Association of metabolic syndrome with obesity measures, metabolic profiles, and intake of dietary fatty acids in people of Asian Indian origin

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ABSTRACT

Objective: The present community-based cross-sectional study was aimed to examine the association of metabolic syndrome (MS) with obesity measures, metabolic profiles, and intake of dietary fatty acids in Asian Indian population.

Patients and Methods: A total of 350 adult (30 years and above) individuals (184 males and 166 females) inhabiting in and around Kolkata, India participated in this study. MS was defined using the protocol specifically designed for Asian Indian population.

Results: The prevalence of MS in the study was 31.4%. The prevalence was significantly higher \((P < 0.01)\) in females (48.2%) as compared to males (16.3%). It was observed that males without MS had significantly higher mean waist circumference (WC; \(P < 0.05\)); waist–hip ratio (WHR; \(P < 0.001\)); triglyceride (TG; \(P < 0.05\)); very low density lipoprotein cholesterol (VLDLc; \(P < 0.05\)) and fasting blood glucose (FBG; \(P < 0.01\)) as compared to females without MS. Significant differences were also observed for dietary intake of total fatty acids (TFA; \(P < 0.001\)); saturated fatty acids (SFA; \(P < 0.001\)) and polyunsaturated fatty acids (PUFA; \(P < 0.001\)) between individuals with and without MS. However, no significant association was observed in individuals with MS after controlling for age and sex. On the other, WC and body mass index (BMI) had significant correlation with SFA: mono unsaturated fatty acids (MUFA; \(P < 0.01\)) in individuals without MS even after controlling for age and sex.

Conclusion: It seems reasonable to argue that while dealing with MS in Asian Indians, clinicians should consider obesity measures, metabolic profiles and dietary fatty acids simultaneously.

Key words: Dietary fatty acids, metabolic syndrome, obesity, Asian Indians

DOI: 10.4103/0975-3583.70911

INTRODUCTION

The metabolic syndrome (MS) may be defined as the constellation of cardiovascular disease (CVD) risk factors, e.g., dyslipidemia, hypertension, hyperglycemia, etc. Persons with MS are essentially at twice the risk of CVD compared to those without the syndrome. It further raises the risk of type 2 diabetes mellitus (T2DM) by about fivefold. In most countries, about 20–30% of the adult population is predisposed to MS.[1] The MS is not a discrete entity known to be caused by a single factor. Moreover, it shows considerable variation in the components among different individuals. This variation is even greater among different racial and ethnic groups.[2] The MS is not restricted to the adults only, the predisposition of MS however, starts much early in life especially during the adolescence and young age.[3–7]

The prevalence of MS is increasing south Asian countries including India, leading to increased morbidity and mortality due to T2DM and CVD.[8] The increasing incidence of MS among the Asian Indians is a reason for concern since if effective interventions are not applied.[9] Lack of habitual physical activity and certain dietary...
patterns, including high-saturated fatty acids (SFA) and low
vegetable intakes, contribute to weight gain and increase
the risk of metabolic disturbances.\[^{10}\]\[^{11}\] Saturated fat intake
is associated with increased risk of coronary heart disease
(CHD); the greatest risk reduction is associated with
poly unsaturated fatty acids (PUFA) followed by mono
unsaturated fatty acids (MUFA).\[^{11}\]

In Asian Indians, there existed significant inverse
association between central obesity measures and intake
of unsaturated fatty acids due to recent shift in dietary
habits causing an increase in the prevalence of obesity
and dyslipidemia in this region.\[^{12,13}\]\[^{11}\] Keeping this view in
mind, the present study was undertaken among the Asian
Indian population living in the eastern part of India with
the following objectives:

- To compare obesity measures, lipids, lipoproteins,
  plasma glucose, and intake of dietary fatty acids in
  subjects with and without MS.
- To study the association of dietary fatty acids, their
  ratios with obesity measures in people with and without
  MS.

**PATIENTS AND METHODS**

**Study population**

The present community based cross-sectional study was
conducted on adult (aged 30 years and above) Asian
Indian men and women from Kolkata (erstwhile Calcutta)
and suburbs, West Bengal, India. A total of 350 individuals
(184 males and 166 females) participated in the study.
Subjects were categorized into two groups: individuals
with MS (\(n = 110\); male = 30, and female = 80) and
without MS (\(n = 240\); male = 154, and female = 86).
Pregnant women, women on hormone therapy (HT), and
individuals with known illness like ischemic heart disease
(IHD), T2DM, and hypertension were not incorporated
in the study. Before the actual commencement of the
study, a public advertisement was circulated regarding
the study with the help of local municipal council
officials. Individuals were selected randomly after they
responded to the local advertisement. The response rate
was as high as 85\%. The institutional ethics committee
of the “Human Genetic Engineering Research Centre”
(HGERC) has had approved the study. Written consent
was obtained prior to actual commencement of the study.

**Anthropometric measures**

Height (nearest 0.1 cm), weight (nearest 0.5 kg), waist
(nearst 0.2 cm), and hip circumferences (nearest 0.2 cm)
were obtained using standard techniques.\[^{14}\]\[^{11}\] The body
mass index (BMI, kg/m\(^2\)) and waist–hip ratio (WHR) were
subsequently computed.

**Blood pressure**

Left arm systolic (SBP) and diastolic (DBP) blood pressure
measurements was twice taken using sphygmomanometer
and stethoscope and was averaged for analyses. A third
measurement was taken only when the difference between
the two measurements was \(\geq 5\) mmHg. A 5 min relaxation
period between measurements was maintained through
out the study. SBP and DBP was measured as appearance
(phase I) and disappearance (phase V) of Korotkoff sound,
respectively.

**Metabolic profiles**

A fasting blood sample (7 mL) was drawn from participants
for the determination of fasting plasma glucose (FBG),
total cholesterol (TC), triglyceride (TG), and high density
lipoprotein cholesterol (HDLc). All subjects were
maintained an overnight fast of \(\geq 12\) h prior to blood
collection. Plasma was separated within 2 h of blood
collection using a microcentrifuge at 1000 rpm for 20 min in
room temperature. Estimation of FBG, TC, TG, and HDLc
were carried out using an ERBA Microscan ELISA Reader
(Trans Asia Biomedicals Limited, Mumbai, India). Low-
density lipoprotein cholesterol (LDLc) and very low-density
lipoprotein cholesterol (VLDLc) was then calculated using
the standard formulas: LDLc = TC – (HDLc + TG/5) and
VLDLc = TG/5. All metabolic profiles were estimated
in mg/dL (mg %) unit. TC:HDLc ratio was calculated
subsequently. The reproducibility of the instruments was
checked periodically using control solutions.

**Dietary fatty acids**

The dietary intake was recorded using the 24 h recall
method for seven consecutive days using a food frequency
schedule prepared in local language. The total fatty acids
(TFA), saturated fatty acids (SFA), monounsaturated fatty
acids (MUFA), and polyunsaturated fatty acids (PUFA)
contents of various foods were obtained using the standard
guidelines.\[^{15}\]\[^{11}\] The standardization to convert foodstuffs in
to fatty acids has been mentioned elsewhere.\[^{12,13,16,17}\]

**Definition of metabolic syndrome**

Subjects with any three or more of the following criteria
were considered under MS: \[^{18}\]\[^{10}\]
- Waist circumference (cm): male > 90; female > 80
- Triglycerides (mmol/L): \(\geq 2.25\)
- HDLc (mmol/L): male < 1.03; female < 1.28
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- Blood pressures (mmHg): SBP ≥ 130 and/or DBP ≥ 85
- Fasting blood glucose (FBG) (mmol/L): ≥ 5.56

**Statistical analysis**

Descriptive statistics such as mean and standard deviation (SD) of anthropometric, obesity measures, metabolic profiles, and intake of dietary fatty acids were undertaken separately for each group (metabolic vs. non-metabolic) and sex. Person’s partial correlation (adjusted for age and sex) between obesity measures (WC and BMI) and dietary fatty acids was also undertaken. All statistical analyses were performed using SPSS (PC+ version 10). A statistical significance (two-tailed) was set at \( P < 0.05 \).

**RESULTS**

The prevalence of MS in the present population was 31.4%. The prevalence was significantly higher \((P < 0.01)\) in females (48.2%) as compared to males (16.3%). The analysis of variance (ANOVA) showed a significantly higher mean WC \((P < 0.001)\); WHR \((P < 0.001)\); TC \((P < 0.001)\); TG \((P < 0.001)\); VLDLc \((P < 0.001)\); TC:HDLc \((P = 0.05)\) and FBG in males with MS as compared to females with MS [Table 1].

However, males without MS had significantly higher mean WC \((P < 0.05)\); WHR \((P < 0.001)\); TG \((P < 0.05)\); VLDLc \((P < 0.05)\), and FBG \((P < 0.01)\) as compared to females without MS [Table 2].

Comparison of dietary intake of fatty acids (g/week) between males and females with and is presented in Table 3. It was observed that males with MS had a significantly higher consumption of MUFA \((P < 0.01)\) and TFA:SFA \((P < 0.001)\), whereas females with MS had significantly

### Table 1: Obesity measures, metabolic profiles, and blood pressures variables between males and females with metabolic syndrome (n = 110)

| Variables        | Male (n = 30) | 95% CI Lower–upper | Female (n = 80) | 95% CI Lower–upper |
|------------------|--------------|---------------------|-----------------|---------------------|
| Age (years)*     | 56.67 ± 13.12 | 51.77 – 61.57       | 51.81 ± 10.39   | 49.50 – 54.12       |
| BMI (kg/m²)      | 24.71 ± 2.93  | 23.6 – 25.80        | 24.32 ± 3.61    | 23.52 – 25.12       |
| WC (cm)**        | 96.83 ± 5.77  | 94.68 – 98.99       | 92.98 ± 6.72    | 91.48 – 94.47       |
| WHR***           | 1.01 ± 0.003  | 0.99 – 1.02         | 0.97 ± 0.004    | 0.95 – 0.97         |
| TC (mmol/L) ***  | 2.42 ± 0.41   | 2.27 – 2.58         | 2.23 ± 0.26     | 2.17 – 2.29         |
| TG (mmol/L) ***  | 1.82 ± 0.37   | 1.68 – 1.96         | 1.65 ± 0.24     | 1.60 – 1.71         |
| HDLc (mmol/L)    | 1.06 ± 0.13   | 1.01 – 1.12         | 1.10 ± 0.11     | 1.08 – 1.13         |
| LDLc (mmol/L) ***| 3.64 ± 0.88   | 3.30 – 3.97         | 3.23 ± 0.59     | 3.09 – 3.36         |
| VLDLc (mmol/L) **| 0.36 ± 0.007  | 0.33 – 0.39         | 0.33 ± 0.004    | 0.32 – 0.34         |
| TC:HDLc***       | 2.32 ± 0.566  | 2.11 – 2.54         | 2.04 ± 0.39     | 1.96 – 2.13         |
| FBG (mg/dL) **   | 6.46 ± 2.14   | 5.67 – 7.26         | 5.22 ± 1.17     | 4.96 – 5.48         |
| SBP (mmHg)       | 145.57 ± 23.41| 136.83 – 154.31     | 149.19 ± 21.69  | 144.36 – 154.01     |
| DBP (mmHg)       | 88.43 ± 9.13  | 85.02 – 91.84       | 87.71 ± 9.02    | 85.71 – 89.72       |

CI = Confidence interval; values are mean ± standard deviation. BMI = Body mass index; WC = waist circumference; WHR = waist–hip ratio; TC = total cholesterol; TG = triglyceride; HDLc = high density lipoprotein; LDLc = low density lipoprotein; VLDLc = very low density lipoprotein; FBG = fasting plasma glucose; SBP = systolic blood pressure; DBP = diastolic blood pressure. Significant sex difference at *P < 0.05; **P < 0.01; ***P < 0.001.

### Table 2: Obesity measures, metabolic profiles, and blood pressures variables between males and females without metabolic syndrome (n = 240)

| Variables        | Male (n = 154) | 95% CI Lower–upper | Female (n = 86) | 95% CI Lower–upper |
|------------------|--------------|---------------------|-----------------|---------------------|
| Age (years)***   | 53.53 ± 12.24| 51.58 – 55.47       | 54.38 ± 11.80   | 42.85 – 47.91       |
| BMI (kg/m²)      | 21.91 ± 4.13 | 21.25 – 22.56       | 22.15 ± 4.75    | 21.13 – 23.17       |
| WC (cm)*         | 88.44 ± 10.13| 86.83 – 90.05       | 85.12 ± 10.50   | 82.86 – 87.37       |
| WHR***           | 0.96 ± 0.006 | 0.95 – 0.97         | 0.92 ± 0.005    | 0.91 – 0.93         |
| TC (mmol/L)      | 2.28 ± 0.28  | 2.23 – 2.32         | 2.24 ± 0.24     | 2.19 – 2.30         |
| TG (mmol/L)      | 1.57 ± 0.27  | 1.53 – 1.61         | 1.49 ± 0.23     | 1.44 – 1.54         |
| HDLc (mmol/L)    | 1.15 ± 0.12  | 1.13 – 1.17         | 1.16 ± 0.12     | 1.14 – 1.19         |
| LDLc (mmol/L)    | 3.34 ± 0.65  | 3.23 – 3.44         | 3.28 ± 0.58     | 3.16 – 3.41         |
| VLDLc (mmol/L)*  | 0.31 ± 0.005 | 0.30 – 0.32         | 0.29 ± 0.004    | 0.28 ± 0.30         |
| TC:HDLc**        | 2.02 ± 0.44  | 1.95 – 2.09         | 1.96 ± 0.37     | 1.87 – 2.04         |
| FBG (mg/dL)      | 4.92 ± 0.88  | 4.78 – 5.06         | 4.64 ± 0.51     | 4.53 – 5.75         |
| SBP (mmHg)       | 130.52 ± 23.43| 126.79 – 134.25     | 126.07 ± 21.65  | 121.43 – 130.71     |
| DBP (mmHg)       | 81.01 ± 11.44| 79.19 – 82.83       | 79.55 ± 10.39   | 77.32 – 81.77       |

Values are mean ± standard deviation. Significant sex difference at *P < 0.05; **P < 0.01; ***P < 0.001.
higher rate of proportion of SFA:MUFA ($P < 0.01$) and SFA:PUFA ($P < 0.001$) consumption than their male counterparts.

However, males without MS had significantly higher proportion of TF:SFA ($P < 0.001$) consumption than their female counterparts. Whereas females without MS had significantly higher SFA ($P < 0.005$) and SFA:MUFA ($P < 0.001$) consumption as compared to males without MS [Table 4].

Pearson’s partial correlations (controlling for age and sex) between adiposity (WC and BMI) and dietary fatty acids are presented in Table 5. It was observed that WC had significant correlation with SFA ($P < 0.01$); MUFA ($P < 0.05$) as well as TF:SFA ($P < 0.01$); SFA:MUFA ($P < 0.01$), and SFA:PUFA ($P < 0.01$). BMI had significant correlation with SFA ($P < 0.05$) and TFA:SFA ($P < 0.05$). However, no significant association was observed in individuals with MS after controlling for age and sex. On the other, WC and BMI had significant correlation with SFA:MUFA ($P < 0.001$) in individuals without MS.

**DISCUSSION**

Our findings hinted that intake of saturated fat may be a major risk factor for the onset of MS in adult Asian Indians. Moreover, it appears that not the total fat but the amount of saturated fat consumed in association with TFA:SFA, SFA:MUFA, and SFA:PUFA was adversely affecting the adiposity level, lipids, blood pressures, and blood glucose levels in this population and in turn cumulatively enhancing the possibility to predispose to MS phenotypes.

### Table 3: Dietary fat intake (g/week) by males and females with metabolic syndrome (n = 110)

| Variables | Male (n = 30) | 95% CI Lower–upper | Female (n = 80) | 95% CI Lower–upper |
|-----------|--------------|---------------------|----------------|-------------------|
| TF        | 615.78 ± 75.46 | 587.6 – 643.9 | 586.23 ± 74.53 | 569.6 – 602.8 |
| SFA       | 190.43 ± 38.58 | 176.0 – 204.8 | 195.82 ± 27.68 | 189.6 – 201.9 |
| MUFA**    | 228.60 ± 27.00 | 218.5 – 238.6 | 213.72 ± 25.65 | 208.0 – 219.4 |
| PUFA      | 159.09 ± 25.76 | 149.4 – 168.7 | 153.82 ± 26.79 | 125.6 – 182.0 |
| TF:SFA*** | 3.30 ± 0.35 | 3.13 – 3.47 | 3.01 ± 0.26 | 2.9 – 3.0 |
| SFA:MUFA*** | 0.83 ± 0.11 | 0.78 – 0.87 | 0.91 ± 0.10 | 0.89 – 0.94 |
| SFA:PUFA*** | 1.21 ± 0.25 | 1.12 – 1.31 | 1.41 ± 0.26 | 1.35 – 1.47 |
| PUFA:MUFA | 0.69 ± 0.009 | 0.66 – 0.73 | 0.72 ± 0.006 | 0.58 – 0.86 |

Values are mean ± standard deviation. TF = Total fat; SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids. Significant sex difference at *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

### Table 4: Dietary fat intake (g/week) by males and females without metabolic syndrome (n=240)

| Variables | Male (n = 30) | 95% CI Lower–upper | Female (n = 80) | 95% CI Lower–upper |
|-----------|--------------|---------------------|----------------|-------------------|
| TF        | 560.09 ± 67.29 | 549.3 – 570.8 | 561.30 ± 71.36 | 546.0 – 576.6 |
| SFA*      | 146.06 ± 20.18 | 142.8 – 149.2 | 152.47 ± 22.89 | 147.5 – 157.3 |
| MUFA      | 215.33 ± 18.11 | 212.4 – 218.2 | 213.32 ± 19.47 | 209.1 – 217.5 |
| PUFA      | 157.26 ± 34.59 | 151.7 – 162.7 | 153.47 ± 32.23 | 146.5 – 160.3 |
| TF:SFA*** | 3.85 ± 0.28 | 3.80 – 3.89 | 3.70 ± 0.26 | 3.6 – 3.7 |
| SFA:MUFA*** | 0.67 ± 0.006 | 0.66 – 0.68 | 0.71 ± 0.007 | 0.69 – 0.73 |
| SFA:PUFA   | 1.00 ± 0.75  | 0.88 – 1.12 | 1.02 ± 0.20 | 0.97 – 1.06 |
| PUFA:MUFA | 0.72 ± 0.12 | 0.70 – 0.74 | 0.71 ± 0.11 | 0.69 – 0.47 |

Values are mean ± standard deviation. Significant sex difference at *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

### Table 5: Partial correlation (controlling for age and sex) between dietary fatty acids and adiposity measures

| Correlation | TFA | SFA | MUFA | PUFA | TF:SFA | SFA:MUFA | SFA:PUFA | PUF A:MUFA |
|-------------|-----|-----|------|------|--------|----------|----------|------------|
| Total population (n = 350) | | | | | | | | |
| WC         | −0.92 | 0.207** | 0.133* | −0.571 | −0.205* | 0.177** | 0.116* | −0.088 |
| BMI        | 0.03 | 0.117* | 0.081 | −0.056 | −0.133* | 0.096 | 0.054 | −0.076 |
| Individuals with metabolic syndrome (n = 110) | | | | | | | | |
| WC         | 0.128 | 0.045 | 0.124 | −0.139 | 0.107 | −0.053 | −0.012 | −0.158 |
| BMI        | 0.003 | −0.080 | −0.003 | −0.103 | 0.164 | −0.110 | −0.069 | −0.102 |
| Individuals without metabolic syndrome (n = 240) | | | | | | | | |
| WC         | −0.01 | −0.036 | 0.118 | −0.027 | 0.060 | −0.165** | 0.031 | −0.108 |
| BMI        | −0.035 | −0.082 | 0.097 | −0.037 | 0.082 | −0.214** | −0.016 | −0.107 |

Significant at *$P < 0.05$; **$P < 0.01$.
The gender differences in prevalence of MS have been found in several other studies as well. It might be due to different cut-off points set as criteria for MS like WC and HDLc. It is important to mention that individuals with impaired glucose tolerance, impaired fasting glucose was observed more frequently in men whereas impaired glucose tolerance occurred relatively more often in women. Lipids accumulation patterns differ between women and men. Premenopausal women more frequently develop peripheral obesity with subcutaneous fat accumulation whereas men and postmenopausal women are more prone to central or android pattern of obesity. In particular, android obesity is associated with increased cardiovascular mortality and the development of T2DM. Inflammation increases cardiovascular risk particularly in women. It has also been mentioned that the pathophysiology of the MS, and its contribution to the relative risk of cardiovascular events and heart failure show gender differences, which has immense potential relevance for prevention, diagnostics, and therapy of the syndrome.

Certain dietary patterns, including high SFA and low vegetable intake, contribute to weight gain and increase the risk of metabolic disturbances, whereas such potentially modifiable lifestyle factors may reduce cardiovascular risk. In a recent study from India revealed that increased dietary ω-6 PUFA and saturated fat intake are significantly associated with fasting hyperinsulinemia and subclinical inflammation, respectively, and might be responsible for the increasing prevalence of insulin resistance, the MS and T2DM in Asian Indians. In another study, it was found that the dietary total fat may increase whereas linoleic acid intake may reduce the risk of MS in Japanese descendants living in Brazil. Study from the United Arab Emirates (UAE) pointed out that poor dietary habits including consumption of high-energy foodstuffs, diets high in total carbohydrates, fat, and simple sugars were associated with MS. In a multiethnic study (comprising of African-Americans, Whites and Hispanics) on healthy children aged 7–12 years revealed that diet composition was more closely related to the components of the MS than was physical activity, with carbohydrate intake being adversely related to WC, TG levels, and glucose levels. Moreover, relationships among diet and MS outcomes were stronger among African-American children reflecting ethnic variation. Our findings of an adverse impact of TFA:SFA intake and inverse relation of adiposity with PUFA, i.e., protection of polyunsaturated fat concerning the association with MS is in accordance with the finding of several other investigations.

However, the major limitation was that the study was performed on a relatively small sample size and therefore not representative of Asian Indian population. Moreover, the dietary fatty acids were obtained through retrospective method and not directly from the isolated plasma. Further prospective studies are required to better compared gene-diet interaction in the growing menace of MS in this part of the world.

ACKNOWLEDGMENTS

AG received financial support (Ref. No. 5/9/48/2006-RHN vide RFC No. RHN/Adhoc/1/2009-10) from the Indian Council of Medical Research (ICMR), Government of India, New Delhi. MD received partial funding [Ref. No. EPSW-176/09-10 (ERO)] from the University Grants Commission (UGC), Government of India, New Delhi. The authors are grateful to the staff and technicians of the HGERC, Kolkata, India for their sincere help in analyzing the metabolic profiles. The authors are also indebted to all the subjects participated in the study.

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Source of Support: Indian Council of Medical Research, University Grants Commission, Government of India, Conflict of Interest: None declared.