British Journal of Nutrition (2019), 122, 1113–1119 doi:10.1017/S0007114519002071

Dietary differences between metabolically healthy overweight-obese and metabolically unhealthy overweight-obese adults

Lucas Jurado-Fasoli*, Alejandro De-la-O, Manuel J. Castillo† and Francisco J. Amaro-Gahete*†

Department of Medical Physiology, School of Medicine, University of Granada, Granada E-18016, Spain

(Submitted 10 May 2019 – Final revision received 26 July 2019 – Accepted 8 August 2019 – First published online 23 August 2019)

Abstract

The aim of the study is to determine the differences in dietary parameters (energy and nutrient intake, adherence to the Mediterranean diet and consumption of food groups) in metabolically healthy overweight-obese (MHOO) *v.* metabolically unhealthy overweight-obese (MUOO) middle-aged adults. A total of fifty-one middle-aged adults were classified as MHOO or MUOO. BMI and blood pressure were evaluated following the recommendations. HDL, TAG and blood glycaemia were measured in blood samples. Blood pressure was also assessed. Dietary factors were assessed through three 24-h recalls, a validated FFQ and the PREvención con DIeta MEDiterránea (PREDIMED) questionnaire. All variables were evaluated between September and October 2016 and 2017. Our results showed that MHOO individuals registered a higher fish consumption ($P = 0{\cdot}035$) and higher compliance (lower consumption) in the commercial sweets and confectionery item of the PREDIMED questionnaire ($P = 0{\cdot}036$). No differences were noted in other dietary factors including energy and nutrient intake, consumption of other food groups and in the PREDIMED total score. A near-significant trend toward significance was observed in nuts consumption, wine and fish items of the PREDIMED questionnaire. In conclusion, higher fish consumption and a higher compliance in the commercial sweets and confectionery item of the PREDIMED questionnaire were observed in MHOO middle-aged adults.

Key words: Metabolic health: Obesity: Overweight: Diet: Dietary factors: Mediterranean diet

The prevalence of obesity has increased worldwide, and it has been estimated that by 2030 nearly 40 % of world's population will be overweight and one in five people will be obese[1]. Obesity is considered to be mostly caused by changes in the so-called obesogenic environment such as the highly processed food and the reduction in or replacement of physical activity[2].

Obesity is related to a higher prevalence of the metabolic syndrome[3]. The metabolic syndrome is a set of risk factors for CVD and diabetes[4]. These factors are dysglycaemia, high blood pressure, raised TAG levels, low HDL-cholesterol levels and obesity[4]. However, it has been identified that some overweight/obese individuals showed a specific phenotype characterised by a healthy metabolic profile, without risk factors and with better cardiovascular prognosis[5]. These metabolically healthy but overweight-obese (MHOO) individuals might have a lower risk of CHD, cerebrovascular diseases, metabolic diseases and CVD, in opposite to metabolically unhealthy but overweight-obese (MUOO) individuals[5,6].

The major risk factors that contribute to the development of an unhealthy metabolic profile and the metabolic syndrome are insulin resistance, a high quantity of visceral adipose tissue and a high waist circumference, among others[7]. The main causes of the metabolic syndrome are inadequate levels of physical activity and unhealthy dietary factors[8]. In this sense, it has been demonstrated that a Western diet pattern based on low intake of fruits and vegetables and a high intake of saturated fat could lead to the development of the metabolic syndrome, contributing to the promotion of a unhealthy metabolic profile[8].

The benefits of a healthy diet in the treatment of the metabolic syndrome are largely known[9]. Different dietary patterns such as the Mediterranean diet, the Dietary Approaches to Stop Hypertension diet and plant-based diets have demonstrated to improve the metabolic syndrome and the profile[10–12].

In this sense, diet could influence the development of the healthy or unhealthy phenotype. Previous studies demonstrated that MHOO adolescents have higher adherence to the Mediterranean diet[13], and MUOO adults adhere to pro-inflammatory diet[14] compared with their counterparts. However, Hankinson *et al.* did not find any dietary differences between MHOO and MUOO adults[15]. Evidence of dietary differences between MHOO and MUOO middle-aged adults is mixed. Therefore, the aim of the present study was to determine

Abbreviations: MHOO, metabolically healthy overweight-obese; MUOO, metabolically unhealthy overweight-obese; PREDIMED, PREvención con DIeta MEDiterránea.

* **Corresponding authors**: Lucas Jurado-Fasoli, fax +34 958246179, email juradofasoli@ugr.es; Francisco J. Amaro-Gahete, email amarof@ugr.es

† Both authors contributed equally to this work.

the differences in dietary parameters (energy and nutrient intake, adherence to the Mediterranean diet and consumption of food groups) in MHOO *v.* MUOO middle-aged adults.

Methods

Participants

A total of fifty-one Caucasian middle-aged adults were included in the present study (41·2 % women; 53·2 (SD 5·5) years). A total of 14·8 % participants were current smokers. Participants were selected from the cohort of the FIT-AGEING study (ClinicalTrials.gov ID: NCT03334357)[16]. The study was approved by the Ethics Committee on Human Research at the University of Granada and 'Servicio Andaluz de Salud' (CEI-Granada; 0838-N-2017), and all participants signed an informed consent. The study protocol and experimental design were applied in accordance with the last revised ethical guidelines of the Declaration of Helsinki. Inclusion criteria were to be sedentary (< 20 min of moderate-intensity physical activity on 3 d/week over the last 3 months), and to have a stable weight over the last 6 months. All participants reported to be free of disease, were not pregnant or were not lactating women, were not taking any medication and/or did not have a major illness that would limit the ability to perform the training programme. All data were collected between September and October 2016 and 2017.

Participants were classified as MHOO or MUOO[5]. Individuals were classified as MHOO if they had a BMI ≥ 25 kg/m^2 and did not met any of the following criteria: (i) serum TAG concentration ≥ 1·71 mmol/l, (ii) HDL-cholesterol concentration < 10·4 mmol/l for men and < 13 mmol/l for women, (iii) systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg and (iv) serum fasting glucose level > 5·55 mmol/l. MUOO individuals were characterised as having a BMI ≥ 25 kg/m^2 and at least one of the above-mentioned risk factors.

Blood sample collection

We collected blood samples from the antecubital vein after 12 h of nocturnal fasting. Serum samples were immediately centrifuged and stored in −80°C freezer until their analyses. All participants were requested to abstain from drugs and/or caffeine, to eat an established dinner before sampling and to not do any physical activity of moderate intensity (24 h before) and/or vigorous intensity (48 h before). Glucose, insulin, HDL, total cholesterol and TAG were measured using specific reagents by Beckman Coulter Diagnostics. All the samples were processed in an analyser (Beckman Coulter AU5832). Insulin was analysed by UniCel Dxl 600 (Beckam Coulter). Homoeostasis model assessment index[17] and LDL-cholesterol were calculated[18]. All samples were collected and processed in the Hospital PTS of Granada (Spain).

Blood pressure measurement

The blood pressure was measured with an automatic monitor (Omrom® HEM 705 CP; Health-care Co.) following the recommendations of the European Society of Cardiology (on the right arm, with the participants in a supine position and after 10 min of rest)[19]. It was measured twice and the mean calculated.

Dietary assessment

We included different tools in order to evaluate dietary outcomes by a precise way. To assess dietary factors, we used three 24-h recalls collected on non-consecutive days, a previously validated semi-quantitative 100-item FFQ[20] and the PREvención con DIeta MEDiterránea (PREDIMED) questionnaire[21]. All questionnaires were administered face to face by a qualified and experimented research dietitian.

Energy and macronutrient intake were assessed using the average of the three 24-h recalls conducted on non-consecutive days (one weekend day included), which has previously demonstrated to determine energy intake within 8–10 % of actual energy intake[22]. We used coloured photographs of different portion sizes of food to help to estimate the quantity of food consumed[23]. The interviews were meal-sequence based and involved a detailed assessment and description of the food consumed. Dietary intake from the 24-h recalls was analysed for energy and macronutrient content using the EvalFINUT® software, which is based on US Department of Agriculture and 'Base de Datos Española de Composición de Alimentos' databases.

Dietary energy density of food and beverages (excluding drinking water) was calculated as the total energy intake (kJ/d) divided by the total weight of daily food intake (g/d)[24].

Food serving consumption was assessed using the FFQ. For each FFQ food item, a commonly used portion size was described (slices, cups, teaspoons, etc.), and the participants were asked how often they had consumed that unit on average over the last 3 months. Emphasis was added to ensure that the answers were related to the long-term dietary factors and not to recent dietary changes. Each FFQ food item was converted into servings, considering the standard portion weight of each FFQ food item collected in the own questionnaire (amount consumed/weight of portion).

The adherence to the traditional MedDiet was assessed using the 14-point questionnaire of adherence to the MedDiet used and validated in the PREDIMED trial[21]. The PREDIMED questionnaire includes twelve questions related to frequency intake of key foods and two questions related to specific dietary habits of the MedDiet. Each question scores 0 or 1 point. The global score ranges from 0 to 14, where 0 point = null adherence and 14 points = complete adherence to the MedDiet.

All dietary outcomes were evaluated in a quiet, bright and spacious room in the Instituto Mixto Universitario Deporte y Salud of the University of Granada (Spain).

Anthropometric measurements and body composition assessment

Weight and height were measured using an electronic scale (model 799; Electronic Column Scale) and the BMI calculated (kg/m^2). Lean mass, fat mass and visceral adipose tissue were evaluated by dual-energy X-ray absorptiometry (Discovery Wi; Hologic, Inc.) following the manufacturer's recommendations.

British Journal of Nutrition

Table 1. Descriptive characteristics of participants
(Mean values and standard deviations)

	MHOO (*n* 15)		MUOO (*n* 36)		
	Mean	SD	Mean	SD	*P**
Age (years)	53·4	5·8	53·2	5·5	0·868
Body composition parameters					
Weight (kg)	83·3	15·8	81·7	12·0	0·373
Height (cm)	168·0	169·4	12·5	9·5	0·668
BMI (kg/m^2)	29·3	2·8	28·4	2·7	0·307
Lean mass (kg)	46·4	14·3	46·4	10·9	0·991
Fat mass (kg)	35·7	6·7	32·7	7·9	0·215
Fat mass (%)	43·2	9·4	40·5	9·1	0·361
Visceral adipose tissue mass (g)	1025·0	352·9	947·9	339·1	0·481
Blood parameters					
Total cholesterol (mmol/l)	54·7	10·3	52·0	7·0	0·358
HDL (mmol/l)	16·6	3·2	13·7	2·7	0·002
LDL (mmol/l)	33·0	9·2	32·4	6·1	0·820
TAG (mmol/l)	1·1	0·3	1·7	0·7	< 0·001
Glucose (mmol/l)	4·9	0·3	5·4	0·7	0·002
Insulin (μIU/ml)	7·9	3·5	9·1	6·5	0·490
HOMA index	1·7	0·8	2·3	2·0	0·321
Systolic blood pressure (mmHg)	115·9	11·1	133·8	14·8	< 0·001
Diastolic blood pressure (mmHg)	73·6	7·8	85·4	11·2	0·001

MHOO, metabolically healthy overweight-obese; MUOO, metabolically unhealthy overweight-obese; HOMA, homoeostatic model assessment for insulin resistance.
* *P* value obtained from independent-samples *t* test.

Statistical analysis

The sample size and power calculations are made based on the data of a randomised control trial (The FIT-AGEING project[16]; ClinicalTrials.gov ID: NCT03334357). The principal aim of the FIT-AGEING study was to determine the effect of different training modalities on health-related parameters (i.e. body composition among others) in sedentary healthy adults. The sample size and the power of the study were based on the data of a pilot sample (*n* 30). We considered different health-related parameters (i.e. body composition among others) and differences between pre- and post-treatment in order to assess the sample size requirements for the one-way ANOVA. A sample size of sixty-eight participants was predicted to provide statistical power of 80 %, considering a type I error of 0·05. Therefore, assuming a maximum loss of 25 %, we decided to recruit eighty participants.

Visual check of histograms, Q–Q and box plots were used to verify the distribution of all variables. The descriptive parameters are reported as means and standard deviations.

Independent-samples *t* tests were used to study the differences in body composition and blood parameters between MHOO and MUOO.

One-way ANOVA was used to test the differences between MHOO and MUOO in dietary parameters. The χ^2 test was used to examine the difference between MHOO and MUOO in the compliance of the PREDIMED items.

One-way ANCOVA was used to study the differences between MHOO and MUOO in dietary parameters, adjusting by sex, age and energy intake.

The analyses were conducted using the Statistical Package for Social Sciences (SPSS, version 25.0, IBM SPSS Statistics; IBM Corporation), and the level of significance was set at < 0·05. Graphical presentations were prepared using GraphPad Prism 5 (GraphPad Software).

Results

Table 1 shows the characteristics of the study participants. There were no differences between MHOO and MUOO participants in the body composition variables.

No significant differences were observed in energy intake (Fig. 1(a)), fat intake (Fig. 1(b)), protein intake (Fig. 1(c)), carbohydrate intake (Fig. 1(d)), fibre intake (Fig. 1(e)) and dietary energy density (Fig. 1(f)) between MHOO and MUOO (all $P \geq 0·352$). These results remained after including sex, age and energy intake in the model (Table 2; all $P > 0·313$).

The SFA, MUFA, PUFA and cholesterol fatty acid intake were similar between groups (Fig. 1; all $P \geq 0·119$). These findings persisted when sex, age and energy intake were included in the model (Table 2; all $P \geq 0·107$).

MHOO individuals had a higher fish consumption compared with MUOO individuals (Table 3; 7·6 (SD 2·9) *v.* 5·8 (SD 2·7) servings/week; $P = 0·035$), which remained after including sex ($F = 4·731$, $P = 0·033$, $\eta^2 = 0·091$), age ($F = 4·778$, $P = 0·034$, $\eta^2 = 0·091$) and energy intake ($F = 4·681$, $P = 0·036$, $\eta^2 = 0·092$) as the covariates (Table 4). MUOO individuals had a higher nut consumption compared with MHOO individuals (Table 3; 5·8 (SD 3·9) *v.* 3·3 (SD 3·8) servings/week; $P = 0·047$), which was attenuated after including sex ($F = 3·967$, $P = 0·052$, $\eta^2 = 0·078$), age ($F = 3·984$, $P = 0·052$, $\eta^2 = 0·078$), energy intake ($F = 3·460$, $P = 0·069$, $\eta^2 = 0·071$) as the covariates (Table 4). No differences were observed in other food group consumption between MHOO and MUOO individuals (Table 3; all $P \geq 0·105$).

We showed significant differences in the percentage of positive score in the commercial sweets and confectionery item of the PREDIMED questionnaire between MHOO and MUOO individuals (Table 5; 100 *v.* 72·2 %; $P = 0·036$), which remained after including sex ($P = 0·036$), age ($P = 0·036$) and energy intake ($P = 0·039$) as the covariates (Table 6). No differences were observed in the remaining items of the PREDIMED questionnaire and in the PREDIMED total score between MHOO and MUOO individuals (Table 5; all $P \geq 0·055$).

All previous analyses were adjusted by cardiorespiratory fitness, muscular fitness and physical activity levels of the participants. No differences were found when we included the above-mentioned variables in the models (data not shown).

Discussion

The present study shows that the MHOO individuals registered a higher fish consumption compared to the MUOO individuals, independently of sex, age and total energy intake. Moreover, a higher compliance (lower consumption) in the commercial sweets and confectionery item of the PREDIMED questionnaire was observed in the MHOO individuals compared with the MUOO individuals, independently of sex, age and total energy intake. However, no differences were noted in other dietary factors including energy and nutrient intake, other food group

Table 2. Differences between metabolically healthy and unhealthy overweight-obese adults in energy and nutrient intake*

	Model 1			Model 2			Model 3		
	F	*P*	η^2	*F*	*P*	η^2	*F*	*P*	η^2
Energy (kJ/d)	0·187	0·667	0·004	0·217	0·643	0·005	–	–	–
Fat (g/d)	0·008	0·929	0·000	0·026	0·874	0·001	0·097	0·756	0·002
Protein (g/d)	0·018	0·894	0·000	0·023	0·881	0·000	0·158	0·693	0·003
Carbohydrates (g/d)	0·255	0·616	0·006	0·284	0·597	0·006	0·072	0·790	0·002
Fibre (g/d)	1·041	0·313	0·024	0·785	0·381	0·018	0·873	0·356	0·021
Dietary energy density (kJ/g)	0·078	0·781	0·002	0·092	0·762	0·002	0·042	0·839	0·001
SFA (g/d)	0·090	0·765	0·002	0·122	0·729	0·003	0·012	0·912	0·000
MUFA (g/d)	0·520	0·475	0·011	0·347	0·559	0·007	0·810	0·373	0·017
PUFA (g/d)	0·805	0·374	0·017	0·584	0·449	0·013	1·882	0·177	0·039
Cholesterol (g/d)	2·560	0·116	0.053	2·474	0·123	0·051	2·702	0·107	0·055

* Values obtained from ANCOVA adjusting by sex (model 1); age (model 2); energy intake (model 3).

Table 3. Participants' intake and consumption of different food groups (Mean values and standard deviations)

	MHOO (*n* 15)		MUOO (*n* 36)		
	Mean	SD	Mean	SD	*P**
Vegetables (servings/d)	3·5	1·5	3·0	1·5	0·355
Fruits (servings/d)	3·3	2·9	2·7	1·4	0·303
Dairy products (servings/d)	1·7	0·9	2·0	1·0	0·445
Olive oil (servings/d)	2·9	0·9	2·6	0·5	0·102
Cereals (servings/d)	2·8	1·0	3·1	0·8	0·235
Eggs (servings/week)	3·2	1·6	3·0	0·9	0·686
White meat (servings/week)	2·2	1·5	2·2	1·4	0·943
Red meat (servings/week)	2·9	1·5	2·2	1·5	0·168
Processed meat (servings/week)	5·4	2·8	4·6	2·4	0·293
Fish (servings/week)	7·6	2·9	5·8	2·7	0·035
Legumes (servings/week)	1·3	1·1	1·8	1·2	0·181
Nuts (servings/week)	3·3	3·8	5·8	3·9	0·047
Vegetable oils (servings/week)	0·3	0·4	0·3	0·7	0·836
Margarine (servings/week)	0·8	1·8	1·0	2·0	0·738
Butter (servings/week)	0·8	1·9	0·7	1·7	0·871
Sweets (servings/week)	5·6	5·5	6·3	6·2	0·698
Alcoholic beverages (servings/week)	7·3	6·2	8·0	7·3	0·752
Soft drinks (servings/week)	3·2	5·2	1·9	2·8	0·262

MHOO, metabolically healthy overweight-obese; MUOO, metabolically unhealthy overweight-obese.
* *P* values from one-way ANOVA.

consumption and in the PREDIMED total score. A near-significant trend toward significance was observed in nut consumption, wine and fish items of the PREDIMED questionnaire.

It is well known that consumption of fish and other sea-derived products are related to lower risk of the metabolic syndrome and other cardiometabolic diseases[25]. A previous meta-analysis found that the incidence of the metabolic syndrome is reduced by 6 % with an increment of one serving/week of fish consumption[25]. In this sense, previous studies have suggested that high fish consumption was associated with lower TAG levels, higher HDL-cholesterol levels and lower risk of the metabolic syndrome in Norwegian middle-aged adults[26] and in Norwegian adults of 26–70 years[27]. High fish consumption was also associated with lower waist circumference, lower TAG levels, higher HDL-cholesterol levels and lower risk of the metabolic syndrome in a 13-year follow-up study from the above-mentioned cohorts[28]. This relationship could be explained by different potential mechanisms based on the fish nutrient contents. Fish are rich in *n*-3 long-chain PUFA (EPA and DHA), proteins, taurine, vitamin D, vitamin B, iodine and Se[26,28]. These nutrients have anti-inflammatory properties[28], reduce abdominal obesity[28], improve blood lipid profile[28], have hypotensive effects[28] and induce fatty acid oxidation[26], these effects are independent of the type of fish (lean or fatty) consumed[26–28]. Therefore, the intake of the above-mentioned nutrients found in fish and sea-derived products could develop a better metabolic profile independent of the BMI status[29].

MHOO individuals have a higher compliance in the commercial sweets and confectionery item of the PREDIMED questionnaire (≤ 2 servings/week). A previous study conducted in adults of 19–70 years demonstrated that a higher consumption of energy-dense snacks (i.e. biscuits, cakes, candies and chocolates) could be a dietary risk factor for the development of the metabolic syndrome[30]. Commercial sweets and confectionery are rich in added sugars, and their consumption is associated with dyslipidaemia, insulin resistance, CVD and type 2 diabetes and increases the risk of metabolic diseases[31], independent of the weight or the total energy intake[31]. Commercial sweets and confectionery are the main sources of *trans*-fatty acids[32]. *Trans*-fatty acid intake influences blood lipids and lipoprotein levels[32], increases systemic inflammation[32], dysregulates endothelial function[32], increases adiposity[32] and disrupts glucose–insulin homoeostasis[32], increasing, therefore, the prevalence of the metabolic syndrome[32,33]. In addition, commercial sweets and confectionery have a high content of refined flours, which are associated with insulin resistance and the metabolic syndrome in adults[34].

In terms of the remaining dietary factors, our results are not in accord with previous studies that observed that (i) a high adherence to the Mediterranean diet pattern protected against the metabolic syndrome[35], mainly due to the higher consumption of olive oil, fruits, vegetables, legumes and nuts[35]; (ii) a high fruit intake was negatively associated with the prevalence of the metabolic syndrome[36], due to their high content in soluble fibre that reduces insulin secretion and regulates blood lipids[36]; (iii) a low saturated fat intake was negatively associated with the prevalence of the metabolic syndrome[36], due to the fact that saturated fat increases visceral adipose tissue and dysglycaemia and

https://doi.org/10.1017/S0007114519002071 Published online by Cambridge University Press

Table 4. Differences between metabolically healthy and unhealthy overweight-obese adults in food group consumption*

	Model 1			Model 2			Model 3		
	F	*P*	η^2	*F*	*P*	η^2	*F*	*P*	η^2
Vegetables (servings/d)	0·695	0·409	0·015	0·833	0·366	0·017	0·838	0·365	0·018
Fruits (servings/d)	0·857	0·359	0·018	1·048	0·311	0·021	1·197	0·280	0·025
Dairy products (servings/d)	0·452	0·505	0·009	0·573	0·453	0·012	0·491	0·487	0·011
Olive oil (servings/d)	2·714	0·106	0·054	2·685	0·108	0·053	2·729	0·105	0·056
Eggs (servings week)	0·134	0·716	0·003	0·166	0·685	0·003	0·182	0·672	0·004
White meat (servings/week)	0·021	0·886	0·000	0·006	0·939	0·000	0·246	0·622	0·005
Red meat (servings/week)	2·568	0·116	0·051	2·014	0·162	0·040	2·306	0·136	0·048
Processed meat (servings/week)	1·332	0·254	0·028	1·084	0·303	0·023	1·604	0·212	0·034
Fish (servings/week)	4·799	0·033	0·091	4·778	0·034	0·091	4·681	0·036	0·092
Legumes (servings/week)	2·013	0·162	0·040	1·808	0·185	0·036	2·441	0·125	0·050
Nuts (servings/week)	3·967	0·052	0·078	3·984	0·052	0·078	3·460	0·069	0·071
Vegetable oils (servings/week)	0·030	0·864	0·001	0·044	0·835	0·001	0·012	0·914	0·000
Margarine (servings/week)	0·223	0·639	0·005	0·096	0·758	0·002	0·089	0·767	0·002
Butter (servings/week)	0·045	0·833	0·001	0·035	0·853	0·001	0·110	0·742	0·002
Sweets (servings/week)	0·113	0·739	0·002	0·169	0·683	0·004	0·027	0·870	0·001
Alcoholic beverages (servings/week)	0·019	0·892	0·000	0·122	0·729	0·003	0·003	0·955	0·000
Soft drinks (servings/week)	1·312	0·258	0·027	1·234	0·272	0·025	1·842	0·181	0·038

* Values obtained from ANCOVA adjusting by sex (model 1); age (model 2); energy intake (model 3).

Table 5. Participants' response frequency of food items included in the PREvención con DIeta MEDiterránea (PREDIMED) questionnaire* (Percentage positive scores; mean values and standard deviations)

Questions in the PREDIMED questionnaire	MHOO (*n* 15) (% positive score)	MUOO (*n* 36) (% positive score)	*P*†
(1) Use of extra virgin olive oil as main culinary lipid	100	100	–
(2) Extra virgin olive oil > 4 tablespoons	86·7	75·0	0·199
(3) Vegetables ≥ 2 servings/d	66·7	55·6	0·354
(4) Fruits ≥ 3 servings/d	26·7	30·6	0·845
(5) Red/processed meats < 1/d	73·3	83·3	0·924
(6) Butter, cream, margarine < 1/d	100	100	–
(7) Soda drinks < 1/d	73·3	80·6	0·726
(8) Wine glasses ≥ 7/week	33·3	13·9	0·093
(9) Legumes ≥ 3/week	26·7	27·8	1·000
(10) Fish/seafood ≥ 3/week	33·3	13·9	0·093
(11) Commercial sweets and confectionery ≤ 2/week	100	72·2	0·036
(12) Tree nuts ≥ 3/week	46·7	55·6	0·650
(13) Poultry more than red meats	80·0	86·1	0·783
(14) Use of sofrito sauce ≥ 2/week	60·0	80·6	0·159
PREDIMED total score			0·334
Mean	9·5	8·9	
SD	1·5	1·9	

MHOO, metabolically healthy overweight-obese; MUOO, metabolically unhealthy overweight-obese.
* Data are presented as percentage of positive score of the question.
† *P* values from χ^2 analysis and one-way ANOVA.

Table 6. Differences between metabolically healthy and unhealthy overweight-obese adults in PREvención con DIeta MEDiterránea (PREDIMED) items and total score*

	Model 1	Model 2	Model 3
	P	*P*	*P*
(1) Use of extra virgin olive oil as main culinary lipid	–	–	–
(2) Extra virgin olive oil > 4 tablespoons	0·199	0·199	0·222
(3) Vegetables ≥ 2 servings/d	0·354	0·354	0·419
(4) Fruits ≥ 3 servings/d	0·845	0·845	0·736
(5) Red/processed meats < 1/d	0·924	0·924	0·843
(6) Butter, cream, margarine < 1/d	–	–	–
(7) Soda drinks < 1/d	0·726	0·726	0·647
(8) Wine glasses ≥ 7/week	0·093	0·093	0·195
(8) Wine glasses ≥ 7/week	0·093	0·093	0·195
(9) Legumes ≥ 3/week	1·000	1·000	0·736
(10) Fish/seafood ≥ 3/week	0·093	0·093	0·195
(11) Commercial sweets and confectionery ≤ 2/week	0·036	0·036	0·039
(12) Tree nuts ≥ 3/week	0·650	0·650	0·576
(13) Poultry more than red meats	0·783	0·783	0·720
(14) Use of sofrito sauce ≥ 2/week	0·159	0·159	0·304
PREDIMED total score	0·348	0·331	0·373

* Values obtained from χ^2 analysis adjusting by sex (model 1); age (model 2); and energy intake (model 3).

(iv) energy-restricted diets and moderate-high-protein diets protected against the prevalence of the metabolic syndrome[37], due to the decrement of abdominal obesity, the regulation of blood lipids and glucose levels[37]. The lack of accordance with previous studies in the remaining dietary factors might be due to the small sample size that may fail to detect meaningful differences between groups or the different nationalities of the previous study[36].

The main weakness of the present study is the cross-sectional design, which does not allow to identify any causal association. Additionally, the study sample was constituted by sedentary middle-aged adults (45–65 years), thus we cannot extrapolate our results to younger and/or physically active individuals. And lastly, we did not differ between lean and fatty fish and this differentiation could be a determinant of the association found.

In conclusion, our study showed that the MHOO individuals registered a higher fish consumption compared with the MUOO individuals, independent of sex, age and total energy intake. Moreover, a higher compliance in the

British Journal of Nutrition

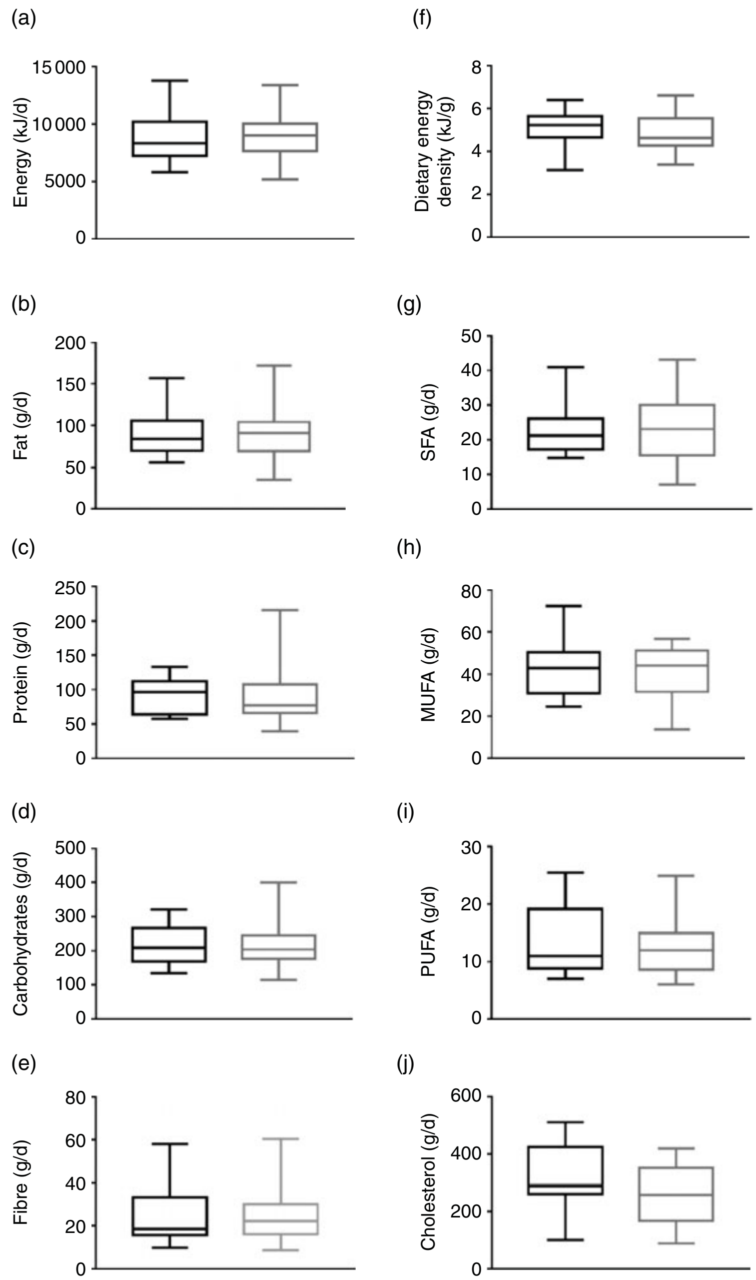

Fig. 1. Dietary intake in metabolically healthy overweight-obese (black plots) and metabolically unhealthy overweight-obese (grey plots) individuals. Data are means and standard deviations. *P* values are from one-way ANOVA. (a) $P = 0{\cdot}640$, (b) $P = 0{\cdot}874$, (c) $P = 0{\cdot}879$, (d) $P = 0{\cdot}594$, (e) $P = 0{\cdot}352$, (f) $P = 0{\cdot}763$, (g) $P = 0{\cdot}728$, (h) $P = 0{\cdot}562$, (i) $P = 0{\cdot}445$, and (j) $P = 0{\cdot}119$.

commercial sweets and confectionery item of the PREDIMED questionnaire was observed in the MHOO individuals compared with the MUOO individuals, independent of sex, age and total energy intake. Longitudinal studies with fish-type differentiation are required to establish causal association.

Acknowledgements

The authors would like to thank all the participants who participated in the study for their time and effort. The present study is part of a PhD thesis conducted in the Biomedicine Doctoral Studies of the University of Granada, Spain. The study is supported by the Spanish Ministry of Education (FPU14/04172 and FPU15/03960). We are grateful to Dr Ángel Gutiérrez for his support with all of the study.

No financial support was received for this study.

L. J. F., A. D. O., M. J. C. and F. J. A. G. conceived and designed the study; L. J. F., A. D. O., M. J. C. and F. J. A. G. acquired data; L. J. F. and F. J. A. G. elaborated the statistical section; L. J. F. and F. J. A. G. drafted the manuscript, and M. J. C. revised the manuscript; all authors read and approved the final manuscript.

The authors report no conflicts of interest.

British Journal of Nutrition

References

1. Kelly T, Yang W, Chen CS, *et al.* (2008) Global burden of obesity in 2005 and projections to 2030. *Int J Obes* **32**, 1431–1437.
2. Meldrum DR, Morris MA & Gambone JC (2017) Obesity pandemic: causes, consequences, and solutions—but do we have the will? *Fertil Steril* **107**, 833–839.
3. Grundy SM (2016) Metabolic syndrome update Scott. *Trends Cardiovasc Med* **26**, 364–373.
4. Alberti KGMM, Eckel RH, Grundy SM, *et al.* (2009) Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation* **120**, 1640–1645.
5. Ortega FB, Lavie CJ & Blair SN (2016) Obesity and cardiovascular disease. *Circ Res* **118**, 1752–1770.
6. Elagizi A, Kachur S, Lavie CJ, *et al.* (2018) An overview and update on obesity and the obesity paradox in cardiovascular diseases. *Prog Cardiovasc Dis* **61**, 142–150.
7. Sherling DH, Perumareddi P & Hennekens CH (2017) Metabolic syndrome: clinical and policy implications of the new silent killer. *J Cardiovasc Pharmacol Ther* **22**, 365–367.
8. Pereira MA, Kottke TE, Jordan C, *et al.* (2009) Preventing and managing cardiometabolic risk: the logic for intervention. *Int J Environ Res Public Health* **6**, 2568–2584.
9. Mozaffarian D (2017) Foods, obesity, and diabetes – are all calories created equal? *Nutr Rev* **75**, 19–31.
10. Tortosa A, Tortosa A, Bes-Rastrollo M, *et al.* (2007) Mediterranean diet inversely associated with the incidence of metabolic syndrome. *Am J Clin Nutr* **30**, 11–13.
11. Lien LF, Brown AJ, Ard JD, *et al.* (2007) Effects of PREMIER lifestyle modifications on participants with and without the metabolic syndrome. *Hypertension* **50**, 609–616.
12. Kahleova H, Levin S & Barnard N (2017) Cardio-metabolic benefits of plant-based diets. *Nutrients* **9**, E848.
13. Arenaza L, Huybrechts I, Ortega FB, *et al.* (2019) Adherence to the Mediterranean diet in metabolically healthy and unhealthy overweight and obese European adolescents: the HELENA study. *Eur J Nutr* **58**, 2615–2623.
14. Park YMM, Choi MK, Lee SS, *et al.* (2019) Dietary inflammatory potential and risk of mortality in metabolically healthy and unhealthy phenotypes among overweight and obese adults. *Clin Nutr* **38**, 682–688.
15. Hankinson AL, Daviglus ML, Van Horn L, *et al.* (2013) Diet composition and activity level of at risk and metabolically healthy obese American adults. *Obesity* **21**, 637–643.
16. Amaro-Gahete FJ, De-la-O A, Jurado-Fasoli L, *et al.* (2018) Exercise training as S-Klotho protein stimulator in sedentary healthy adults: rationale, design, and methodology. *Contemp Clin Trials Commun* **11**, 10–19.
17. Matthews DR, Hosker JP, Rudenski AS, *et al.* (1985) Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* **28**, 412–419.
18. Friedewald WT, Levy RI & Fredrickson DS (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* **18**, 499–502.
19. Williams B, Mancia G, Spiering W, *et al.* (2018) 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J* **39**, 3021–3104.
20. Vioque J, Navarrete-Muñoz E-M, Gimenez-Monzó D, *et al.* (2013) Reproducibility and validity of a food frequency questionnaire among pregnant women in a Mediterranean area. *Nutr J* **12**, 26.
21. Schroder H, Fito M, Estruch R, *et al.* (2011) A short screener is valid for assessing Mediterranean diet adherence among older Spanish men and women. *J Nutr* **141**, 1140–1145.
22. Baugh E, Hedrick VE, Marinik EL, *et al.* (2015) Dietary intake modification in response to participation in a resistance training program for sedentary older adults with prediabetes: findings from the Resist Diabetes Study. *Eat Behav* **15**, 379–382.
23. López MDR & Martín-Lagos RA (2010) *Guía para estudios dietéticos: álbum fotográfico de alimentos (Guide to Dietary Studies: Photographic Food Album)*. Granada: Editorial Universidad de Granada.
24. Ledikwe JH, Blanck HM, Khan LK, *et al.* (2005) Dietary energy density determined by eight calculation methods in a nationally representative United States population. *J Nutr* **135**, 273–278.
25. Kim YS, Xun P & He K (2015) Fish consumption, long-chain omega-3 polyunsaturated fatty acid intake and risk of metabolic syndrome: a meta-analysis. *Nutrients* **7**, 2085–2100.
26. Karlsson T, Rosendahl-Riise H, Dierkes J, *et al.* (2017) Associations between fish intake and the metabolic syndrome and its components among middle-aged men and women: the Hordaland Health Study. *Food Nutr Res* **61**, 1347479.
27. Tørris C, Molin M & Cvancarova Småstuen M (2016) Associations between fish consumption and metabolic syndrome. A large cross-sectional study from the Norwegian Tromsø Study: Tromsø 4. *Diabetol Metab Syndr* **8**, 18.
28. Tørris C, Molin M & Cvancarova Småstuen M (2017) Lean fish consumption is associated with beneficial changes in the metabolic syndrome components: a 13-year follow-up study from the Norwegian Tromsø Study. *Nutrients* **9**, 247.
29. Castilló Garzón MJ (2008) Changing the Spanish-Mediterranean diet. In *Wild-Type Food in Health Promotion and Disease Prevention: The Columbus Concept*, pp. 479–491 [F De Meester and RR Watson, editors]. Totowa, NJ: Humana Press.
30. Mirmiran P, Bahadoran Z, Delshad H, *et al.* (2014) Effects of energy-dense nutrient-poor snacks on the incidence of metabolic syndrome: a prospective approach in Tehran Lipid and Glucose Study. *Nutrition* **30**, 538–543.
31. Stanhope KL (2016) Sugar consumption, metabolic disease and obesity: the state of the controversy. *Crit Rev Clin Lab Sci* **53**, 52–67.
32. Micha R & Mozaffarian D (2009) Trans fatty acids: effects on metabolic syndrome, heart disease and diabetes. *Nat Rev Endocrinol* **5**, 335–344.
33. Cascio G, Schiera G & Di Liegro I (2012) Dietary fatty acids in metabolic syndrome, diabetes and cardiovascular diseases. *Curr Diabetes Rev* **8**, 2–17.
34. Radhika G, Van Dam RM, Sudha V, *et al.* (2009) Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57). *Metabolism* **58**, 675–681.
35. Di Daniele N, Noce A, Vidiri MF, *et al.* (2017) Impact of Mediterranean diet on metabolic syndrome, cancer and longevity. *Oncotarget* **8**, 8947–8979.
36. De Oliveira EP, McLellan KCP, Vaz De Arruda Silveira L, *et al.* (2012) Dietary factors associated with metabolic syndrome in Brazilian adults. *Nutr J* **11**, 13.
37. de la Iglesia R, Loria-Kohen V, Zulet MA, *et al.* (2016) Dietary strategies implicated in the prevention and treatment of metabolic syndrome. *Int J Mol Sci* **17**, E1877.