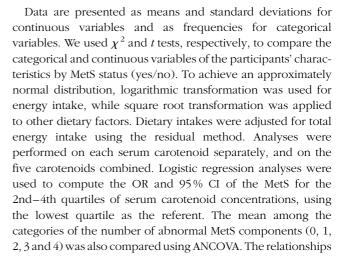The diagnostic criteria for the MetS are based on the 2005 definition of the International Diabetes Federation[31]:

(1) abdominal obesity – waist circumference ≥90 cm in men or ≥80 cm in women; (2) elevated TAG – TAG ≥1·7 mmol/l; (3) low HDL-C – HDL-C <1·03 mmol/l in men or <1·30 mmol/l in women; (4) elevated BP – BP ≥130/85 mmHg or current use of anti-hypertensive medications; (5) elevated fasting glucose – fasting blood glucose ≥5·6 mmol/l, or previously diagnosed type 2 diabetes, or taking oral anti-diabetic agents or insulin.

Statistical analyses

Sample size was estimated based on the prevalence (13·7 %) of the MetS in China in 2005[4] and the reported OR (0·76) of the MetS for the highest (v. lowest) quartile of serum carotenoids[20]. In each group, 537 subjects would provide 80 % power to detect the association (OR 0·76) at the significance of 0·05.

Data are presented as means and standard deviations for continuous variables and as frequencies for categorical variables. We used χ^2 and t tests, respectively, to compare the categorical and continuous variables of the participants' characteristics by MetS status (yes/no). To achieve an approximately normal distribution, logarithmic transformation was used for energy intake, while square root transformation was applied to other dietary factors. Dietary intakes were adjusted for total energy intake using the residual method. Analyses were performed on each serum carotenoid separately, and on the five carotenoids combined. Logistic regression analyses were used to compute the OR and 95 % CI of the MetS for the 2nd−4th quartiles of serum carotenoid concentrations, using the lowest quartile as the referent. The mean among the categories of the number of abnormal MetS components (0, 1, 2, 3 and 4) was also compared using ANCOVA. The relationships

Table 1. Characteristics of the study participants*

(Mean values and standard deviations)

	MetS⁻† (1903)		MetS⁺‡ (245)		
	Mean	SD	Mean	SD	P
Age (years)	57·4	5·0	58·6	4·9	<0·001
BMI (kg/m²)	22·9	2·9	25·9	2·7	<0·001
Waist circumference (cm)	81·3	8·7	90·6	6·8	<0·001
Systolic blood pressure (mmHg)	122	17	136	17	<0·001
Diastolic blood pressure (mmHg)	77	10	87	10	<0·001
Physical activity (MET-h/d)§	22·8	6·5	21·0	5·2	0·203
Serum lipids (mmol/l)					
Fasting blood glucose	4·62	0·80	5·24	1·34	<0·001
TAG	1·45	0·92	2·18	1·29	<0·001
Total cholesterol	5·45	1·09	5·41	1·01	0·564
HDL-cholesterol	1·42	0·33	1·12	0·21	<0·001
LDL-cholesterol	3·63	0·92	3·63	0·90	0·956
Serum antioxidant (μmol/l)					
α-Tocopherol	33·3	11·1	35·6	14·0	0·003
Retinol	2·35	0·66	2·45	0·61	0·014
α-Carotene	0·044	0·033	0·033	0·022	<0·001
β-Carotene	0·622	0·455	0·437	0·313	<0·001
β-Cryptoxanthin	0·105	0·097	0·080	0·067	<0·001
Lycopene	0·110	0·076	0·097	0·076	0·011
Lutein + zeaxanthin	0·484	0·240	0·415	0·231	<0·001
Total carotenoids‖	1·37	0·73	1·06	0·56	<0·001
	n	%	n	%	
Education (years)					<0·001
<9	514	27·0	96	39·2	
9−12	913	48·0	98	40·0	
>12	476	25·0	51	20·8	
Household income (yuan/month per person)					0·337
<2000	645	33·9	72	29·4	
2000−3000	892	46·9	125	51·2	
>3000	366	19·3	48	19·4	
Smoker¶	265	13·9	30	12·2	0·493
Alcohol drinking**	108	5·7	16	6·5	0·662

MetS, metabolic syndrome; MET, metabolic equivalent.
* Continuous and categorical variables were described by means and standard deviations or numbers and percentages, and evaluated by t test and χ^2 test, respectively, to compare the categorical and continuous variables of the participants with and without the MetS.
† Participants without the MetS.
‡ Participants with the MetS.
§ Physical activities included daily occupational, leisure-time and household chores, evaluated by MET-h/d.
‖ Total carotenoids indicate the sum of α-carotene, β-carotene, β-cryptoxanthin, lycopene and lutein + zeaxanthin.
¶ Smokers were defined as having smoked at least one cigarette daily for at least six consecutive months.
** Alcohol drinkers were defined as having had wine (beer, white wine and red wine) at least once per week for at least six consecutive months.

between the carotenoids and the MetS were examined after adjusting for age, sex and energy intake (model 1), and further controlling for other covariates (model 2), including marital status, education level, occupation (light, moderate and heavy physical activity), household income, smoking status, alcohol drinking, physical activity, dietary intakes of fibre, cholesterol, SFA, vitamin C, α-tocopherol and β-carotene, and serum levels of retinol and α-tocopherol. *P* for the linear trend was estimated by treating quartiles or category number as continuous variables. We performed subgroup analyses, respectively, stratified by age, sex, alcohol drinking, smoking status, education level and household income, and tested their interactions. Considering type I error caused by multiple comparisons, significance levels were adjusted by using Bonferroni correction ($\alpha = 0.05$/number of tests). All statistical analyses were performed using SPSS for Windows (version 13.0; SPSS, Inc.).

Results

In the present community-based cross-sectional study, the prevalence of the MetS was estimated as 11·4 %. The demographic and health-related characteristics are presented in Table 1. As expected, participants with the MetS had elevated BMI, waist circumference, BP, TAG and fasting blood glucose and lower HDL-C concentrations than those without the MetS ($P < 0.001$). Significant differences were also evident by age group and educational level ($P < 0.001$). In addition, participants with the MetS had lower serum carotenoids but higher retinol and α-tocopherol concentrations than those without the MetS (*P* range <0.001–0.014). No difference was detected

in the remaining characteristics (e.g. household income, smoking status and alcohol drinking) of the participants.

Consistent inverse associations of each individual level and total amount of the five carotenoids were observed with the prevalence of the MetS in the two models (Table 2). In the maximum-adjusted model, OR of the MetS for the highest (*v.* lowest) quartile were 0·31 (95 % CI 0·20, 0·47) for α-carotene, 0·23 (95 % CI 0·15, 0·36) for β-carotene, 0·44 (95 % CI 0·29, 0·67) for β-cryptoxanthin, 0·39 (95 % CI 0·26, 0·58) for lycopene, 0·28 (95 % CI 0·18, 0·44) for lutein + zeaxanthin and 0·19 (95 % CI 0·12, 0·30) for total carotenoids (all *P* for trend <0.001). Base on the study size analysed, we had $\geq 86\%$ power to detect the associations (OR ≤ 0.44) at an α value of 0·008 (= 0·05/six tests). The covariate-adjusted means of serum carotenoid concentrations by the number of abnormal MetS components are shown in Table 3. The serum levels of each individual carotenoid and their combination decreased significantly as the number of abnormal MetS components increased (*P* for trend range <0.001–0.023).

We examined the association between carotenoids and the MetS across different subgroups stratified by sex, smoking status and alcohol drinking (Table 4). Inverse associations between the MetS and total carotenoids and each individual carotenoid were observed only among non-smokers or non-drinkers. No significant interactions were found between carotenoids and age, sex, alcohol drinking, smoking status, education level and household income (see online supplementary Table S1), as they were related to the prevalence of the MetS (*P* for interaction >0.003, $\alpha = 0.05$/eighteen tests).

Table 2. Metabolic syndrome by the quartiles of serum carotenoid concentrations

(Odds ratios and 95 % confidence intervals; *n* 537)

	Quartile 1	Quartile 2		Quartile 3		Quartile 4		
	OR	OR	95 % CI	OR	95 % CI	OR	95 % CI	*P* for trend
α-Carotene (μmol/l)	0·015	0·027		0·042		0·086		
Case (*n*)	85	72		54		34		
Model 1*	1·00	0·80	0·56, 1·12	0·54	0·38, 0·79	0·33	0·21, 0·50	<0·001
Model 2†	1·00	0·81	0·58, 1·15	0·55	0·38, 0·80	0·31	0·20, 0·47	<0·001
β-Carotene (μmol/l)	0·18	0·38		0·62		1·22		
Case (*n*)	98	63		49		35		
Model 1	1·00	0·55	0·39, 0·78	0·39	0·27, 0·57	0·26	0·17, 0·40	<0·001
Model 2	1·00	0·52	0·37, 0·75	0·36	0·24, 0·53	0·23	0·15, 0·36	<0·001
β-Cryptoxanthin (μmol/l)	0·032	0·055		0·095		0·226		
Case (*n*)	81	65		58		41		
Model 1	1·00	0·71	0·50, 1·01	0·62	0·43, 0·89	0·41	0·27, 0·61	<0·001
Model 2	1·00	0·74	0·52, 1·08	0·68	0·47, 0·99	0·44	0·29, 0·67	<0·001
Lycopene (μmol/l)	0·041	0·075		0·110		0·208		
Case (*n*)	89	57		51		48		
Model 1	1·00	0·54	0·38, 0·78	0·46	0·32, 0·67	0·43	0·29, 0·63	<0·001
Model 2	1·00	0·56	0·39, 0·80	0·45	0·31, 0·67	0·39	0·26, 0·58	<0·001
Lutein + zeaxanthin (μmol/l)	0·22	0·37		0·51		0·81		
Case (*n*)	89	61		57		38		
Model 1	1·00	0·63	0·44, 0·90	0·58	0·41, 0·84	0·37	0·25, 0·56	<0·001
Model 2	1·00	0·59	0·41, 0·85	0·50	0·34, 0·74	0·28	0·18, 0·44	<0·001
Total carotenoids (μmol/l)	0·58	0·98		1·43		2·34		
Case (*n*)	101	59		52		33		
Model 1	1·00	0·49	0·34, 0·69	0·36	0·24, 0·52	0·26	0·18, 0·40	<0·001
Model 2	1·00	0·44	0·31, 0·64	0·35	0·24, 0·53	0·19	0·12, 0·30	<0·001

* Model 1 adjusted for age, sex and energy.
† Model 2 further adjusted for marital status, education level, occupation, household income, smoking status, alcohol drinking, physical activity, serum total cholesterol, dietary intakes of fibre, cholesterol, SFA, vitamin C, vitamin E and β-carotene, and serum levels of retinol and α-tocopherol.

Table 3. Covariate-adjusted mean of serum carotenoid concentrations by number of metabolic syndrome components
(Mean values with their standard errors)

	Number of metabolic syndrome components											
	0		1		2		3		4		P for trend	P for difference†
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM		
n	617		748		531		217		35			
α-Carotene (μmol/l)	0.043	0.004	0.040	0.004	0.033***	0.004	0.028***	0.004	0.022**	0.006	<0.001	<0.001
β-Carotene (μmol/l)	0.655	0.045	0.548***	0.044	0.445***	0.045	0.365***	0.049	0.365***	0.079	<0.001	<0.001
β-Cryptoxanthin (μmol/l)	0.103	0.010	0.093	0.009	0.078***	0.010	0.066***	0.011	0.070	0.017	0.002	<0.001
Lycopene (μmol/l)	0.115	0.008	0.099**	0.008	0.094***	0.008	0.088***	0.009	0.091	0.015	0.023	<0.001
Lutein + zeaxanthin (μmol/l)	0.481	0.024	0.439**	0.024	0.399***	0.027	0.370***	0.029	0.324***	0.043	<0.001	<0.001
Total carotenoids (μmol/l)	1.397	0.070	1.219***	0.068	1.049***	0.070	0.917***	0.076	0.872***	0.124	<0.001	<0.001

Mean value was significantly different from that of the zero number of metabolic syndrome components: ** $P < 0.01$, *** $P < 0.001$ (Bonferroni's test).

† P for group difference was obtained from ANCOVA. Covariates adjusted for the following factors: age, sex and energy; marital status; education level; occupation; household income; smoking status; alcohol drinking; physical activity; serum total cholesterol; dietary intakes of fibre, cholesterol, SFA, vitamin C, vitamin E and β-carotene; serum levels of retinol and α-tocopherol.

Discussion

We evaluated the relationships between serum carotenoids and the MetS in a sample of middle-aged and elderly Chinese adults and found a dose–response inverse linear association between carotenoids and the prevalence of the MetS. Furthermore, the concentration of carotenoids decreased significantly as the number of abnormal MetS components increased.

In agreement with our findings, several observational studies have found serum carotenoid concentrations to be inversely associated with the MetS[15,20]. The Third National Health and Nutrition Examination Survey (1988–94) of 8808 adults reported that those with the MetS had significantly lower concentrations of individual carotenoids (except lycopene) and total carotenoids compared with those without the MetS, after adjusting for confounding factors similar to those in the present study[15]. They also showed decreasing levels of individual carotenoids (except lycopene) and total carotenoids as the number of MetS components increased[15]. Similar results have been obtained in other cross-sectional studies[20]. In a RCT involving 5220 adults taking a supplement containing a combination of antioxidants (vitamins C and E, β-carotene, Zn and Se), participants in the top tertile of baseline serum β-carotene levels had a 66 % (range 47–79 %) lower risk of developing the MetS (P for trend <0·001) than those in the bottom tertile after a follow-up period of 7·5 years[21]. However, the RCT did not observe any long-term benefits or adverse effects of β-carotene in the prevention of the MetS[21]. There are several possible explanations for the discrepancy. The population of the intervention study may have had a higher prevalence of risk factors such as smoking status and alcohol drinking, as inverse relationships have previously been found in non-smokers but not in smokers[19,20]. In the present study, the inverse associations were only observed among non-drinkers, but not among drinkers. Thus, the interaction of serum carotenoids with other risk factors should be taken into account. Furthermore, the effectiveness of individual carotenoids may rely on the levels of other carotenoids. Our findings indicated that individual carotenoids other than β-carotene were related to the MetS, suggesting that carotenoids may interact synergistically, and thus supplementing with a single β-carotene may be ineffective. Finally, β-carotene plays a role in improving antioxidant capacity only at relatively low concentrations, but loses this capacity at higher concentrations, and high-dose supplements of a single carotenoid might interfere with the absorption or bioavailability of others[11,32].

There are several biological mechanisms accounting for the protective role of carotenoids. It is well known that increased oxidative stress plays a key role in the MetS and its components[6,7]. Carotenoids are very efficient physical and chemical quenchers of singlet oxygen ($^{1}O_{2}$) and potent scavengers of other reactive oxygen species[9]. They are also known to inhibit lipid peroxidation and to scavenge lipid peroxyl radicals, preventing the propagation of free radical-mediated chain reactions. Furthermore, carotenoids can act synergistically as an effective 'radical-trapping antioxidant' in biological membranes, thereby protecting cells against

Table 4. Multivariate-adjusted OR of the metabolic syndrome for each quartile of serum carotenoid concentrations by subgroups of sex, smoking status and alcohol drinking*

(Odds ratios and 95 % confidence intervals)

	Quartile 1	Quartile 2		Quartile 3		Quartile 4		
	OR	OR	95 % CI	OR	95 % CI	OR	95 % CI	P for interaction
α-Carotene (μmol/l)								
Sex								0·203
Women	1·00	0·76	0·51, 1·13	0·59	0·39, 0·90	0·29	0·17, 0·47	
Men	1·00	1·01	0·46, 2·21	0·83	0·36, 1·94	0·46	0·17, 1·27	
Smoking								0·595
Non-smoker	1·00	0·78	0·53, 1·15	0·55	0·37, 0·83	0·29	0·19, 0·46	
Smoker	1·00	1·00	0·42, 2·41	0·46	0·13, 1·65	0·48	0·10, 2·23	
Alcohol drinking								0·048
No	1·00	0·81	0·56, 1·17	0·54	0·36, 0·80	0·29	0·18, 0·46	
Yes	1·00	2·29	0·56, 9·43	1·07	0·22, 5·20	1·51	0·29, 7·96	
β-Carotene (μmol/l)								
Sex								0·260
Women	1·00	0·56	0·38, 0·83	0·45	0·29, 0·69	0·23	0·14, 0·39	
Men	1·00	0·42	0·19, 0·92	0·21	0·08, 0·53	0·25	0·10, 0·61	
Smoking								0·507
Non-smoker	1·00	0·60	0·41, 0·89	0·44	0·29, 0·66	0·26	0·16, 0·41	
Smoker	1·00	0·42	0·15, 1·16	0·12	0·02, 0·94	1·05	0·33, 3·43	
Alcohol drinking								0·040
No	1·00	0·55	0·38, 0·80	0·35	0·23, 0·53	0·21	0·13, 0·33	
Yes	1·00	0·39	0·07, 2·13	0·71	0·13, 3·85	1·43	0·33, 6·16	
β-Cryptoxanthin (μmol/l)								
Sex								0·328
Women	1·00	0·91	0·61, 1·37	0·76	0·50, 1·17	0·59	0·37, 0·94	
Men	1·00	0·62	0·27, 1·42	0·90	0·42, 1·91	0·33	0·12, 0·93	
Smoking								0·249
Non-smoker	1·00	0·76	0·51, 1·14	0·72	0·48, 1·08	0·49	0·32, 0·75	
Smoker	1·00	0·87	0·36, 2·14	0·53	0·17, 1·65	0·20	0·03, 1·59	
Alcohol drinking								0·693
No	1·00	0·71	0·48, 1·03	0·66	0·44, 0·97	0·44	0·29, 0·67	
Yes	1·00	2·24	0·58, 8·74	1·47	0·31, 7·01	0·41	0·04, 4·14	
Lycopene (μmol/l)								
Sex								0·116
Women	1·00	0·73	0·49, 1·08	0·43	0·27, 0·67	0·39	0·25, 0·61	
Men	1·00	0·79	0·35, 1·81	0·51	0·20, 1·32	0·85	0·37, 1·95	
Smoking								0·362
Non-smoker	1·00	0·59	0·40, 0·88	0·45	0·30, 0·68	0·38	0·25, 0·57	
Smoker	1·00	0·35	0·10, 1·25	0·61	0·20, 1·90	0·78	0·25, 2·49	
Alcohol drinking								0·068
No	1·00	0·57	0·39, 0·84	0·48	0·32, 0·71	0·36	0·24, 0·55	
Yes	1·00	0·91	0·20, 4·18	0·30	0·03, 2·93	2·06	0·53, 7·99	
Lutein+zeaxanthin (μmol/l)								
Sex								0·481
Women	1·00	0·59	0·39, 0·88	0·59	0·39, 0·90	0·31	0·19, 0·50	
Men	1·00	1·19	0·55, 2·58	0·66	0·27, 1·59	0·60	0·24, 1·50	
Smoking								0·208
Non-smoker	1·00	0·55	0·37, 0·81	0·53	0·36, 0·79	0·30	0·19, 0·47	
Smoker	1·00	1·30	0·52, 3·26	0·82	0·26, 2·53	0·73	0·22, 2·47	
Alcohol drinking								0·371
No	1·00	0·57	0·39, 0·83	0·49	0·33, 0·74	0·27	0·17, 0·43	
Yes	1·00	1·02	0·25, 4·19	0·72	0·16, 3·28	0·72	0·15, 3·47	
Total carotenoids (μmol/l)								
Sex								0·598
Women	1·00	0·52	0·34, 0·78	0·44	0·29, 0·69	0·18	0·10, 0·31	
Men	1·00	0·59	0·27, 1·29	0·30	0·12, 0·77	0·49	0·21, 1·16	
Smoking								0·852
Non-smoker	1·00	0·50	0·34, 0·75	0·38	0·25, 0·58	0·19	0·12, 0·31	
Smoker	1·00	0·21	0·06, 0·72	0·31	0·09, 1·08	0·71	0·19, 2·61	
Alcohol drinking								0·095
No	1·00	0·50	0·34, 0·73	0·34	0·22, 0·51	0·17	0·11, 0·28	
Yes	1·00	0·12	0·02, 1·02	0·74	0·19, 2·88	0·94	0·20, 4·54	

* Model adjusted for the following factors: age, sex (except for sex stratification) and energy; marital status; education level; occupation; household income; smoking status; alcohol drinking; physical activity; serum total cholesterol, dietary intakes of fibre, cholesterol, SFA, vitamin C, vitamin E and β-carotene; serum levels of retinol and α-tocopherol. α for interaction = 0·05/eighteen tests = 0·0028.

different reactive nitrogen species[10]. In addition to their anti-oxidant properties, carotenoids can modulate lipoxygenase enzyme activity, probably inhibiting this inflammatory enzyme and thus the production of molecules with pro-inflammatory properties[33]. Furthermore, carotenoids also exhibit anti-inflammatory effects by inhibiting C-reactive protein, leucocytes, fibrinogen and inflammatory cytokines[34,35]. Gouranton et al.[13] suggested that lycopene inhibited pro-inflammatory cytokine production, such as IL-6, IL-1β and TNF-α, to prevent obesity-associated pathologies, such as insulin resistance.

There are several limitations to the present study. First, the cross-sectional design limits the ability to draw inferences regarding temporality and causation. It is not possible to conclude from the present study whether the lower concentrations were the result of a lower consumption of dietary carotenoids or increased utilisation of carotenoids due to the oxidative stress effects of the MetS. However, a previous RCT found that the baseline concentration of β-carotene was negatively associated with the incidence of the MetS after a 7·5-year follow-up, indicating that low levels of carotenoids might be causally involved in the development of the MetS[21]. Second, participants were volunteers who might have healthier behaviours and higher incomes and education levels than the general population. However, we did not observe any significant difference in the associations studied among the subgroups stratified by smoking status, alcohol drinking, education level or income, suggesting good generalisability across different statuses of these factors. Third, we could not obtain accurate data on the participants' intake of micronutrients from supplements. However, few supplements contain these carotenoids in China. Fourth, although we adjusted for major sociodemographic characteristics, lifestyle factors, dietary intakes of antioxidants, and serum levels of α-tocopherol and retinol simultaneously, confounding by unknown or unmeasured factors cannot be completely ruled out. Finally, the participants were middle-aged and elderly Chinese adults living in urban areas, which limits the generalisability of these results to other populations.

In general, we found an association between higher serum carotenoid levels and a lower prevalence of the MetS in Chinese adults. The present study adds to the accumulating evidence that antioxidant carotenoids may have a protective effect against the MetS. However, additional longitudinal studies and RCT are needed to confirm the beneficial association between carotenoids and the incidence of the MetS in this population.

Supplementary material

To view supplementary material for this article, please visit http://dx.doi.org/10.1017/S000711451400316X

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