Original article

Relation between soluble CD36 and dietary fatty acid composition in metabolic syndrome patients

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Abstract. *Background and aim:* The CD36 fatty acid receptor, known as the scavenger receptor, is expressed in many cells and tissues. Dietary fatty acids are thought to play a role in the pathogenesis of metabolic disorders such as obesity, insulin resistance, and atherosclerosis. This study aimed to determine the relationship between soluble CD36 (sCD36) fatty acid receptors and dietary fatty acids in individuals with metabolic syndrome (MetS). *Methods:* This study included 33 patients with MetS and 32 healthy individuals aged 18-65 years. The participants' sociodemographic characteristics, biochemical parameters, anthropometric measurements, type of dietary fat, fatty acid pattern, and amount of fat consumed were recorded. The sCD36 fatty acid receptor levels in individuals were analyzed. *Results:* Blood pressure and biochemical measurements (fasting glucose, HbA1c, insulin, Homa-IR, triglyceride, total cholesterol, HDL, LDL, AST, ALT, and CRP) of individuals with MetS were higher than those of the control group ($p<0.05$). However, HDL and sCD6 levels did not differ between the groups ($p>0.05$). Individuals with MetS had lower olive oil, higher corn oil and tail oil consumptions ($p<0.05$). However, no difference was found between the groups in terms of other types of fat and dietary fatty acid patterns ($p>0.05$). No correlation was observed between the sCD36 receptor levels and dietary fatty acid type in individuals with MetS ($p>0.05$). *Conclusions*: Soluble CD36 level is not a possible biomarker for MetS owing to its similar levels in these patients.

Key words: Metabolic Syndrome, CD36, Lipids

Introduction

Metabolic syndrome (MetS) is a disease in which at least three of the following complications occur: impaired glucose regulation, abdominal obesity, high blood pressure, and dyslipidemia (high triglyceride and low HDL) (1). The worldwide prevalence of MetS ranges from 24% to 51% (2,3). The etiopathogenesis of MetS components is not dependent on a single factor. Although the pathophysiological mechanisms that cause MetS cannot be fully explained, obesity due to reasons such as sedentary life, unbalanced diet, and high calorie intake caused by modern life triggers the emergence of MetS and aggravates its course (4). Especially in the presence of abdominal obesity, insulin sensitivity decreases owing to a decrease in adiponectin levels. In addition, dietary fatty acids, which are the main regulators of glucose and lipid homeostasis, play an important role in the development of MetS (5). While dietary unsaturated fatty acids increase insulin sensitivity and reduce the risk of MetS by stopping the inflammatory process, saturated fatty acids (SFA) contribute to the formation of MetS (5,6).

It is a member of the CD36 B2 scavenger receptor family and is also known as fatty acid translocase (FAT), glycoprotein III b (GPIIIb), and glycoprotein IV (GPIV). CD36 is secreted by many tissues including adipocytes, platelets, myocytes, macrophages,

endothelial cells, and liver cells. Oxidized LDL, long-chain fatty acids (LCFAs), and phospholipids are important ligands of CD36 (7). The most important role of CD36 in lipid metabolism is the transport of LCFA. CD36 is required for LCFA uptake into cardiomyocytes, adipocytes, enterocytes, and skeletal myocytes. CD36 is involved in long-term regulation of LCFA uptake by the plasma membrane. Dietary fat and fatty acids affect CD36 levels, particularly in the liver, intestines, and macrophages. Dietary SFA and trans fatty acids negatively affect CD36-mediated cholesterol homeostasis, whereas monounsaturated fatty acid (MUFA) positively contribute to this balance. The effects of polyunsaturated fatty acid (PUFA) vary depending on the amount of n-3 and n-6 ingested and the n-3:n-6 ratio (8).

CD36 receptors play an important role in metabolic disorders such as impaired glucose regulation and insulin resistance, as they control the transport and uptake of LCFAs (9). The CD36 receptor, which plays a role in the uptake of LCFA and shows high affinity for LCFA, is present in taste cells and contributes to energy balance by providing appetite control. Therefore, it is suggested to play an important role in the mechanism of obesity (10). In addition, plasma CD36 levels have been reported to increase during hyperlipidemia (11). All these mechanisms show that the CD36 receptor may play an active role in hyperglycemia, abdominal obesity, hyperlipidemia, and MetS where all these are together (9-11).

When the literature is examined, studies have examined the change in CD36 levels in individuals with impaired glucose metabolism, but the results are contradictory (12-17). In addition to studies showing that defects in fatty acid and lipid transport in the absence of CD36 receptor levels contribute to insulin resistance (12,13), other study has reported that these two factors are not related (14). On the other hand, studies have shown that the level of CD36 receptors is higher in individuals with impaired glucose metabolism than in healthy individuals (15-17). Studies have examined the relationship between atherosclerosis (18), diabetes (12,14,16,17), insulin resistance (15), abdominal obesity (19), and sCD36 levels. However, studies evaluating CD36 levels in patients with MetS in which these components are combined are very limited (20,21). In addition, no study has compared the dietary fat and fatty acid types of individuals with MetS with healthy individuals and examined the relationship between fatty acid types and CD36. Therefore, in this study, we aimed to compare the dietary fat and fatty acid types of MetS and healthy individuals and determine whether there is a relationship between dietary fat and fatty acid types and sCD36 levels.

Methods

Study design and participants

This case-control study was conducted on 33 individuals diagnosed with MetS according to the IDF-2005 MetS diagnostic criteria aged 18-65 years, who were admitted to the Internal Medicine Outpatient Clinic of Ordu University Training and Research Hospital between May 2019-November 2019, and 32 healthy individuals with a similar age and body mass index (BMI) to individuals with MetS. The study started with 81 participants but was completed by 65 people because the individuals did not comply with the study criteria, and there were participants who wanted to leave the study. Individuals who were pregnant and lactating, used cigarettes and alcohol, had any chronic disease other than MetS, used regular medication, regularly took nutritional supplements, and had experienced a weight change in the last three months were not included in the study. Institutional permission was obtained from the Ordu Provincial Health Directorate before starting the study (letter dated December 18, 2018 and numbered 35335454-799). Ethics committee approval was received from Ordu University Clinical Research Ethics Committee (Ethics Committee decision dated February 21, 2019, and numbered 2019/41). Written informed consent was obtained from all participants after the researcher provided detailed information about the study. This study was conducted in accordance with the principles of the Declaration of Helsinki.

Demographic and anthropometric characteristics

The sociodemographic characteristics of the participants (sex, age, educational status, occupation, and marital status) and their disease history were

determined using a personal information form prepared by the researchers.

Body weight measurements of all participants included in the study were made with the help of an electronic scale (Tanita BC 418) with an accuracy of 0.1 kg, on an empty stomach and wearing light clothes. The participants' body composition (fat mass, muscle mass and body water) was also determined using the same electronic scale. The height of the participants was brought to the Frankfurt plane without shoes and with the help of a stadiometer; waist and hip circumferences were measured using a nonstretchable tape measure (22). Waist circumference was measured between the lowest bone of the rib and the cristailiac; by measuring the circumference passing through the middle point, hip circumference was measured by standing on the side of the individual and measuring the circumference passing through the widest point with a nonstretchable tape measure (23). BMI was calculated by dividing body weight (kg) by height (cm) squared (BMI: body weight/height squared) (24).

Assessment of dietary intake

The nutritional habits of the participants (main meal consumed, snack consumed, skipping meals status, skipped meals, snack foods, eating outside situations, and fast food eating frequency) were determined using the information form prepared by the researcher. Fast food is defined as easily prepared processed food that is served as a quick meal or can be taken away (25). We defined eating outside as eating meals prepared away from home outside the home. The daily food intake of the participants was determined using 3 day food records. The foods recorded in the food consumption registration form were entered into the Nutrition Information System (BEBIS) 8.1, and the daily fatty acid types and amounts of the participants were calculated in detail. The food frequency questionnaire was used to determine the type and amount of fat consumed by participants.

Biochemical and clinical parameters

The blood pressure and biochemical parameters (fasting glucose, HbA1c, insulin, HOMA-IR, triglyceride, total cholesterol, HDL, LDL, AST, ALT, and CRP) of the participants were obtained from patient records. To determine the sCD36 levels, one tube of blood was collected from each individual after 8 hours of fasting, and the serum was separated by centrifugation. The separated serums were stored at -80°C until analysis. Serum CD36 levels were determined by using commercial kits (Elabscience Human GP4/CD36 Platelet Membrane Glycoprotein 4 ELISA Kit) by ELISA method, and analyzed in accordance with the user manual given by the company in Hacettepe University Orhan Köksal Research Laboratory.

Statistical analysis

The SPSS 20 package program was used for the statistical analysis of this study. Based on a similar study (26), power (G*Power 3.1.9.2) analysis was performed, and the sample size was calculated. In the evaluation, when Δ (Effect Size): 0.75, SD (Standard Deviation): 0.80, the number of samples determined for power: 0.80, β: 0.20, and α: 0.05, was determined for each group, and a minimum of 29 for one group and a total of 58 for the two groups. The distribution of all data was evaluated using the normality test. In the tables, mean ± standard deviation values were used for normally distributed data, and median (minimum-maximum) values were used for non-parametric distributed data. While performing the statistical analysis of the data, parametric tests were used for normally distributed data and non-parametric tests were used for non-normally distributed data. Statistical significance was set at $p<0.05$.

Results

General characteristics, disease history and eating habits

In this study, 33 individuals with MetS were included in the MetS group and 32 healthy individuals were included in the control group. There were no statistically significant differences between the groups in terms of sex, age, marital status, educational status and occupation ($p>0.05$); however, family history of heart disease and obesity was higher in the MetS group ($p<0.05$) (Table 1).

Table 1. General characteristics, disease history, physical activity and eating habits.

	MetS	Control	
	n (%)	n (%)	p
Sex			
Female	25 (75.8 %)	25 (78.1 %)	0.821[a]
Male	8 (24.2 %)	7 (21.9 %)	
Age			
Mean±SD	43,82 ± 10,16	44,38 ± 9,93	
Med (min-max)	46 (21-62)	45.5 (21-62)	0.990 [c]
Marital status			
Married	26 (78.8 %)	22 (68.8 %)	0.653[a]
Single	5 (15.2 %)	7 (21.9 %)	
Divorced	2 (6.1 %)	3 (9.4 %)	
Education Status			
Primary school	3 (9.1 %)	4 (12.5 %)	0.721[a]
Middle school	6 (18.2 %)	3 (9.4 %)	
High school	11 (33.3 %)	13 (40.6 %)	
University	13 (39.4 %)	12 (37.5 %)	
Occupation			
Housewife	15 (45.5 %)	14 (43.8 %)	0.881[a]
Self-employment	6 (18.2 %)	4 (12.5 %)	
Officer	10 (30.3 %)	11 (34.4 %)	
Other	2 (6.1 %)	3 (9.4 %)	
Disease history of family			
Diabetes	20 (60.6 %)	14 (43.8 %)	0.174[a]
Hypertension	18 (54.5 %)	12 (37.5 %)	0.168[a]
Hearth disease	14 (42.4 %)	5 (15.6 %)	**0.018***[a]
Obesity	29 (87.9 %)	15 (46.9 %)	**0.000***[a]
Main meal consumed			
Mean±SD	2,64 ± 0,49	2,66 ± 0,48	
Med (min-max)	3 (2-3)	3 (2-3)	0.868[b]
Snack consumed			
Mean±SD	1,18 ± 0,85	0,75 ± 0,88	
Med (min-max)	1 (0-3)	0 (0-2)	**0.048***[b]
Skipping meal status	27 (81.8 %)	32 (100.0 %)	**0.011***[a]
Skipped meal			
Breakfast	9 (27.32 %)	6 (18.8 %)	0.276[a]
Lunch	11 (33.3 %)	12 (37.5 %)	
Dinner	-	-	
Snacks	7 (21.2 %)	14 (43.8 %)	

Consumed snack foods			
Packaged foods	11	7	0.157[a]
Nuts	5	5	
Pastries	5	5	
Fresh fruits	5	3	
Dry fruits	1	2	
Yoghurt	-	1	
Eating outside	27 (81.8 %)	32 (100.0 %)	**0.041***[a]
Fastfood eating frequency			
More than once a week	13 (39.4 %)	4 (12.5 %)	**0.007***[a]
1 time in 15 days	4 (12.1 %)	1 (3.1 %)	
Once a month	4 (12.1 %)	14 (43.8 %)	
Less than 1 time per month or none	12 (37.4 %)	13 (40.6 %)	

MetS, metabolic syndrome.
* P< 0.05 is significant.
[a] Chi-square test was used for categorical data
[b] Mann-Whitney test was used for numerical and non-normally distributed data
[c] Independent sample t test was used for numerical and normally distributed data

When the data on the nutritional habits of the participants were compared, it was determined that individuals with MetS had more snacks, a lower frequency of eating outside the home and skipping meals, and a higher frequency of eating fast food (p<0.0.5). There was no significant difference between the groups in terms of the foods consumed as snacks or number of main meals consumed daily (p>0.05) (Table 1).

Anthropometric and biochemical measurements

Anthropometric measurements of the groups are shown in Table 2. Since we matched the BMI between the groups during data collection, all anthropometric measurements of the participants, including BMI, waist circumference, hip circumference, waist/hip ratio, and body fat percentage were similar (p>0.05) (Table 2).

When the blood pressure and biochemical measurements of the participants were compared, it was found that the systolic and diastolic blood pressure, fasting glucose, HbA1c, insulin, HOMA-IR, triglyceride, total cholesterol, LDL, AST, ALT and CRP levels were significantly higher in the MetS group (p<0.05). There were no statistically significant differences in HDL or serum sCD6 levels between the groups (p>0.05) (Table 2).

Dietary fat, fatty acids and relation between CD36

Data on the type and amount of fat consumed by the participants are shown in Table 3. The most consumed oil types in the MetS group were sunflower oil (33.18 g/day), corn oil (17.75 g/day) and butter (16.48 g/day); sunflower oil (30.97 g/day), butter (16.64 g/day) and hazelnut oil (10.63 g/day) in the healthy control group. When the types of fat consumed by the groups were compared, it was found that individuals with MetS consumed more corn oil and tail fat and less olive oil (p<0.05), and no difference was found between the groups in terms of other types of fat (p>0.05) (Table 3). While the total fat intake of individuals with MetS was 130.33 g, the cholesterol amount was 428.96 mg, and the percentage of energy from fat was 37.02%, the total amount of fat they take with diet was 148.12 g, the amount of cholesterol was 443.79 and the percentage of energy from fat was 38.41% in the control group. There was no statistically significant difference between the dietary intakes of total fat and

Table 2. Anthropometric and Biochemical Measurements.

	MetS	Control	
	Mean ± SD Med (min-max)	Mean ± SD Med (min-max)	p
BMI (kg/m^2)	35.68 ± 6.06	35.82 ± 5.83	0.926
Waist circumference (cm)	104.09 ± 11.3	103.63 ± 10.65	0.865
Hip circumference (cm)	116.09 ± 10.74	116.19 ± 9.21	0.969
Waist/hip ratio (cm/cm)	0.9 ± 0.1	0.9 ± 0.09	0.850
Body fat percentage (%)	35.94 ± 6.72	38.11 ± 5.04	0.147
Systolic Blood Pressure (mmHg)	13.03 ± 0.95	12.46 ± 0.62	**0.007***
Diastolic Blood Pressure (mmHg)	9.42 ± 1.36	8.35 ± 0.78	**<0.001***
Fasting Glucose (mg/dL)	115.82 ± 13.51	91.75 ± 5.74	**<0.001***
HbA1c (%)	6.1 ± 0.75	5.54 ± 0.25	**<0.001***
İnsulin (ng/ml)	26.48 ± 13.78	14.82 ± 8.34	**<0.001***
Homa-IR (index)	7.73 ± 4.71	3.4 ± 2.04	**<0.001***
Triglyceride (mg/dL)	196.7 ± 153.51	114.38 ± 53.76	**0.006***
Total Cholesterol (mg/dL)	204.97 ± 38.38	175.13 ± 30.56	**0.001***
HDL (mg/dL)	48.55 ± 10.95	51.56 ± 13.22	0.320
LDL (mg/dL)	120.91 ± 33.48	100.68 ± 26.50	**0.009***
AST (U/L)	21.7 ± 6.48	16.81 ± 3.39	**<0.001***
ALT (U/L)	26.82 ± 12.3	15.75 ± 4.41	**<0.001***
CRP (mg/dL)	0.6 ± 0.49	0.37 ± 0.3	**0.029***
CD36 (ng/mL)	0.22 ± 0.07	0.24 ± 0.17	0.541

MetS, metabolic syndrome.
* P< 0.05 is significant.
Independent sample t test was used

cholesterol and the percentage of energy from fat and fatty acid types (p>0.05) (Table 3).

Correlation analysis was performed to determine whether there was a relationship between serum sCD36 levels and dietary total fat, cholesterol, amount of different fatty acids, and percentage of energy from fat. No correlation was found between serum sCD36 levels and dietary total fat, cholesterol, amount of different fatty acids, or percentage of energy from fat (p>0.05) (Table 4).

Discussion

According to our results, although individuals with MetS have higher fasting glucose, HbA1c, insulin, homa-IR, triglyceride, total cholesterol, LDL, AST, ALT and CRP levels than healthy individuals with similar BMI, CD36 levels are similar. Compared to the healthy control group, it is observed that individuals with metabolic syndrome consume more corn oil and tail fat and less olive oil. There is no relationship between dietary fatty acids and sCD36 levels.

It is unclear whether individuals with MetS have higher sCD36 levels than healthy individuals (12-17). Some studies indicate that CD36 secretion from monocytes and macrophages increases in the presence of hyperglycemia and hyperlipidemia, which are components of MetS (18, 20, 21). Other studies have shown that the level of CD36 receptors is higher in individuals with impaired glucose metabolism than in healthy individuals (15, 16, 17). Zhang et al. reported no correlation between CD36 and glucose regulation (14). In a study by Castelblanco et al., who

Table 3. The Types and Amounts of Fat Consumed and Dietary Fatty Acid Pattern.

	MetS	Control	
	Mean ± SD Med (min-max)	Mean ± SD Med (min-max)	p
Olive oil (g)	1.26 ± 4.52	6.62 ± 10.42	**0.011***
Hazelnut oil (g)	4.56 ± 7.41	10.63 ± 17.32	0.075
Sunflower oil (g)	33.18 ± 15.04	30.97 ± 18.04	0.592
Corn oil (g)	17.75 ± 18.78	5.7 ± 13.7	**0.004***
Butter (g)	16.48 ± 10.77	16.64 ± 12.63	0.957
Tallow (g)	0.03 ± 0.07	0.03 ± 0.1	0.744
Tail fat (g)	0.1 ± 0.15	0.03 ± 0.1	**0.004***
Margarine (g)	0.54 ± 0.79	0.45 ± 1.4	0.764
Fat (g)	130.33 ± 31.4	148.12 ± 49.08	0.172
Fat (%)	37.02 ± 5.1	38.41 ± 3.96	0.321
Cholesterol (mg)	428.96 ± 186.22	443.79 ± 206.31	0.806
PUFA (g)	27.99 ± 11.23	32.47 ± 18.96	0.360
PUFA (%)	7.97 ± 2.26	8.12 ± 2.18	0.825
MUFA (g)	47.83 ± 12.49	55.15 ± 20.8	0.177
MUFA (%)	13.95 ± 3.09	14.65 ± 3.57	0.496
SFA (g)	44.34 ± 11.6	48.35 ± 11.81	0.269
SFA (%)	12.87 ± 2.44	13.02 ± 2.07	0.825
SCFA (g)	1.87 ± 0.68	1.98 ± 0.68	0.574
MCFA (g)	1.96 ± 0.6	2.09 ± 0.54	0.447
LCFA (g)	111.78 ± 28.9	126.38 ± 43.55	0.209
n-3 (g)	3.33 ± 1.4	4.41 ± 4.86	0.343
n-6 (g)	23.35 ± 9.98	26.65 ± 14.65	0.400
n-6/n-3 ratio	7.34 ± 2.41	7.45 ± 3.53	0.910

MetS, metabolic syndrome.
* P< 0.05 is significant.
Independent sample t test was used

examined the relationship between sCD36 and diabetes, no significant difference was found in sCD36 levels between patients with type 1 or type 2 diabetes and healthy controls. However, in the present study, CD36 levels were increased in individuals with dyslipidemia (17). In this study, no significant differences were found between groups in terms of sCD36 levels. This similarity in CD36 levels between the groups might be explained by the enrolment of newly diagnosed patients who have not begin their treatment yet. Additionally, the similarity in the types of fat consumed by the groups may explain this situation. High fasting blood glucose, blood pressure and triglyceride levels, low HDL levels, and presence of abdominal obesity are among the diagnostic criteria for MetS (27). Therefore, in the study, these parameters were expected to be higher than those in the control group.

The type and amount of dietary fat is important for the development and progression of diseases (28). Studies have shown that olive oil consumption, a good source of MUFAs, is more protective against insulin resistance than n-6 PUFA consumption (29), reduces the need for insulin for glucose regulation (30), improves systemic inflammation (31), and reduces the prevalence

Table 4. The Relation Between sCD36 and Dietary Fatty Acid Pattern.

	MetS		Control	
	r	p	r	p
Fat (g)	0.034	0.888	-0.006	0.978
Fat (%)	-0.098	0.681	0.079	0.720
Kolesterol (mg)	0.325	0.162	-0.146	0.505
PUFA (g)	0.254	0.280	-0.005	0.983
PUFA (%)	0.285	0.224	0.068	0.759
MUFA (g)	-0.146	0.539	0.145	0.509
MUFA (%)	-0.234	0.322	0.219	0.316
SFA (g)	0.026	0.912	-0.230	0.290
SFA (%)	-0.071	0.767	-0.369	0.083
SCFA (g)	-0.115	0.630	-0.309	0.151
MCFA (g)	-0.035	0.884	-0.337	0.116
LCFA (g)	0.074	0.756	0.029	0.895
n-3 PUFA (g)	0.240	0.308	-0.126	0.567
n-6 PUFA (g)	0.236	0.316	0.045	0.837
n-6/n-3	0.021	0.931	0.371	0.082

MetS, metabolic syndrome
* P< 0.05 is significant
Pearson correlation test was used

of MetS (32). Yubero-Serrano et al. reported that olive oil consumption has a protective effect against MetS and type 2 diabetes by regulating inflammation, oxidative stress, coagulation, platelet aggregation, fibrinolysis, and endothelial functions (33). In addition, diets containing high MUFA levels can reduce LDL concentrations without lowering HDL levels compared with diets containing PUFA (34). Studies examining the relationship between olive oil consumption and blood pressure also show that olive oil consumption reduces the risk of high blood pressure (35, 36). However, in a study by Miller et al., substitution of dietary saturated fatty acids (SFA) with PUFA in individuals with MetS was associated with a greater reduction in triglycerides and improvement in endothelial function compared to MUFA. Accordingly, PUFAs may be preferred for reducing the risk of cardiovascular diseases (37). High serum SFA levels are strongly associated with insulin resistance, inflammation and MetS (38). In this study, olive oil consumption by individuals with MetS was significantly lower than that of the healthy control group, whereas consumption of corn oil and tail oil was higher. Considering these data and the results of previous studies, olive oil consumption, which is a good source of MUFA, may plays a more protective role in the formation of MetS than corn oil, which is a source of n-6 PUFA. Additionally, tail fat which is a source of SFA may be considered a risk factor for the formation of MetS components.

Some studies have shown that the sCD36 receptor is involved in LCFA uptake by hepatocytes, adipocytes, myocytes, and endothelial cells (39,40). It is estimated that CD36 receptors regulate various mechanisms such as vascular contraction, angiogenesis, and tissue glucose metabolism by controlling fatty acid uptake (41,42). All these mechanisms suggest that the CD36 receptor may play an active role in hyperglycemia, hyperlipidemia, abdominal obesity and hence MetS (39-42). However, high levels of saturated fatty acids in the cell membrane have negative effects on insulin activation. Increased saturated fatty acid levels impair GLUT-4 gene expression in skeletal muscles, limiting glucose entry into the muscle and impairing insulin signalling. Consequently, hyperglycemia occurs. High levels of PUFAs in the cell membrane, particularly a high ratio of n-3: n-6 fatty acids, enhance insulin sensitivity (16,43). In this study, no significant differences were found between the dietary fatty acid types of groups. In addition, no relationship was found between the dietary fatty acid type and sCD36 levels. The reason why the fatty acid types in the diets of individuals with MetS are similar to healthy individuals may be that the age and BMI of the groups were similar. Therefore, the lack of difference between the two groups may indicate that the disease process may be associated with other processes independent of CD36 and fatty acids.

Consumption of foods high in sugar contributes to the development of MetS (44). Healthy eating habits (predominantly consumption of fruits, vegetables, low-fat milk, nuts, poultry, fish, and whole-grain legumes) reduce the risk of MetS, whereas a western-style diet (predominantly consumption of refined grains, sugary drinks, fast foods, foods high in sugar and packaged ready-made foods) increases the risk of MetS (45). Although this study showed that individuals with MetS consumed more snacks, foods high in sugar and packaged ready-made foods were preferred.

Contrary to expectations, the frequency of eating outside the home was lower in individuals with MetS than that in healthy individuals. However, the fact that the foods that individuals with MetS prefer outside are mostly foods high in sugar and packaged ready-made foods explains this situation.

Individuals with a family history of MetS are known to have a high risk of developing MetS, and therefore, cardiovascular disease (46,47). A previous study showed that the prevalence of hypertension was higher in individuals with a family history of hypertension, and the presence of hypertension in family history was found to be associated with obesity, central obesity, and MetS (48). In the present study, individuals with MetS had a history of heart disease and obesity in their families, and there were no significant differences between the groups in terms of history of diabetes or hypertension in their families. Therefore, the risk of MetS may be higher in individuals with family history of heart disease or obesity.

Limitations and strengths of the study

This study has some limitations. One of the limitations of the study is using the information form prepared by the researcher to assess dietary intake rather than using a validated form. The other limitation of the study is that the data on food consumption of the participants were self reported. The last limitation of the study is that only individuals who visited to the internal medicine outpatient clinic of a hospital were included. Therefore, the results obtained reflect the region where the research was conducted. The strengths of the study are that individuals with metabolic syndrome and healthy individuals included in the study were examined by a physician and referred to the diet outpatient clinic and the study was conducted face to face. This study will shed light on future studies on metabolic syndrome and sCD36.

Conclusion

This is a rare nutritional study examining the relationship between sCD36 and dietary fatty acid patterns in individuals with MetS. There was no significant difference in sCD36 levels between individuals with MetS and age-, sex- and BMI-matched healthy individuals. In addition, there was no significant relationship between the sCD36 levels and dietary fatty acid patterns. According to these results, the disease process may be related to other processes independent of CD36 and fatty acids. More comprehensive clinical nutritional studies examining the relationship between CD36 levels and dietary fatty acid patterns are required to obtain stronger evidence.

Acknowledgements: We thank the study participants, dieticians working in the nutrition outpatient clinic where the study was conducted and Tuğçe TEKİN GÜLER. for helping perform the sCD36 analysis of the serum samples.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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