FAT AND CARBOHYDRATES IN THE DIET: ITS METABOLIC CONTRIBUTION TO OBESITY IN CHILEAN WOMEN

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Abstract

The effects of changes in dietary fatty acid composition on macronutrient oxidation were studied on 8 controls and 6 obese women, 30-45 y old. Anthropometry, body composition, physical activity, continuous indirect calorimetry (200 min), plasma fatty acids, serum insulin and glucose were measured. The study was performed using a crossover design: baseline and three-period 2wk each: canola oil supplementation, washout and sunflower oil supplementation. Subjects were provided with commercially available oil (1 L/wk) either canola or sunflower during the corresponding period.

Oil treatment produced significant modifications of plasma fatty acid profile, according to the type of oil consumed. In both groups, most of the fat provided was oxidised (ratio oxidised/intake: 79-102%) in the two dietary regimes, no differences were found between groups. Controls however, had a higher fat oxidation (mg/kg fat-free mass) with sunflower compared to canola treatment. Changes in plasma polyunsaturated/saturated ratio (P/S) from canola to sunflower treatment were not associated to fat oxidation. Changes in plasma n6/n3 ratio from canola to sunflower treatment showed a positive association with fat oxidation in controls (r=0.72) and a negative association in obese (r=-0.79). Carbohydrate oxidation was on average 20-29% of CHO intake. Glucose oxidation was not associated to n6/n3 ratio from canola to sunflower treatment, but it was inversely correlated to P/S ratio changes in both groups. With sunflower treatment obese showed a higher CHO oxidation (mg/kg fat-free mass) associated to a greater insulin response compared to controls.

This study showed that the type of oil can induce differences in substrate oxidation. Canola oil intake could be stimulated based on its smaller insulin response in subjects predisposed to hyperinsulinemia and insulin resistance.

1. INTRODUCTION

Chile has been through the epidemiology transition and its population's lifestyle is now quite comparable to developed countries. Within the many similarities, diet and physical activity patterns are found almost invariably associated to obesity among less privileged groups. In Chile, obesity is highly prevalent, reaching >40% in middle age women in the lower socio-economic population group. Dietary energy is supplied by carbohydrates (50-60%) from foods such as pasta, bread and rice. The amount of fat on average is <30% of the energy intake (25-27% from dietary studies, 30% from food balance sheets).

Dietary components must be seen not only from the energy balance perspective but also from the composition of the dietary macronutrients, such as fat. It is known that their metabolism varies according to chain length, number of double bonds and isomerism. The available information suggests that the type of fatty acids in the diet can influence the metabolic route of macronutrients. This differential effect of fatty acids is based on its proper oxidation rate ([1,2]), enzymatic modulation ([3,4,5]) and gene expression ([6,7]). In humans ([2]), the oxidation rate of several individual fatty acids varying in chain length, unsaturation and isomerism have been evaluated. After 9h of α-linolenic acid (ALA) and linoleic acid (LA) intake, it was found an oxidation rate of 23.6 ± 6.6% and 16.1 ± 6.6%, respectively. From this study and others, it has been concluded that oxidation rate is: (a) inversely related to chain length, (b) directly proportional to the number of double bonds, (c) higher for cis compared to trans fatty acids and (d) higher for n3 compared to n6 fatty acids. On the other hand, studies in rats have described the effect of different oils enriched with several types of fatty acids (LA, ALA,

saturated or marine oils) on the enzyme activities related to mitochondrial fat oxidation or fat synthesis. The enzymes related to fat oxidation had higher activity when animals were fed with the more oxidative fatty acids, whereas enzymes related to fat synthesis were decreased. The opposite pattern was observed in less oxidative fatty acids ([3,4]). Similar results were observed for gene expression of the same enzymes ([6,7]). In this regard, it can be suggested that diets enriched with a particular type of fatty acid could increase or decrease the degree of oxidation and/or storage of fat, affecting the rate of fat accretion and/or weight gain. This has been found in rats with diets rich in ALA or fish oil ([7]), and in humans with the fish oil ([8]).

2. HYPOTHESES

The ratio of polyunsaturated n6/n3 fatty acids in the diet is inversely associated to total post-prandial fat oxidation.

3. MATERIALS AND METHODS

3.1. Sample

Fourteen healthy, middle age (30-50 years old) women, were divided in two nutritional categories: normal weight (n=8) defined as BMI 20-25 kg/m2 and obese (n=6) 30-40 kg/m^2. Exclusion criteria was the presence of chronic diseases such as diabetes, hypertension, hyperlipemia and thyroid disorders. Persons with impaired oral glucose tolerance test (OGTT) were also excluded. All individuals had stable weight at least 3 months before and during the test, with no medication and/or hormonal treatment. Menstrual cycle was regular in all cases. The experimental protocol was approved by the Ethical Committee of the Institute of Nutrition and Food Technology (INTA). Informed consent was given to all subjects at beginning of the study. Baseline characteristics of subjects are showed in Table 1, 2 and 3.

TABLE 1: CHARACTERISTIC OF THE SUBJECTS

	Control	Obese
Number of cases	8	6
Age (y)	37.2 ± 6.8	38.0 ± 5.0
Weight (kg)	56.4 ± 5.4	78.8 ± 11.4
Height (m)	1.6 ± 0.0	1.6 ± 0.0
BMI (kg/m^2)	23.0 ± 1.5	32.5 ± 3.7
Body Fat (%)*	27.5 ± 5.1	46.1 ± 6.8
Fat free mass (kg)*	40.8 ± 3.7	42.7 ± 9.2

*By Hydrodensitometry

TABLE 2: GLYCEMIC AND INSULINEMIC LEVELS DURING OGTT

	Control	Obese
Fasting glucose (mg/dL)	88.2 ± 9.0	93.7 ± 14.7
Fasting insulin (μU/mL)	7.7 ± 2.3	10.3 ± 5.7
Post OGTT glucose (mg/dL)	102.8 ± 18.7	100.5 ± 18.7
Post OGTT insulin (μU/mL)	34.7 ± 13.1	45.9 ± 11.8

TABLE 3: PLASMA LIPID PROFILE

	Control	Obese
Total cholesterol (mg/dL)	160.9 ± 28.3	143.4 ± 21.3
HDL (mg/dL)	40.7 ± 11.4	43.2 ± 12.4
LDL (mg/dL)	100.4 ± 33.1	81.8 ± 26.6
Triacylglycerols (mg/dL)	99.2 ± 41.8	92.4 ± 40.1

3.2. Baseline measurements

Under fasting conditions the following measurements were performed: OGTT using 75 g anhydrous glucose at times 0 and 2 h, plasma lipids (total cholesterol, HDL-cholesterol, LDL-cholesterol, triacylglycerols), plasma fatty acids profile, resting energy expenditure and hydrodensitometry.

3.3. Protocol

The study was performed using a crossover design. After baseline measurements, 2 weeks of dietary treatment were followed by 2 weeks washout period and finally the second dietary treatment. Considering that the most frequent oil consumed in this population group is a blend of soybean and canola oil, it was decided to start with canola oil, this way the plasma fatty acids profile were supposedly maintained. Subjects were asked to replace the oil consumed by the one provided for the study. Each person received 1 liter of oil per week to be consumed by the whole family. In addition, subjects were asked to add a total 10 mL/day to their meals to ensure a minimum of oil intake. At the end of this period, subjects came to INTA under fasting conditions to measure their resting energy expenditure for 200 min following an experimental breakfast. A blood sample was drawn at -15, -10 and -5 min before breakfast to measure serum glucose and insulin. A fasting blood sample was used to measure plasma fatty acids profile. Blood samples to measure serum glucose and insulin were taken at 30, 45, 60, 90 and 120 min, counted from beginning of breakfast. The same procedure was followed for the second dietary treatment.

3.4. Characteristic of the experimental breakfast and edible oil

Breakfast supplied 40% of total energy expenditure, obtained from fasting energy expenditure measured on a previous day multiplied by a factor 1.3 to account for physical activity. Energy intake distribution was 55%, 38% and 5% for carbohydrates, fat and protein, respectively. Breakfast had a constant polyunsaturated/saturated (P/S) ratio, varying only in the n6/n3 ratio, which was manipulated to obtain the maximum difference between diets based on the amount

of butter, margarine and oil type. The other foods used were bread, marmalade, tea or decaffeinated coffee, sugar and aspartame. Canola and sunflower oils were chosen as ALA and LA rich sources, respectively. Energy and macronutrient content were measured by proximal analysis and fatty acids profile by gas chromatography. Tables 4 to 6 show characteristics of the breakfast and edible oils used.

TABLE 4: CHARACTERISTICS OF THE TEST MEALS

	Canola breakfast		Sunflower breakfast	
	Control	**Obese**	**Control**	**Obese**
Energy (kcal)	360.1 ± 35.5	409.2 ± 53.5	359.9 ± 35.5	409.3 ± 53.5
Protein (g)	4.5 ± 0.4	5.1 ± 0.7	4.6 ± 0.5	5.3 ± 0.7
Fat (g)	15.4 ± 1.5	17.5 ± 2.3	15.0 ± 1.5	17.0 ± 2.2
Carbohydrate (g)	50.0 ± 4.9	56.8 ± 7.4	51.1 ± 5.1	58.1 ± 7.6

TABLE 5: FATTY ACID (FA) PROFILE OF THE TEST MEALS

	Canola breakfast		Sunflower breakfast	
	Control	**Obese**	**Control**	**Obese**
Saturated FA (g)	3.4 ± 0.3	3.9 ± 0.5	5.0 ± 0.5	5.6 ± 0.7
Monounsaturated FA (g)	4.9 ± 0.5	5.6 ± 0.7	3.2 ± 0.3	3.7 ± 0.5
Polyunsaturated FA (g)	4.9 ± 0.5	5.6 ± 0.7	7.1 ± 0.7	8.1 ± 1.1
PUFA/SFA ratio	1.4 ± 0.0	1.4 ± 0.0	1.4 ± 0.0	1.4 ± 0.0
18:2n6 (g)	4.0 ± 0.4	4.6 ± 0.6	6.9 ± 0.7	7.8 ± 1.0
18:3n3 (g)	0 .69 ± 0.1	0.79 ± 0.1	0.02 ± 0.0	0.02 ± 0.0
n6/n3 ratio	5.8 ± 0.0	5.8 ± 0.0	385.6 ± 0.2	385.6 ± 0.2

TABLE 6: FATTY ACID PROFILE OF THE EDIBLE OILS

	Canola oil	Sunflower oil
Saturated FA (%)	13.4	12.0
Monounsaturated FA (%)	38.8	23.9
Polyunsaturated FA (%)	47.8	64.1
18:2n6 (g)	40.9	63.9
18:3n3 (g)	6.93	0.16
n6/n3 ratio	5.9	399.3

3.5. Measurements

3.5.1. Anthropometry

Weight was measured every 2 weeks with a minimum clothing on a DETECTO® platform scale, 100 g precision and 160 kg capacity. Height was measured with stadiometer attached to the scale, 1 mm precision.

3.5.2. Body composition

Body fat was measured by hydrodensitometry based on Archimedes's principle with a simultaneous determination of residual lung volume using helium dilution. Underwater weighing was obtained with a CONDOR® digital scale, 10 g precision and 10 kg capacity.

3.5.3. Plasma analysis

Blood samples were obtained from an antecubital vein. Samples for glucose, insulin and lipid profile were drawn without anticoagulant, centrifuged at 2500 rpm for 10 min. Serum samples were immediately stored at –20°C. Samples for plasma fatty acids profile were drawn in Vacutainer-EDTA®, immediately centrifuged at 2500 rpm, 10 min and 4°C. The lipid extract was obtained by the Folch method ([9]) and stored at –18°C for later analysis. Glucose was analyzed by glucose oxidase method (Kit Roche Diagnostic, Mannheim), Insulin by RIA (DPC, Los Angeles, CA, USA). Serum cholesterol and triacylglycerols were assessed by colorimetric techniques (Kit Roche Diagnostic, Mannheim). HDL-cholesterol was measured by precipitation (Kit Roche Diagnostic, Mannheim), LDL-cholesterol by Friedewald formula. Plasma fatty acids profile was determined by gas chromatography on a Carlo Erba GC 6000 serie 2, with a flame ionisation detector and 30 m capillary silica Supelco®. This column identifies cis-fatty acids from C10:0 to C24:1 including long chain PUFA.

3.5.4. Serum glucose, insulin and insulin resistance

Serum and insulin glucose were expressed as area under the curve (AUC) according to standard procedure ([10]). Insulin resistance was determined by Homeostatic Model Assessment (HOMA) through the formula [glycemia * insulinemia]/22.5 ([11]).

3.5.5. Resting energy expenditure and substrate oxidation

Measurements were performed after and overnight fast and after the intake of test meals. Fasting, post-prandial resting energy expenditure and substrate oxidation were measured with an indirect calorimeter Sensor Medics 2900® with a canopy system. Data was analysed discarding the first 10 min of the test. Data was collected during 30 and 200 min for fasting and post-prandial conditions, respectively. Total CHO and fat oxidation was obtained from the formula proposed by Livesey et al. ([12]). Urinary nitrogen was obtained from urine produced at the middle and the end of the test. Nitrogen was determined by Kjedahl method. Non protein respiratory quotient (NPRQ) was calculated after subtracting the amount of O_2 and CO_2 related to protein oxidation.

3.5.6. Physical activity

Physical activity was assessed by heart rate monitoring (HRM) using a Polar Vantage® continuously for 24 h in one occasion during the study. Data was downloaded through a computer interface. Time and duration of major activities performed throughout the day were obtained by a recall questionnaire. Sleeping time was determined from the night time HRM. The average HRM during sleeping was used as the unit to compare the information obtained for the rest of day, calculating a multiple of physical activity for each time point. Multiples of physical activity were classified in 3 categories: <1.3 as sedentary, 1.3-1.5 light and >1.5 moderate. With the aim to differentiate physical activity pattern throughout the day, percent of time invested on sedentary, light and moderate activities were calculated for the whole 24 day and the time awake.

3.5.7. Dietary Intake

Three non-consecutive 24-hour recall questionnaires were applied to each women to characterise their food intake. Food composition was calculated from the Chilean food composition database. Usual meals were chemically analysed by proximal analysis and its food composition was included in the food database (see Tables 4-6).

3.6. Statistical analysis

Data is presented as mean ± standard deviation (SD). For differences between supplementation periods and nutritional status t-test was used. For comparison between dietary treatment, paired t-test was applied. Pearson correlation was used to assess association between variables. Statistical significance was defined as $\alpha = 0.05$.

4. RESULTS

4.1. Plasma fatty acids profile

Changes in the plasma linoleic acid levels were not significantly different among periods and groups, baseline (885 ± 288 μg/mL), canola (884 ± 297 μg/mL) and sunflower (887 ± 206 μg/mL). In relation to plasma α-linolenic acid, there was a significant reduction in the sunflower compared to canola treatment. Baseline values were 18.8 ± 10.2 μg/mL; canola 23.3 ± 14.4 μg/mL and sunflower 14.2 ± 5.4 μg/mL (canola v/s sunflower, $p<0.03$). For polyunsaturated n6/n3 ratio, paired t-test comparisons showed that the whole group had a significant change among periods, being this difference mainly due to changes in the control group ($p = 0.02$). Group changes are shown in Table 7 and individual changes in Figure 1A.

TABLE 7: CHANGES IN POLYUNSATURATED N6/N3 RATIO ACCORDING DIETARY TREATMENT

	Baseline	**Canola (Can)**	**Sunflower (Sun)**	**Rate of change (Sun/Can)**
Control	56.7 ± 15.4	44.4 ± 15.3	70.9 ± 18.0	1.79 ± 0.71
Obese	48.3 ± 12.8	43.9 ± 18.2	65.4 ± 26.2	1.60 ± 0.49
Total	53.1 ± 14.5	44.2 ± 15.9	68.5 ± 21.1	1.71 ± 0.61

In relation to plasma polyunsaturated/saturated fatty acids (P/S) ratio, sunflower treatment produced a significant increase in the P/S ratio compared to canola treatment in the whole group (paired t-test, $p = 0.03$), with a higher change in obese subjects (Table 8). Individual variation for P/S ratio is shown in Figure 1B.

Figure 1A

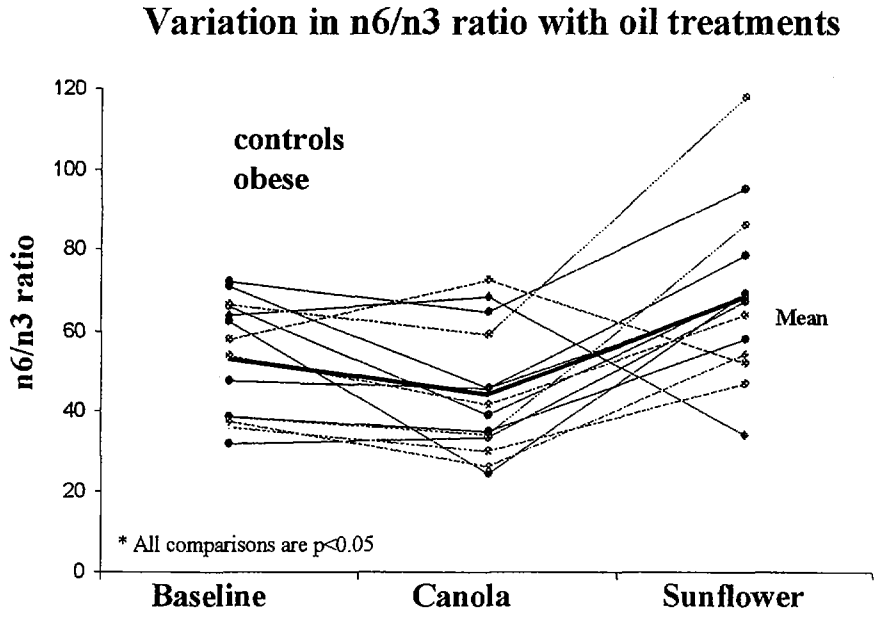

Figure 1B

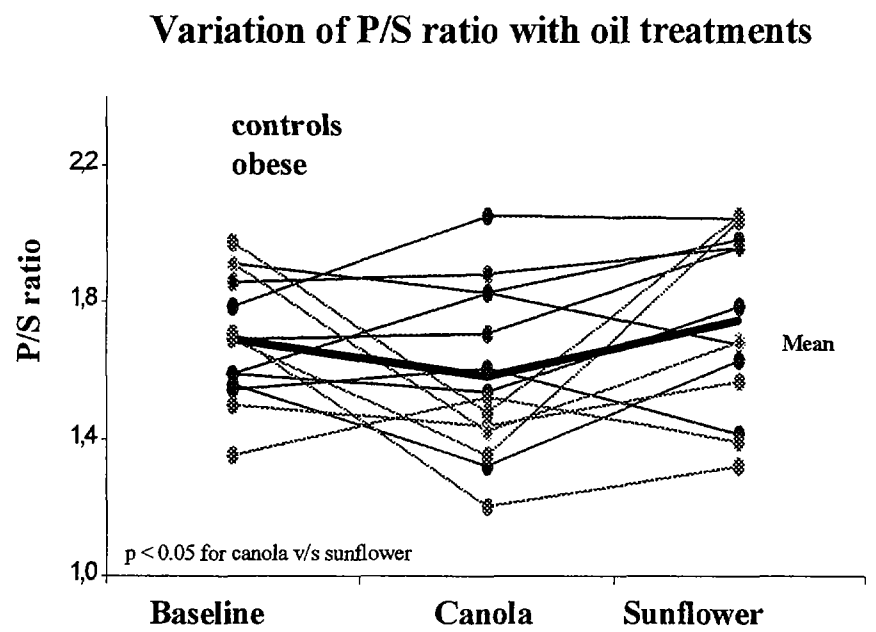

TABLE 8. CHANGES IN PLASMA P/S RATIO ACCORDING DIETARY TREATMENT

	Baseline	Canola	Sunflower	Rate of Change Sun/Can
Control	1.69 ± 0.14	1.72 ± 0.23	1.80 ± 0.22	1.06 ± 0.12
Obese	1.69 ± 0.23	1.40 ± 0.11	1.67 ± 0.31	1.20 ± 0.22
Total	1.69 ± 0.18	1.58 ± 0.24	1.75 ± 0.26	1.12 ± 0.18

$p < 0.05$ for all comparisons between sunflower and canola

4.2. Resting Energy Expenditure

As expected resting energy expenditure under fasting conditions was higher in obese women compared to control subjects. This difference was caused by the increased body mass and fat free mass since RMR was not different when calculated on per kg body weight or per kg fat free mass (FFM) basis. Respiratory quotient was similar in both groups.

TABLE 9. RESTING METABOLIC RATE (RMR)

	Control	Obese
RMR (kcal/d)	1289.3 ± 138.4	1587.5 ± 231.5*
RMR (kcal/kg/d)	22.9 ± 1.8	19.5 ± 1.7
RMR (kcal/FFM/d)	31.7 ± 3.5	36.7 ± 5.2
Respiratory Quotient	0.74 ± 0.03	0.76 ± 0.05
% CHO oxidised	20 ± 30	14 ± 12
% Fat oxidised	90 ± 20	85 ± 12

* $p<0.05$

4.3. Macronutrient oxidation

Macronutrient oxidation during the 200 min following the intake of test meals was studied by continuous indirect calorimetry. Results for carbohydrate and fat oxidation during the two oil treatments are described below.

4.4. Carbohydrate oxidation

Glucose oxidation was analysed as a proportion of carbohydrate intake (Ox/In). Dietary treatment with sunflower oil did not produce a statistically significant change in the Ox/In ratio during feeding conditions compared with canola treatment, whether in control (20.8 ± 11.5 v/s 22.1 ± 7.6, for canola and sunflower treatment, respectively) and obese subjects (20.0 ± 15.5 v/s 29.3 ± 7.0 for canola and sunflower treatment, respectively). Nevertheless, for sunflower treatment the amount of glucose oxidised per kg fat free-mass in 200 min was significantly higher in obese compared to controls (403 ± 84 mg/kg FFM v/s 279 ± 102 mg/kg FFM, $p<0.03$). The changes from canola to sunflower treatments (RC) for plasma n6/n3 ratio was not associated to RC for glucose oxidation in both groups. Similarly, the RC for P/S ratio was negatively related to RC for glucose oxidation, both in controls ($r = -0.51$) and obese ($r = -0.48$).

4.5. Fat oxidation

As performed with glucose oxidation, the ratio Ox/In was calculated. There was no significant change in this ratio with dietary treatment, whether in control (78.6 ± 19.4 v/s 100.4 ± 25.6, for canola and sunflower treatment, respectively) and obese subjects (92.9 ± 28.9 v/s 102.4 ± 9.5, for canola and sunflower treatment, respectively). In controls the amount of fat oxidised per kg fat free-mass in 200 min was significantly higher for sunflower compared to canola treatment (368 ± 93 mg/kg FFM v/s 295 ± 79 mg/kg FFM, $p<0.02$). Comparisons were made between the rate of change (RC) of sunflower v/s canola treatment (sunflower/canola ratio) for substrate oxidation as well as for plasma fatty acids. The RC for plasma n6/n3 ratio and RC for fat oxidation (mg/kg FFM) had a positive correlation with fat oxidation in controls ($r = 0.72$), and negatively associated in obese ($r = -0.79$). On the other hand, RC for plasma P/S ratio was not associated to fat oxidation in both groups.

4.6. Serum glucose and insulin

The AUC for glucose was not different for nutritional conditions or dietary treatment comparisons. Although in obese, sunflower glucose AUC was on average 116 ± 3 % of the canola treatment glucose AUC (p = NS).

Obese had higher insulin AUC compared to controls with sunflower treatment ($p<0.05$). Obese had also significantly higher insulin AUC with sunflower compared to canola treatment ($p<0.02$) (Table 10). The change in insulin AUC from canola to sunflower was not associated to increased glucose oxidation in obese ($r = 0.17$). Plasma fatty acids either as RC for P/S or n6/n3 ratio were not related to insulin response.

Fat oxidation, was negatively associated to insulin AUC in controls ($r = -0.65$), but not in obese for sunflower treatment. The same was found for canola treatment, with a lower association ($r = -0.41$).

TABLE 10. GLUCOSE AND INSULIN AUC

	Insulin AUC		**Glucose AUC**	
	Canola	**Sunflower**	**Canola**	**Sunflower**
Control	1114 ± 255	1158 ± 432[b]	1071 ± 823	1127 ± 949
Obese	1862 ± 1286[a]	2303 ± 1379 [a,b]	1247 ± 853	1653 ± 940

a: $p<0.02$; b: $p< 0.05$

4.7. Insulin resistance

Homeostatic model assessment (HOMA) used to diagnose insulin resistance in our study, revealed that on average there were no differences between the groups and treatments. The corresponding fasting glucose and insulin levels are shown in Table 11.

TABLE 11: SERUM GLUCOSE, INSULIN AND HOMA BY NUTRITIONAL CONDITIONS AND DIETARY TREATMENT.

	Baseline		Canola		Sunflower	
	Control	**Obese**	**Control**	**Obese**	**Control**	**Obese**
Insulin (μU/mL)	7.7±2.2	10.3±5.6	8.1±4.6	12.3±7.5	7.8±2.3	11.3±7.0
Glucose (mg/dL)	88.2±9.0	93.7±14.7	87.7±13.2	93.2±16.6	89.7±4.9	97.5±15.0
HOMA	1.7±0.7	2.4±1.5	1.8±1.2	2.9±2.0	1.8±0.5	2.8±1.6

4.8. Physical activity

The pattern of physical activity did not show any differences between controls and obese both during the whole day or the time awake (Table 12). In general, the two groups can be considered as sedentary, with no more than 20% of active time devoted to moderate activities. Sleeping time was nearly 8 h in both groups.

On average, heart rate (HR) during daytime, expressed as multiples of the HR during sleeping time, was not higher than 1.3 in both groups (1.32 ± 0.05 in obese and 1.32 ± 0.11 in controls). This is another way of demonstrating the degree of sedentarism found in the study women.

TABLE 12: PHYSICAL ACTIVITY PATTERN AS PERCENT OF THE WHOLE DAY (INCLUDING SLEEPING TIME) AND DAYTIME (AWAKENED TIME)

Whole day	**Control**	**Obese**
Sedentary	65.2 ± 8.4	59.4 ± 7.2
Light	21.9 ± 7.2	25.7 ± 5.2
Moderate	12.9 ± 8.8	15.0 ± 3.7
Time awake		
Sedentary	47.2 ± 12.8	44.9 ± 14.5
Light	32.8 ± 8.2	34.5 ± 8.8
Moderate	20.2 ± 14.4	20.6 ± 7.1

4.9. Dietary Intake

There were no significant differences between the two groups in the amount of energy and/ or any of the nutrients analysed (Table 13). Obese women tended to have a lower energy intake but due to the large variability observed in the two groups this difference did not reach statistical significance. The trend nevertheless, may be explained by a higher degree of underreporting in obese women as has been identified by other authors.

TABLE 13: DIETARY INTAKE BY 24-H RECALL QUESTIONNAIRES IN THE STUDIED WOMEN

Nutrient	Control	Obese
Energy (kcal/d)	2051 ± 740	1892 ± 532
Protein (g)	66 ± 36	58 ± 19
Carbohydrates (g)	319 ± 108	281 ± 69
Fibre (g)	7 ± 6	9 ± 6
Total fat (g)	57 ± 28	61 ± 31
Saturated (g)	13 ± 8	17 ± 9
Monounsat (g)	16 ± 9	21 ± 12
Polyunsat (g)	14 ± 8	17 ± 12
n-6 fatty acids (g)	9.12 ± 6.15	10.98 ± 7.36
n-3 fatty acids (g)	0.81 ± 0.61	0.74 ± 0.69

5. DISCUSSION

This study was able to show a significant modification in the plasma fatty acid profile with the use of two types of oil. The observed changes were in the expected direction. Plasma α-linolenic acid was 1.7-fold higher for canola compared to sunflower treatment. Other study ([13]) found an increment of 3.6-fold higher compared to baseline after 2 weeks supplementation with foods enriched with n3 fatty acids. Their estimated intake was on average 7-fold higher than our canola supplementation, which is about 1–1.5 g/d. It is necessary to mention that our study was designed to achieve an n3 fatty acid intake close to recommendation with available foods, modifying only the type of oil consumed. The minimum recommended intake of α-linolenic acid according British Nutrition Foundation for our study subjects should be 0.4 – 0.5 g/d ([14,15]), whereas the adequate amount is 2 – 2.5 g/d according to a National Institute of Health report ([16]). To comply with those recommendations is considered a difficult task with the usually available foods and this presently being discussed by Adam ([17]).

Knowing the differential rate of fatty acid oxidation in animal ([1]) and human ([2]) studies, diets predominantly enriched on particular fatty acids could influence not only its own rate of oxidation and deposition, but also the route followed by other fatty acids in the body. Our study found that sunflower treatment in control group, induced a higher rate of fat oxidation, which was directly associated to an increased polyunsaturated n6/n3 ratio ($r = 0.72$). These results are contrary to our hypothesis, since we expected a higher rate of fat oxidation with canola treatment, because of its greater content of ALA. Nevertheless, in any circumstances the amount of LA will exceed largely the amount of ALA eaten. For example, considering the amount of LA supplied by the canola breakfast, it represented 81% of the total PUFA intake. The corresponding value for sunflower meal was 97% (see Table 5). Based on the recent data published by Delany et al. ([2]), the oxidation rate at 3h for LA was 7.5% and ALA 12.5%.

When these proportions are applied to our test meal, the expected mean differences in fat oxidation will be 2.5 and 3.5 g for canola and sunflower breakfast, respectively.

Glucose oxidation was negatively associated to the rate of change of P/S ratio from canola to sunflower treatment in both groups. The same finding was described for high dietary P/S ratio which induced a lower glucose oxidation ([18]) and reduced glycolitic enzyme activity ([19]). In spite of this, it can not be assured that this mechanism is operating in our study since the modifications in P/S ratio produced by our dietary treatments were small compared with these studies.

In our study both treatments produced greater insulin AUC in obese compared to controls. This difference was statistically significant only for sunflower treatment. This is in accordance to animal studies ([20,21]), where LA rich diets have induced insulin resistance associated to hyperinsulinemia. In spite of this, our obese subjects can not be considered as insulin resistant, since their insulin and glucose response to OGTT was normal and no changes in HOMA among periods were found.

In summary, this study showed that fatty acids changes within the range of normal diet can induce differences in substrate oxidation, which are dependent on nutritional condition of the subjects. Canola oil intake could be stimulated based on its smaller insulin response, and could be used to prevent hyperinsulinemia and insulin resistance in predisposed subjects. Finally, n3 fatty acids recommended intake should take into account how feasible is to meet the proposed level within the range of the usually available foods. On the other hand, development of foods that are technologically or naturally enriched, should be encouraged.

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