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ORIGINAL ARTICLE

Plasma Fatty Acid Composition and Estimated Desaturase Activities Reflect Dietary Patterns in Subjects with Metabolic Syndrome

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Abstract Changes in plasma fatty acid (FA) composition and desaturase activities are observed in metabolic syndrome (MS). However, whether these changes are a reflection of dietary intakes of fats and FAs is not well established. The current study was aimed at assessing plasma FA composition and desaturase enzyme activities as biomarkers of dietary intakes in subjects with MS. Case control study was done on 41 MS patients and was compared with age matched 45 controls. Dietary intakes, anthropometric and clinical parameters were measured. FA composition was analysed using gas chromatography-flame ionisation detector and desaturase enzyme activities were estimated as ratios of product to precursor FAs. Higher levels of 14:0, 16:0, 16:1, 18:1, D9D-18 activity and lower levels of 18:0 and 18:2 n-6 were seen in MS group when compared to controls ($p < 0.05$). Strong positive correlations were seen between plasma triglyceride (TG) levels and 14:0, 16:0, 16:1, 18:1, total saturated fatty acid, total monounsaturated fatty acid, and D9D activities, while 18:0, 18:2 n-6 and total polyunsaturated fatty acid were negatively correlated with TG. Positive correlations were seen between plasma 14:0, 18:1 and D9D-18 activity with total energy intake and carbohydrate (CHO) intakes but not with fat intake. Plasma FA profile appears to be a better index of total energy intake and CHO intake than fat intake, suggesting it might be a good reflection of endogenous FA metabolism. Changes in FA composition may therefore serve as an early index of dysregulation of FA metabolism, resulting in increased risk of MS.

Keywords Plasma fatty acids · Delta 9-desaturase · Dietary carbohydrates · Endogenous fatty acid metabolism · Metabolic syndrome

Abbreviations

MS	Metabolic syndrome
CVD	Cardiovascular disorder
FA	Fatty acid
SFA	Saturated fatty acid
MUFA	Monounsaturated fatty acid
PUFA	Polyunsaturated fatty acid
D9D	Delta D9 desaturase
D6D	Delta D6 desaturase
D5D	Delta D5 desaturase
D4D	Delta D4 desaturase
CHO	Carbohydrate

Introduction

Metabolic syndrome (MS), a key risk factor driving the cardiovascular disease (CVD) epidemic, is rapidly increasing in India and around the world, leading to increased mortality and morbidity [1–4]. MS is a cluster of metabolic risk factors such as insulin resistance, obesity, altered glucose metabolism, dyslipidemia and arterial hypertension [5]. A number of reviews implicate dietary fats, especially saturated fatty acids (SFA) and trans fatty

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acids as risk factors for MS [6, 7]. Indian diets tend to have lower levels of fat, SFA and higher levels of n-6 polyunsaturated fatty acids (PUFA) [8, 9] and yet the incidence of obesity and MS is extremely high [10]. One possible explanation is that there is a disconnect between the data on dietary intake and its correlation with risk factors of MS. This is partly due to the fact that assessment of dietary intakes using food frequency questionnaires or 24-h dietary recalls is prone to subjective errors. Therefore surrogate biomarkers of dietary intakes are of utmost importance to identify the real associations between dietary intakes and CVD risk.

The fatty acid (FA) profile of adipose tissue has been suggested as a gold standard biomarker of dietary fat quality [11]. However this involves invasive procedures such as obtaining adipose tissue biopsies, and is therefore not feasible on a routine basis. Recent studies suggest that plasma or erythrocyte FA profile can also serve as surrogate biomarkers, wherein altered FA composition has been linked to MS and CVD [12, 13]. Data on Indian populations comparing FA composition and dietary intakes especially with respect to MS are sparse. An innovative way in which these data can be used is to calculate the activity of FA desaturases, which have been implicated as risk factors for obesity and MS, especially in Asian populations [14]. In addition to being a reflection of dietary fat intake, desaturase activities can provide information on de novo FA synthesis as well as the ability to form biologically important long chain n-6 and n-3 FA (Fig. 1) [15]. Therefore the current study was aimed at assessing plasma FA composition and desaturase enzyme activities as potential biomarkers of dietary intakes in subjects with MS.

Methods and Materials

Study Design and Subjects

Subjects diagnosed with MS based on World Health Organisation (WHO) criteria (Table 1) (n = 41) were recruited from the Nutrition out-patient department of St. John's Medical College, Bangalore. Sample size was calculated to detect a difference of 8% in LA levels between normal controls and subjects with MS with 80% power and 5% level of significance [12]. The corresponding age and sex matched control subjects (n = 45) were recruited from the staff and students of college. All subjects were in the age range of 35–60 years. The study was approved by the St. John's National Academy of Health Sciences Institutional Ethical Review Board, Bangalore. Written informed consent was obtained from each subject before enrolling them for the study.

Anthropometry and Dietary Information

Subjects' anthropometry included weight that was measured using a digital scale (Soehnle, Germany) recorded to the nearest 0.1 kg and height measured to the nearest 0.1 cm using a stadiometer. Body mass index (BMI, kg/m^2) was computed and skinfold thickness such as biceps, triceps and subscapular were measured using Holtain calipers. Dietary information was obtained using a standardised 24-h dietary recall questionnaire, administered on 3 different days which included two weekdays and a weekend/holiday. The dietary recall process was administered by a trained technician, using standardized measures

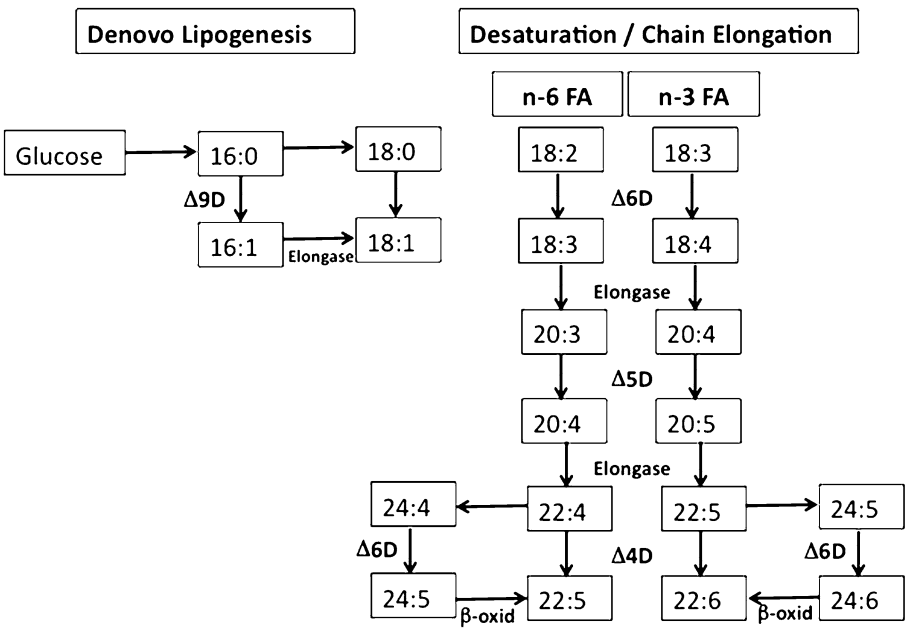

Fig. 1 Denovo lipogenesis and desaturation and elongation of fatty acids. Δ9D—Δ 9 desaturase, Δ6D—Δ 6 desaturase, Δ5D—Δ 5 desaturase, Δ4D—Δ 4 desaturase, β-oxid—β oxidation

Table 1 WHO criteria for metabolic syndrome

Risk factors	Definition
Type 2 diabetes mellitus or impaired fasting glucose or impaired glucose tolerance +	
Any 2 of the following	
High blood pressure	≥140 mm Hg systolic or ≥90 mm Hg diastolic
Plasma triglycerides	>150 mg/dl
HDL-cholesterol	<35 mg/dl in men or <39 mg/dl in women
Obesity	Body mass index (BMI) >30 kg/m^2 and/or waist:hip ratio >0.9 in men and >0.85 in women
Urinary albumin excretion rate	>20 μg/min

to quantify portion sizes. The individual's intake data was then incorporated into a programme using a nutrient database developed at the St John's Research Institute as indicated in a previous publication [16] to obtain the food and nutrient intake.

Clinical Data and Plasma Fatty Acid Analysis

After a thorough medical examination, fasting blood samples were collected from subjects for routine blood biochemistry examination such as insulin, glycated haemoglobin (HbA1c), lipids and fasting blood glucose (FBS). Plasma was separated from each sample and was used for FA estimation. Total lipids were extracted using chloroform: methanol (1:2) mixture and transmethylation of all FA from this fraction were carried out using 2% conc. H_2SO_4. Fatty acid methyl esters was analysed using gas chromatography with a flame ionization detector (Varian 3800; Varian, Palo Alto, CA, USA) and a fused silica column (FAME, Varian 50 m, 0.2 mm capillary column) with nitrogen as carrier gas. C17:0 (H3500 Sigma Aldrich) was used as an internal standard, total FA content of the samples was calculated and each identified FA was expressed as a percentage of the total FA. D9D-16, D9D-18, D6D, D5D and D4D activities were estimated by calculating the ratios of 16:1n-7/16:0, 18:1n-9/18:0, 20:3n-6/18:2n-6, 20:4n-6/20:3n-6 and 22:6n-3/22:5n-3, respectively.

Statistical Analysis

Data is expressed as arithmetic mean ± SD. Differences in baseline characteristics, dietary intake, anthropometry and plasma FA composition between groups (control vs MS) were assessed using independent Student's *t* test. Relationships among BMI, percent fat, W/H ratio, blood chemistry, dietary intakes and proportions of FA were assessed by Spearman's rank correlations. For all analysis, p values <0.05 were considered statistically significant. Statistical analysis was done using SPSS software (IBM SPSS Statistics for Windows, Version 21, Armonk, NY: IBM Corp).

Results

The general characteristics and dietary intakes of control and MS groups are presented in Table 2. The average values of BMI, waist/hip ratio (W/H), triglycerides (TG), FBS and HbA1c were significantly higher and HDL-C was significantly lower in the MS group when compared to controls. The dietary intakes of macronutrients in the two groups were not different.

The plasma FA composition and desaturase activity between the groups are represented in Table 3. The levels of myristic acid (14:0), palmitic acid (16:0), palmitoleic acid (16:1n-7), oleic acid (18:1n-9), α-linolenic acid (18:3 n-3), eicosapentaenoic acid (20:5 n-3), adrenic acid (22:4n-6), total SFA, total MUFA and D9D-18 were significantly higher ($p < 0.05$ for all), while the levels of stearic acid (18:0), linoleic acid (18:2n-6), docosapentanoic acid

Table 2 General characteristics and dietary intakes of study population

	Controls (n = 45)	MS (n = 41)	p value
BMI (kg/m^2)	23.6 ± 2.8	26.8 ± 4.8	<0.001
W/H	0.84 ± 0.08	0.91 ± 0.08	0.001
FBS (mg/dL)	81.9 ± 10.6	124.6 ± 52.5	<0.001
TC (mg/dL)	191.7 ± 40.6	195.6 ± 50.5	0.691
TG (mg/dL)	132.2 ± 75.0	299.7 ± 172.9	<0.001
HDL-C (mg/dL)	41.9 ± 9.9	35.6 ± 7.8	0.002
SBP (mmHg)	114.5 ± 11.7	119.8 ± 15.4	0.102
DBP (mmHg)	74.7 ± 8.9	78.3 ± 12.0	0.155
Insulin (mg/dL)	11.7 ± 5.7	15.1 ± 7.3	0.045
HOMA-IR	1.5 ± 0.72	2.0 ± 0.90	0.012
HbA1c (%)	5.6 ± 0.4	7.4 ± 2.1	<0.001
Dietary intake			
Energy (Kcal/day)	1810.0 ± 524.0	1811.0 ± 440.0	0.992
CHO (g/day)	282.0 ± 82.0	273.0 ± 63.0	0.602
Protein (g/day)	57.0 ± 19.0	57.0 ± 14.0	0.963
Fat (g/day)	50.2 ± 18.0	48.2 ± 15.0	0.595

All values expressed as mean ± SD

BMI body mass index, *W/H* waist hip ratio, *FBS* fasting blood sugar, *TC* total cholesterol, *TG* triglyceride, *HDL-C* high density lipoprotein cholesterol, *LDL-C* low density lipoprotein cholesterol, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HOMA-IR* homeostatic model of assessment of insulin resistance, *HbA1C* glycated haemoglobin, *CHO* carbohydrates

Table 3 Plasma fatty composition and estimated desaturase activity in control and metabolic syndrome

	Control (n = 45)	MS (n = 41)	*p* value
14:0	1.18 ± 0.52	1.52 ± 0.62	0.007
16:0	24.2 ± 2.0	26.4 ± 2.9	<0.001
18:0	7.8 ± 0.7	7.1 ± 0.8	<0.001
16:1 n-7	2.15 ± 1.06	2.68 ± 1.09	0.027
18:1 n-9	19.1 ± 2.3	22.0 ± 3.6	<0.001
18:2 n-6	33.4 ± 4.7	27.5 ± 5.7	<0.001
18:3 n-3	0.53 ± 0.45	0.76 ± 0.26	0.005
20:3 n-6	1.91 ± 0.50	1.77 ± 0.36	0.145
20:4 n-6	6.8 ± 1.7	6.8 ± 2.1	0.998
20:5 n-3	0.07 ± 0.15	0.21 ± 0.27	0.006
22:4 n-6	0.19 ± 0.21	0.34 ± 0.13	<0.001
22:5 n-6	0.53 ± 0.28	0.35 ± 0.18	0.001
22:5 n-3	0.15 ± 0.17	0.28 ± 0.12	<0.001
22:6 n-3	0.92 ± 0.58	1.01 ± 0.49	0.425
SFA	33.2 ± 2.6	35.0 ± 3.2	0.005
MUFA	21.2 ± 2.8	24.7 ± 4.2	<0.001
PUFA	44.5 ± 4.6	39.0 ± 6.8	<0.001
D9D-16	0.09 ± 0.04	0.10 ± 0.04	0.167
D9D-18	2.46 ± 0.42	3.18 ± 0.76	<0.001
D6D-18	0.06 ± 0.02	0.07 ± 0.01	0.162
D5D	3.69 ± 0.98	3.90 ± 1.22	0.368
D4D	3.60 ± 1.42	3.46 ± 1.29	0.689

All values expressed as mean ± SD. Fatty acids are expressed as percentage of total fatty acids

SFA saturated fatty acid, *MUFA* monounsaturated fatty acid, *PUFA* polyunsaturated fatty acid, *D9D* delta D9 desaturase, *D6D-18* delta D6 desaturase, *D5D* delta D5 desaturase, *D4D* delta D4 desaturase

(22:5n-6), and total PUFA were significantly lower ($p < 0.001$) in subjects with MS.

For all correlation analyses, the total data set of 86 subjects was considered. Correlation analysis of plasma FA composition with metabolic risk factors for MS is presented in Table 4. Most of the FA correlated well with plasma TG levels—some were positively correlated (14:0, 16:0, 16:1, 18:1, total SFA, total MUFA, D9D and D6D-18) ($p < 0.01$) whereas others were strongly negatively correlated (18:0, 18:2 n-6, and total PUFA) ($p < 0.01$). C16:0 correlated positively with maximum number of risk factors (W/H ratio, TG, insulin and HOMA-IR) ($p < 0.01$) and negatively with HDL-C ($p < 0.01$). On the other hand, 18:0 and 18:2 n-6 showed a negative correlation with a number of risk factors (W/H, FBS, TC, TG and HOMA-IR) ($p < 0.01$) and positive correlation with HDL-C ($p < 0.01$, $p < 0.05$ respectively). Of the desaturases, D9D-18 showed a positive correlation with W/H, FBS, total cholesterol (TC) and TG whereas a negative correlation was seen with HDL-C (all $p < 0.01$). D6D-18 was seen to correlate with BMI, TC ($p < 0.05$), TG, insulin and HOMA-IR ($p < 0.01$) whereas D5D correlated negatively with insulin ($p < 0.05$) and HOMA-IR ($p < 0.01$).

Correlation of plasma FA with dietary intakes of energy, fat and carbohydrates (CHO) (n = 86) is shown in Table 5. Energy and CHO intake were seen to correlate positively with plasma 14:0 ($p < 0.01$), D9D-18 ($p < 0.01$), 18:1 (energy-$p < 0.01$, CHO-$p < 0.05$), total MUFA (energy-$p < 0.01$, CHO-$p < 0.05$) but negatively with 18:0 ($p < 0.05$). However total energy intake was seen to correlate negatively with 18:2 n-6 ($p < 0.05$) and total PUFA ($p < 0.01$).

Discussion

Assessment of dietary intakes of total fat and FA are usually performed using subjective methods such as Food Frequency Questionnaires (FFQ) and 24-h dietary recalls but these don't often serve as good predictors of specific metabolic disorders. Therefore there continues to be a need for more objective measures of dietary fat intake. Although FA composition of adipose tissue is considered the best biomarker of fat intake [11, 17], plasma or RBC FA have also been shown to be reasonably reliable markers especially of recent intake of fats [18, 19]. However contradictory findings exist which suggest that dietary fat may not be the only macronutrient affecting the FA composition [20]. Therefore the current study aimed at assessing plasma FA composition in order to understand the activities of desaturases, their relation to dietary factors and their possible association with MS in an Indian population.

There were substantial differences detected in the FA composition of plasma between the two groups (Table 3). The predominant saturated FA, 16:0, was significantly higher in the plasma of MS subjects, and this was accompanied by higher levels of both MUFA—16:1 as well as 18:1. High concentrations of 16:0, 18:1, and total SFA have been reported earlier in subjects with MS [21]. It is interesting to note however that while the levels of 16:0 were elevated, the levels of 18:0 were lower in the MS group. Recent study has shown that plasma FA, especially SFA do not alter in response to dietary fat, however the FA composition is influenced more by dietary CHO, suggesting an influence on denovo lipogenesis [20].

In the current study it was seen that many plasma FA, such as 14:0, 18:1, as well as D9D-18 activity correlated strongly with total energy as well as CHO intake (Table 5). The fact that stronger correlations were seen for non-essential FA emphasises the likelihood that the differences in plasma SFA and MUFA composition may be due to alterations in endogenous FA synthesis rather than a reflection of dietary intakes of fats. This is also confirmed

Table 4 Correlation analysis between plasma fatty acids, desaturase activities and various metabolic risk factors

	BMI	W/H	FBS	TC	TG	HDL-C	Insulin	HOMA-IR
14:0	0.020	0.214	0.035	0.315**	0.646**	−0.198	0.314**	0.370**
16:0	0.008	0.330**	0.175	0.115	0.647**	−0.326**	0.254*	0.344**
18:0	−0.158	−0.477**	−0.218*	−0.353**	−0.633**	0.403**	−0.111	−0.168
16:1	0.105	0.191	0.177	0.324**	0.496**	−0.081	0.11	0.188
18:1	−0.069	0.482**	0.178	0.132	0.642**	−0.299**	0.069	0.142
18:2 n-6	−0.108	−0.323**	−0.279**	−0.204	−0.682**	0.251*	−0.227	−0.307*
20:3 n-6	0.318**	−0.088	−0.036	0.136	−0.104	−0.053	0.260*	0.228
20:4 n-6	0.284**	−0.204	0.075	−0.009	−0.244*	0.173	−0.027	−0.063
22:6 n-3	0.200	0.022	0.183	0.266*	0.181	0.012	0.120	0.145
SFA	−0.045	0.182	0.098	0.051	0.502**	−0.212*	0.246*	0.318**
MUFA	−0.021	0.473**	0.194	0.225*	0.693**	−0.276**	0.105	0.177
PUFA	0.015	−0.388**	−0.169	−0.181	−0.680**	0.289**	−0.218	−0.298*
D9D-16	0.123	0.123	0.159	0.329**	0.400**	−0.037	0.050	0.116
D9D-18	0.045	0.572**	0.219*	0.283**	0.743**	−0.384**	0.103	0.170
D6D-18	0.262*	0.148	0.170	0.229*	0.394**	−0.191	0.335**	0.372**
D5D	0.017	−0.154	0.079	−0.169	−0.205	0.202	−0.293*	−0.316**

BMI body mass index, *W/H* waist hip ratio, *FBS* fasting blood sugar, *TC* total cholesterol, *TG* triglyceride, *HDL-C* high density lipoprotein cholesterol, *HOMA-IR* homeostatic model of assessment of insulin resistance, *SFA* saturated fatty acid, *MUFA* monounsaturated fatty acid, *PUFA* polyunsaturated fatty acid, *D9D* delta D9 desaturase, *D6D-18* delta D6 desaturase, *D5D* delta D5 desaturase

Spearman's correlation analysis. * $p < 0.05$; ** $p < 0.01$

by the fact that no correlation was seen between plasma levels of essential FA such as 18:2 n-6 and 18:3 n-3 and dietary intakes of the same (data not shown).

There was a higher activity of D9D-18 in the MS group, although no differences were seen in the other desaturases (Table 3). This is interesting since desaturases have been associated with risk factors for Type 2 diabetes mellitus (T2DM) and CVD, more specifically with South Asians [22]. A study has shown that the activities of all the desaturases (D9D, D6D and D5D) exhibit ethnic variations, with lower activities seen in Asian Indians living in the UK [23] which was further associated with a higher risk of CVD in them. Results of the current study appear to corroborate this hypothesis, at least for D9D, in Indians living in India too.

The plasma FA composition was seen to correlate well with plasma lipids, especially with TG and HDL-C (Table 4). While a number of FA as well as D9D-16/18 and D6D-18 correlated positively with TG, a strong negative correlation was seen with 18:0 and 18:2 n-6. This is not surprising since endogenous FA synthesis and especially the D9D activity is key in TG synthesis in humans [24]. The fact that these results mirror the correlations with dietary CHO appears logical since plasma TG levels are dependent on CHO intake rather than fat intake [25]. This further confirms that the effects seen here are a reflection of endogenous FA synthesis rather than of fat or FA intake.

Animal and human studies suggest that D9D activity plays an important role in the development of obesity. One study demonstrated that Stearoyl-CoA desaturase-1 (SCD-1) (D9D) deficient mice showed reduced body adiposity and increased insulin sensitivity and inhibition of diet induced obesity. Reduction in lipid synthesis and increased lipid oxidation was observed in SCD-1 deficient mice [26–29]. Our results corroborate these findings where D9D-18 correlates strongly with W/H ratio, which is a good index of body fat distribution. Previous studies have reported that D9D is down-regulated by 18:2 n-6, and it is possible that the lower 18:2 n-6 levels seen in the MS group in the current study could be reflected in increased TG biosynthesis mediated by D9D. Studies in adults and adolescents have reported that higher activity of estimated D9D, D6D-18 and low 18:2 n-6 were all directly associated with development of MS [20, 30]. Similar results were seen in the current subjects with MS. The higher D9D-18 activity seen in the MS group, in spite of no obvious differences in diet, suggests that metabolic dysregulation of the FA synthesis machinery probably occurs in MS, leading to a pro-inflammatory FA profile.

In the present study although the D6D-18 activity was not significantly different between the two groups (Table 3), it was seen to be positively correlated with plasma TG levels (Table 4). D6D-18 converts 18:2 n-6 to γ-linolenic acid (18:3 n-6) which is further elongated and

Table 5 Correlation between plasma fatty acids and dietary intakes of calories, CHO and fat

	Energy	CHO	Fat
MA	0.346**	0.346**	0.134
PA	0.265*	0.185	0.056
SA	−0.274*	−0.252*	−0.077
POA	0.208	0.088	0.051
OA	0.371**	0.257*	0.242*
LA	−0.223*	−0.097	−0.107
DGLA	0.001	−0.026	0.098
AA	−0.17	−0.186	−0.012
DHA	−0.079	−0.158	−0.105
SFA	0.207	0.137	0.038
MUFA	0.347**	0.227*	0.179
PUFA	−0.305**	−0.201	−0.125
D9D-16	0.137	0.020	0.023
D9D-18	0.399**	0.311**	0.215
D6D	0.157	0.039	0.166
D5D	−0.206	−0.175	−0.174

CHO carbohydrates, *MA* myristic acid, *PA* palmitic acid, *SA* stearic acid, *POA* palmitoleic acid, *OA* oleic acid, *LA* linoleic acid, *DGLA* dihomo γ-linolenic acid, *AA* arachidonic acid, *DHA* docosahexanoic acid, *SFA* saturated fatty acid, *MUFA* monounsaturated fatty acid, *PUFA* polyunsaturated fatty acid, *D9D* delta D9 desaturase, *D6D-18* delta D6 desaturase, *D5D* delta D5 desaturase

Spearman's correlation analysis at * $p < 0.05$; ** $p < 0.01$

desaturated to arachidonic acid (20:4 n-6). It has been reported that higher D6D-18 activity increased the risk of MS in middle aged men [31]. It is also observed that D6D-18 gene expression could be altered in MS group due to hyperinsulinemia induced by obesity [32, 33]. This is corroborated in the present study, where D6D-18 correlated strongly with both plasma insulin as well as HOMA-IR. D6D-18 is also associated with inflammation as the PUFAs synthesised by D6D-18 participate in synthesis of eicosanoids which are known to be inflammatory mediators, and also act as ligands for transcriptional factors that are involved in inflammatory processes [34]. Therefore higher D6D-18 activity with low 18:2 n-6 levels plausibly indicate an increased metabolic risk.

The key strength of this study is the detailed dietary data in combination with anthropometric as well as biochemical measurements and erythrocyte FA composition in newly diagnosed MS subjects, which could possibly serve as early indicators of dysregulation of lipid metabolism. The correlations seen between different fatty acids and dietary calorie and carbohydrate intakes are especially interesting since, to date, there are really no good biomarkers of calorie or CHO intakes. This is particularly important in a country like India where carbohydrate intakes are relatively high. With more such data it may be possible to use plasma FA composition as an objective measure of dietary calorie and CHO intakes. The key limitation lies in the relatively small sample size, thus limiting the interpretation of results to associations between parameters.

In summary, this study suggests that plasma FA composition and desaturase activities of D9D-16, D9D-18 and D6D-18 could be valuable tools in assessing the response of the denovo FA synthesis to dietary intakes, especially of calories and CHO. Furthermore, if the observed associations between desaturase activities and indices of MS hold true in larger populations, they could in future be used as quantitative predictors of MS. Further such studies in different parts of India would help to strengthen these findings.

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Author Contributions All of the authors listed above have contributed to the work and have read, participated in the writing and agreed upon the submitted version of the manuscript.

Compliance with Ethical Standards

Conflict of interest The authors have no conflict of interest to disclose.

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