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Relationship between yoghurt consumption and components of metabolic syndrome: A cross-sectional study in the west of Iran

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ABSTRACT

The association between total, low-fat, and whole-fat yoghurt consumption with the risk of metabolic syndrome (MetS) and its components was assessed using a cross-sectional study for which 973 adults were selected using a randomised-multistage-cluster sampling method. Dietary intake was assessed using of a validated, 168 food-item, self-administrated, semi-quantitative food-frequency-questionnaire. Consumption (servings per week) of yoghurt among individuals with and without MetS were 4.5 ± 3.9, and 5.8 ± 5.9, respectively ($p < 0.001$), and for high-fat yoghurt were 2.1 ± 2.9, and 3.1 ± 5.8, respectively ($p < 0.001$). Yoghurt consumption was inversely associated with the risk of high triacylglycerol concentration after adjustment for confounders. After adjustment for potential confounders, by differentiation between low- and high-fat yoghurts, there was no significant association with MetS. However, low-fat yoghurt was significantly associated with abdominal adiposity and high fasting plasma glucose (FPG). Low-fat yoghurt consumption was associated with a lower risk of FPG and abdominal adiposity.

1. Introduction

Metabolic syndrome (MetS) is defined as a complex of metabolic disorders, including central obesity, insulin resistance and hyperglycaemia, dyslipidaemia, and hypertension (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004). Unfortunately, the incidence of type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) are rapidly increasing around the world (Guariguata et al., 2014; Mahmood, Levy, Vasan, & Wang, 2014), and people with metabolic syndrome are at increased risk of diabetes and CVD (Eckel, Grundy, & Zimmet, 2005; Simmons et al., 2010).

In previous studies, the prevalence of metabolic syndrome among Iranian adolescents and adults was respectively, 10 and 30 percent (Delavari, Forozanfar, Alikhni, Sharian, & Kelishadi, 2009; Esmaillzadeh, Mirmiran, Azadbakht, Etemadi, & Azizi, 2006), which is higher than the prevalence in developed countries (Mozumdar & Liguori, 2011). In several studies, dairy consumption was inversely associated with the occurrence of one or several components of metabolic syndrome (Babio et al., 2015; Dugan, Barona, & Fernandez, 2014; Kim, 2013; Samara et al., 2013; Sayón-Orea et al., 2015). However, other studies have shown no association between dairy consumption and the prevalence or incidence of the MetS (Snijder et al., 2007, 2008).

There are a few epidemiological studies that have differentiated between types of dairy products, specifically yoghurt (including skim, low or full fat yoghurt) (Astrup, 2014; Donovan & Shamir, 2014). Although the micronutrient composition of yoghurt is similar to that of milk, it is highly concentrated with proteins, vitamins (such as B2 and B12) and minerals (including calcium, magnesium, potassium, and zinc) (Wanga, Livingston, Fox, Meigs, & Jacques, 2013). In addition, yoghurt contains several kinds of bacteria, such as lactic acid bacteria (Shihata & Shah, 2000).

The health benefits of yoghurt have been extensively promoted and studied in animal samples (Broussalian & Westhoff, 1983), but limited epidemiologic evidence is available and potential mechanisms are unknown (Liu et al., 2005; Recker, Bammi, Barger-Lux, & Heaney, 1988). In the study by Abreu et al. (2014) no association was found between cardiometabolic risk (CMRS) score and yoghurt intake, whereas milk intake was inversely related to CMRS in adolescents.

Although there is conflicting evidence in connection between yoghurt consumption and T2DM (Chen et al., 2014), it is inversely associated to CVD risk factors and metabolic syndrome (Ivey et al.,

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2011; Wanga et al., 2013). It is possible that some components of dairy products, such as the type and fat level, modify the effect of dairy intake on T2DM.

The purpose of this study was to determine the association between yoghurt consumption with metabolic syndrome and its components, including waist circumference, blood pressure, fasting plasma glucose (FPG), high-density lipoprotein cholesterol (HDL-C), and prevalence of the metabolic syndrome in Khorramabad's adults.

2. Materials and methods

2.1. Subjects

In this cross-sectional study of a representative sample of Khorramabad residents, 1200 individuals aged ≥18 y were selected by a multistage cluster, random sampling method from July 2011 to February 2012. A total of 16 treatment and health centres were randomly selected. From registered households in these centres, 1200 randomly selected subjects were invited to participate in this study. Persons on diet, taking certain medication (e.g., anti-hypertensive and anti-hyperlipidaemia drugs), in pregnancy or lactation or with reported daily energy intakes outside the range of 800–4200 kcal d^{-1} were excluded. Some of the completed questionnaires contained problems and were not usable. Therefore, 1009 subjects (273 men and 736 women) aged 18–75 y remained for the study. Thirty six persons did not end the study because of personal reasons (e.g., lack of time or unexpected reasons that forced them to cancel continuation the study). Finally 973 subjects with complete data remained for statistical analysis.

Each subject provided written informed consent. The protocol of the study was approved by the ethic committee of Lorestan University of Medical Sciences (63/89-2010).

Participants were asked to complete a package containing an informed consent form, an exclusion criteria form, a demographic form, an International Physical Activity Questionnaire (IPAQ, 2006), and a semi-quantitative food-frequency questionnaire (SQ-FFQ; Esmaillzadeh et al., 2007). Three workshops with regard to the package were carried out for 5 dieticians who then went to participants homes and trained the participants on how to complete the package. After one week, the dieticians returned to the homes, gathered the completed packages, and then checked them. If any response to each question or food item was ambiguous the participant was called to resolve the problem; if resolution was not possible the questionnaire was omitted from the study.

2.2. Dietary assessment

Dietary intake was assessed with the use of a validated, 168 food-item, self-administrated SQ-FFQ. The FFQ consisted of a list with a standard serving size (Willett format). Participants were asked to report their frequency of intake of each food item during the previous year on a daily, weekly, or monthly basis. Yoghurt items were categorised as 4-subgroups that are in Iranian supermarkets: creamy (>5% fat), high-fat (3–5%), low-fat (1–3%), and fat-free (<1%). Each food item provided 9 choices of possible responses, ranging from “never or less than once a month” to “six or more times per day”. The selected frequency choice indicated by the participants for each food/beverage was converted to daily intake, and then portion sizes of consumed foods were converted from household measures to grams. High-fat and low-fat yoghurt consumption was calculated as the summation of ≥3% and <3% fat, respectively. All the completed questionnaires were controlled by trained dieticians.

2.3. Assessment of other variables

Subjects were invited to come to an obesity clinic in city centre to measure anthropometric indices. Weight was measured by a trained nurse using digital scales at mid-afternoon, while subjects were minimally clothed and not wearing shoes. Height was measured using a stadiometer while the subjects were standing, wearing no shoes, and held the shoulders in a relaxed position. Body mass index (BMI) was estimated as the weight (in kg) divided by the square of the height (in m^2). Waist circumference (WC) was measured at the narrowest level, and hip circumference was measured at the maximum, with the use of an upstretched tape with no pressure to body surface. Measurements were recorded to the almost 0.1 cm and 0.1 kg.

Extra information about age, gender, marital status (single or married), smoking status (i.e., non-smoker, current smoker, ex-smoker), alcohol intake (yes or no), monthly income (ranging from ≤ US$300 to ≥ US$900), education level (i.e., illiterate, ≤ 12 years, and >12 years), medical history and current use of medications was obtained with the use of questionnaire eliciting information. Physical activity was assessed by using the IPAQ (IPAQ, 2006). The short version of IPAQ (seven items) was used, providing information on weekly time spent walking, in vigorous activity, moderate–intensity activity, and in sedentary activity. This was finally expressed as metabolic equivalent hour per week (MET-h wk^{-1}) and the sum analysed as total physical activity. Blood pressure was measured twice after the participants sat for 15 min, using a mercury sphyngometer (ERKA, Germany). Subjects were referred to a biochemical laboratory near to the obesity clinic for blood sample collection. Fasting blood samples for the measurement of glucose and lipid concentrations were drawn after 12 h overnight fasting by an expert laboratory technician. Plasma glucose concentration was measured on the day of blood collection using a commercial kit based on an enzymatic colorimetric method using glucose oxidase (Pars Azmoon Inc., Tehran, Iran). High-density lipoprotein cholesterol (HDL-C) was measured after precipitation of the apolipoprotein B-containing lipoproteins with phosphotungstic acid (Pars Azmoon Inc., Tehran, Iran). Serum triacylglycerol concentrations were assayed with a commercial kit based on enzymatic colorimetric method with glycerol phosphate oxidase (Pars Azmoon Inc., Tehran, Iran). Blind duplicates were used for quality control for all analyses.

2.4. Definition of terms

Metabolic syndrome was defined as the presence of ≥3 of the following 5 risk factors as recommended by American Heart Association (Alberti et al., 2009): (i) abdominal obesity (waist circumference ≥ 102 cm in men and ≥88 cm in women), (ii) elevated blood pressure (≥130/85), (iii) high serum triacylglycerol concentrations (≥150 mg dL^{-1}), (iv) low serum HDL-C (<40 mg dL^{-1} in men or < 50 mg dL^{-1} in women), and (v) high FPG (≥100 mg dL^{-1}).

2.5. Statistical analysis

SPSS software (version 19, SPSS Inc., Chicago, IL, USA) was used to conduct data analyses. To describe the data, the frequency distribution tables and parameters such as mean, standard deviation (SD) and percentage were used. Chi-square test and t-test were used to examine significantly differences in the distribution of subjects regard to quality and quantity variables, respectively. A logistic regression model was used to provide odds ratio (OR). The first adjustment was for age, cigarette smoking (yes or no), physical activity (MET-h wk^{-1}), and history of diabetes and heart disease

(yes or no). The second adjustment was for BMI (kg $(m^2)^{-1}$) to examine whether the relation was mediated by obesity. In the third model energy intake (kcal d^{-1}) was also added. Last, an adjustment for milk and cheese intake was made.

Finally, the OR and 95% confidence interval (95% CI) were calculated for independent and confounding variables. Statistically significant differences were considered to be at a P-value ≤ 0.05.

3. Results

3.1. Associations between basic characteristics and dietary intake of subjects with prevalence of the metabolic syndrome

The prevalence of metabolic syndrome in the study population was 29%, and it was more common in individuals with a family history of diabetes than those without a family history of diabetes (76% versus 24%).

The mean ± SD of age, BMI, physical activity and dietary intakes and the distribution of participants with regard to smoking, gender, history of diabetes and history of heart disease among subjects with or without metabolic syndrome are shown in Table 1. Significant differences were observed between the age, BMI, history of diabetes, history of heart disease, and physical activity of those who have metabolic syndrome in compared with those who have not ($P < 0.05$), However, no significant differences between two groups in terms of gender, smoking, and dietary intake were observed.

3.2. Comparison between yoghurt consumption and the risk of metabolic syndrome and its components

The rate of yoghurt consumption according to metabolic syndrome and its components status among participants with and without metabolic syndrome are shown in Table 2. Subjects with metabolic syndrome had lower yoghurt consumption. Yoghurt consumption was inversely associated with the risk of metabolic syndrome and high serum triacylglycerol concentrations (Table 2).

Table 1
The status of age, gender, BMI, physical activity, history of heart disease and diabetes, use of medication and smoking among subjects with or without metabolic syndrome (MetS).[a]

Variable	MetS status		
	With N = 282	Without N = 691	Total
Age (y)	40.6 ± 9.7***	31.2 ± 9.9	33.9 ± 10.8
Gender			
Male (%)	20.9	25.8	24.4
Female (%)	79.1	74.2	75.6
BMI (kg $(m^2)^{-1}$)	30.7 ± 4.5***	24.9 ± 4.3	26.7 ± 5.1
Physical activity (Met h wk^{-1})	39.6 ± 63.9***	53.9 ± 78.3	49.6 ± 74.6
History of heart disease (%)	5.7*	2.7	3.6
History of diabetes (%)	6.7***	0.9	2.6
Current daily smoker (%)	2.5	2.5	2.5
Dietary intake			
Total energy (kcal d^{-1})	1871 ± 1285	1907 ± 781	1897 ± 955
Carbohydrate (% total energy)	48.7	50.3	49.7
Protein (% total energy)	15.2	14.7	14.9
Fat (% total energy)	36.1	35	35.4
Fibre (g d^{-1})	12.1 ± 12.2	12.8 ± 7.7	12.6 ± 11.1
Calcium (mg d^{-1})	908 ± 2217	875 ± 493	885 ± 1263

[a] Abbreviations are: MetS, metabolic syndrome; Met, metabolic equivalent. Asterisks indicate significant difference: *$P < 0.05$; ***$P < 0.001$.

Table 2
Comparison between yoghurt consumption and the risk of metabolic syndrome and its components.[a]

Parameter	High-fat yoghurt		Low-fat yoghurt		Total yoghurt	
	Mean	SD	Mean	SD	Mean	SD
MetS status						
With N = 282	2.1***	2.9	2.42	2.1	4.5***	3.9
Without N = 691	3.1	5.8	2.74	3.2	5.8	7.6
WC status						
High N = 422	2.7	4.8	2.65	2.7	5.3	5.9
Threshold value N = 696	2.9	5.4	2.65	3.1	5.7	7.4
BP status						
High N = 277	2.3*	3.1	2.59	2.6	4.9	4.6
Threshold value N = 696	3.1	5.8	2.67	3	5.7	7.5
HDL-C status						
High N = 356	2.9	5.7	2.69	3	5.6	7.4
Threshold value N = 696	2.7	4.1	2.58	2.8	5.3	5.5
Serum TG status						
High N = 305	2***	2.7	2.47	2.3	4.5**	3.8
Threshold value N = 696	3.2	5.9	2.73	3.1	5.9	7.8
FPG status						
High N = 138	2.3	4.2	2.33	2.4	4.7	5.8
Threshold value N = 696	2.9	5.3	2.7	2.9	5.6	6.9

[a] Abbreviations are: MetS, metabolic syndrome; WC, waist circumference; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; TG, triacylglycerol; FPG, fasting plasma glucose. MetS is defined as the presence of ≥3 of the following components: (i) WC > 88 cm for women and >102 cm for men; (ii) high serum triacylglycerol ≥ 150 mg dL^{-1}; (iii) low HDL cholesterol (50 mg dL^{-1} for women and <40 for men); (iv) fasting blood glucose≥100 mg dL^{-1}; (v) elevated blood pressure (≥130/85 mm Hg). Asterisks indicate statistical differences: *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

3.3. Multivariate-adjusted odds ratios (and 95% CIs) for metabolic syndrome and its components across total, high fat, and low fat yoghurt consumption

According to Table 3 and with application of logistic regression model, the association between yoghurt consumption with metabolic syndrome and its components (after adjustment for confounding variables) can be seen. Yoghurt consumption was significantly inversely associated with the metabolic syndrome after adjustment for energy intake but not for milk and cheese intakes. Among the metabolic syndrome components, there was a significant trend lower triacylglycerol with higher yoghurt consumption.

Also, the association between high-fat and low-fat yoghurt consumption with the metabolic syndrome and its components were assessed separately. However high-fat yoghurt associated with lower risk of high triacylglycerol concentration ($P < 0.05$), low-fat yoghurt consumption was inversely associated with lower risk of abdominal adiposity and high FPG levels ($P < 0.05$).

4. Discussion

In this study, it was found, by comparing the association of low-fat and high-fat yoghurt with the metabolic syndrome and its components, that high-fat yoghurt associated with lower risk of high serum levels of triacylglycerol, while low-fat yoghurt associated inversely with the risk of abdominal adiposity and elevated levels of serum FPG. This is similar to results in a study by Wanga et al. (2013) showing a positive effect of a fermented product on the metabolic syndrome and its profile.

Table 3
Multivariate-adjusted odds ratios (OR) and 95% confidence intervals (CIs) for metabolic syndrome and its components across mean yoghurt consumption.[a]

Parameter	Total yoghurt consumption			High-fat yoghurt consumption			Low-fat yoghurt consumption		
	OR	95% CI for OR	*P*-value[a]	OR	95% CI for OR	*P*-value[a]	OR	95% CI for OR	*P*-value[a]
Metabolic syndrome									
Model 1	0.969	0.939–0.999	0.046	0.962	0.920–1.006	0.089	0.957	0.904–1.013	0.127
Model 2	0.959	0.924–0.996	0.03	0.963	0.915–1.013	0.145	0.927	0.867–0.992	0.027
Model 3	0.963	0.928–1.000	0.052	0.968	0.920–1.018	0.205	0.933	0.872–0.999	0.046
Model 4	0.969	0.932–1.008	0.113	0.976	0.927–1.027	0.347	0.942	0.879–1.01	0.091
Abdominal adiposity									
Model 1	1.012	0.987–1.038	0.35	1.021	0.987–1.055	0.232	1.002	0.948–1.059	0.952
Model 2	1.027	0.991–1.065	0.148	1.062	1.011–1.116	0.018	0.964	0.896–1.038	0.334
Model 3	1.010	0.976–1.045	0.565	1.045	0.999–1.094	0.055	0.930	0.866–0.999	0.046
Model 4	1.002	0.967–1.038	0.931	1.037	0.989–1.088	0.133	0.921	0.856–0.990	0.026
Elevated blood pressure									
Model 1	0.985	0.957–1.014	0.299	0.977	0.938–1.019	0.248	0.985	0.933–1.041	0.598
Model 2	0.984	0.954–1.015	0.318	0.982	0.940–1.025	0.405	0.976	0.921–1.036	0.427
Model 3	0.991	0.964–1.02	0.558	0.987	0.949–1.027	0.531	0.993	0.939–1.050	0.804
Model 4	0.991	0.962–1.022	0.579	0.987	0.947–1.029	0.546	0.994	0.939–1.053	0.839
Low HDL-C									
Model 1	0.998	0.975–1.021	0.845	0.995	0.966–1.025	0.736	1.003	0.954–1.055	0.899
Model 2	0.998	0.975–1.021	0.844	0.997	0.968–1.028	0.864	0.996	0.947–1.048	0.882
Model 3	0.995	0.972–1.019	0.687	0.996	0.966–1.027	0.785	0.989	0.940–1.041	0.671
Model 4	0.993	0.968–1.017	0.553	0.993	0.962–1.025	0.68	0.984	0.934–1.038	0.559
High TAG concentrations									
Model 1	0.950	0.919–0.982	0.002	0.927	0.882–0.975	0.003	0.947	0.896–1.002	0.057
Model 2	0.945	0.912–0.979	0.002	0.927	0.880–0.976	0.004	0.934	0.880–0.992	0.026
Model 3	0.952	0.92–0.984	0.004	0.935	0.89–0.982	0.008	0.946	0.892–1.002	0.06
Model 4	0.956	0.922–0.990	0.012	0.941	0.894–0.99	0.018	0.954	0.899–1.013	0.123
Elevated plasma glucose									
Model 1	0.955	0.911–1.001	0.055	0.963	0.905–1.024	0.228	0.912	0.836–0.995	0.038
Model 2	0.953	0.908–1	0.052	0.965	0.907–1.028	0.270	0.901	0.823–0.987	0.025
Model 3	0.956	0.911–1.004	0.073	0.969	0.911–1.031	0.318	0.907	0.829–0.994	0.036
Model 4	0.964	0.917–1.014	0.155	0.98	0.921–1.043	0.522	0.918	0.837–0.997	0.047

[a] Regression model at the level of significance 0.05 is used. Model 1 is adjusted for age, gender, cigarette smoking, physical activity, and history of diabetes and heart disease; Model 2 is further adjusted for BMI; Model 3 is additionally adjusted for energy intake; Model 4 is additionally adjusted for milk and cheese intake.

Low-fat dairy products have been reported to affect metabolic risk factors in some studies. The general recommendations for saturated fatty acids (SFA) is below 10% of total energy intake, and as low as possible for trans fatty acids, since these fatty acids may unfavourably affect the serum lipoprotein profile and insulin sensitivity (Mensink, Zock, Kester, & Katan, 2003; Riccardi, Giacco, & Rivellese, 2004). In contrast, Van Meijl and Mensink (2011) reported that low-fat dairy products failed to protect against the development of the metabolic syndrome. This result may be due to the fact that glucose and insulin concentrations of its subjects were within the normal range. Another intervention study found similar results in European subjects and showed reducing SFA has no effect on insulin sensitivity in weight-stable obese metabolic syndrome subjects (Tierney et al., 2011).

In this study, the association of yoghurt on metabolic syndrome and its components by differentiation between low-fat and high-fat yoghurt was assessed. In contrast, Wanga et al. (2013) reported that participants who consumed greater amounts of yoghurt had better diet quality that led to greater adequacy for some shortfall nutrients, and did not focus on its effect on metabolic syndrome and its profiles. In the Wanga et al. (2013) study, subjects who consumed higher amounts of yoghurt also consumed higher amounts of fibre, fruit, vegetables, nuts, seeds and whole grains. This was consistent with the previous survey that the dietary variety of nutrient-dense foods was positively associated with nutrient sufficiency (Babio et al., 2015).

In this study high-fat yoghurt was inversely associated with serum triacylglycerol levels. This is in contrast with the results of previous studies and general recommendations; similar studies reported that yoghurt consumption associated with elevated levels of triacylglycerol (Ejtahed et al., 2011; Jacobsen, Lorenzen, Toubro, Krog-Mikkelsen, & Astrup, 2005). On the other hand, in an elderly female population Cho et al. (2011) found a positive relationship between serum calcium and the risk of having high triacylglycerol levels. Another study conducted by Palacios, Bertra, Rios, and Soltero (2011) did not find significant changes in serum lipids except triacylglycerol in a sample of Puerto Rican obese adults. This represents the significant association of yoghurt on serum triacylglycerol levels; however, the mechanism was unknown. The association may be caused by short- and medium-chain fatty acids in yoghurt fat (Marten, Pfeuffer, & Schrezenmeir, 2006). It has been reported that dairy fat reduces the postprandial triglyceride response compared with an oil rich in polyunsaturated fatty acids (Mekki et al., 2002).

As yoghurt and dairy products are the best dietary sources of calcium, and in several studies calcium has been shown to improve lipid profiles, this seems to be a probable reason for the association between yoghurt consumption and low serum TAG level (Abedini, Falahi, & Roosta, 2015; Ma et al., 2011; Samara et al., 2013). Calcium could influence the binding of fatty acids and bile acids in the intestine, thus interfering with the intestinal absorption of fat. Similar results have been reported in support of decreased fat and bile absorption (Ejtahed et al., 2011; Jacobsen et al., 2005).

In the current study, low-fat yoghurt consumption had an inverse association with elevated levels of serum FPG. This is in agreement with some previous evidence (Asemi et al., 2013; Struijk et al., 2013; Wanga et al., 2013). Struijk et al. (2013) found a modest beneficial effect of fermented dairy on glucose regulation measures among middle-aged Danish men and women, but no distinction was made between low-fat and high-fat fermented products. Asemi et al. (2013) found in another controlled trial that daily consumption of yoghurt for 9 weeks in the third trimester

improved insulin sensitivity and helped glucose regulation in Iranian pregnant women. This study examined the effects of probiotic and conventional yoghurt and showed that compared with conventional yoghurt, probiotic yoghurt consumption improved insulin resistance, but had no significant effect on FPG compared with the conventional yoghurt.

Although the precise mechanisms of fermented products on FPG levels are unknown, it could be partly explained by an inverse association between magnesium and insulin resistance, fasting serum insulin, and glucose (Babio et al., 2015). Also, it has been showed that milk, and particularly whey protein, appears insulinotropic, but fermentation may counteract this insulinotropic effect of milk (Östman, Liljeberg Elmståhl, & Björck, 2001).

In this study an inverse association between abdominal adiposity and low fat yoghurt consumption was observed. In agreement with this, some previous studies showed that dairy consumption significantly associated with abdominal obesity, but none of these separated high- and low-fat dairy products (Babio et al., 2015; Shin, Yoon, Lee, Kim, & Oh, 2013).

Some of the relationship remained even after adjustment for confounding variables, such as BMI, total energy, milk and cheese intake. This means that this association may due to the nature of yoghurt consumption, not to other dairy foods or total energy intake. On the other hand, changes in the microbes in gut microbiota (dysbiosis) have been linked to metabolic syndrome and association disorders and the composition of gut microbiota can be modulated by diet (D'Aversa et al., 2013). Therefore, some changes in indices (especially TAG and FPG) may be explained by this phenomenon.

Strengths of the current study include consideration of the potential relationship of both the type of yoghurt on the metabolic profile and the use of logistic regression models and simultaneous adjustment of confounding variables (such as milk and cheese consumption, total energy intake, BMI and physical activity) in the association of yoghurt consumption with metabolic syndrome and its components. However, some limitations should be also noted. The cross-sectional study design mainly be used to test association and does not indicate any causal relationships; therefore, longitudinal studies are needed to determine mechanisms. Although our analyses carefully controlled for yoghurt consumption, other foods such as vegetables and whole grains that have a significant impact on metabolic syndrome were not reviewed. Finally, the FFQs could introduce a selection bias and may affect dietary information collection.

5. Conclusion

In summary, the present cross-sectional study showed that low and high fat yoghurt consumption was inversely association with some components of MetS. Also, high-fat yoghurt associated with the lower risk of high serum levels of triacylglycerol, while low-fat yoghurt associated with the inversely risk of abdominal adiposity and elevated levels of serum FPG. Although the health benefits of yoghurt have been extensively promoted, potential mechanisms are as yet unknown. Future longitudinal studies are needed to obtain evidence for causality and to confirm our results.

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