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Postprandial triglyceridaemia is modulated by insulin resistance but not by grade of obesity in abdominal and morbid obese subjects

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Abstract

Background: Obesity is associated with high cardiovascular risk. Postprandial lipidaemia has been associated with cardiovascular disease risk. Our aim was to identify whether anthropometric parameters, insulin resistance (IR) and/or fasting plasma triglycerides may determine postprandial changes in lipoprotein concentrations in abdominal and morbid obese subjects.

Methods: We have studied 20 non-diabetic, normolipidaemic subjects with abdominal obesity, 20 morbid obese subjects and 20 healthy individuals, that have similar age and gender. In all of them a standardised oral fat load test (OFLT) with unsaturated fat was performed.

Results: During the OFLT, the postprandial triglycerides response was significantly higher in subjects with abdominal obesity compared with morbid obese subjects (4 hours triglycerides pick value and AUC of triglycerides). Both obese groups showed significantly higher postprandial triglycerides response compared with healthy subjects. Dividing the obesity group according to the presence of IR, we found that IR was an important factor related with postprandial lipaemia but not BMI or waist circumference. In addition, postprandial glycaemia and insulinaemia significantly decreased in all studied subjects, being the highest decrease in morbid obese subjects and in subjects with IR. Postprandial triglyceridaemia significantly correlated with IR parameters and not with anthropometric parameters in AO and MO subjects.

Conclusion: In subjects with AO and MO, postprandial triglycerides values are higher than healthy individuals and independently predicted by fasting IR parameters. Furthermore, unsaturated fat improved IR state.

1 | INTRODUCTION

Obesity is an increasingly prevalent disease, which has reached epidemic proportions. According to the World Health Organization about 13% of the world's adult population was obese in 2014, affecting over 600 million people worldwide.[1] In addition, obese patients have a higher risk for cardiovascular disease and suffer from other comorbidities such as dyslipidaemia, type 2 diabetes mellitus or hypertension.[2] Insulin resistance (IR) has been implicated in the pathogenesis of these metabolic comorbilities,[3] and is a risk factor for cardiovascular disease.[4]

However, clinical experience and recent studies support that not all obese patients are at high metabolic and cardiovascular risk.[5] Therefore, the challenge is to identify clinical or biochemical markers indicating those obese individuals with a higher risk of developing metabolic or vascular complications.

Previous studies have assessed a correlation of fasting plasma lipoproteins values and cardio-metabolic complications. However, this situation only is present after a long nighttime fasting period.[2,3] On the contrary, individuals in developed countries spend most of the day in a postprandial state. Fat consumption may stimulate a transient metabolic and low inflammatory response.[4] The magnitude of this response reflects the subject's ability to efficiently adjust to nutrient intake. It can be altered in metabolic risk phenotypes such obesity or increased visceral adipose tissue.[6] In fact, increased levels of non-fasting triglycerides have been associated with vascular complications.[7]

Nonetheless, the differences of lipoprotein concentrations in postprandial state between individuals with morbid obesity (MO), individuals with abdominal obesity (AO) and healthy subjects are poorly understood yet. The knowledge of these differences could be useful to predict which individuals are at a higher risk for developing metabolic and vascular complications. It also would be of interest to evaluate whether adiposity (body mass index—BMI—and/or waist circumference) or IR are responsible of the postprandial lipoprotein response.

Thus our objective was to study the impact of an oral fat load test (OFLT) on serum lipoproteins in individuals with MO, AO and healthy volunteers and to evaluate which is the most important predictor of postprandial changes in obesity.

What's known

- Obesity has been considered as a risk factor for several diseases. However, not all the subjects with obesity develop metabolic disturbances or cardiovascular disease.
- One of the factors implicated in the development of cardiovascular disease is postprandial lipaemia.

What's new

- It would be of interest to detect which obese subjects will be at risk for developing metabolic complications.
- The most important factor implicated in postprandial lipaemia in subjects with obesity is insulin resistance, but not BMI or waist circumference.
- It could be useful to evaluate the presence of insulin resistance in all the patients with obesity in order to intensify the treatment.
- The general recommendation in obese subjects is to reduce general fat ingestion. However, based on our results, we speculate that unsaturated fat could be added in the usual recommendations for obesity.

2 | SUBJECTS AND METHODS

2.1 | Subjects

We selected by consecutive sampling 20 patients with AO (9 women) and 20 patients with MO (9 women) from the Obesity Unit of our center and 20 healthy individuals (9 women) with similar age and gender distribution.

We defined AO as waist circumference cut-off points of ≥102 cm for men and ≥88 cm for women.[8] Patients with AO had a BMI between 30 and 35 kg/m^2. The diagnosis of MO was based on a BMI ≥ 40 kg/m^2.[1,2] Healthy individuals had a BMI between 20 and 25 kg/m^2 and normal waist circumference (<88 cm for women/<102 cm for men).

All selected subjects were non-diabetic (fasting glucose < 100 mg/dL and HbA1c < 6.5%) and non-dyslipidaemic (total plasma cholesterol concentration and triglycerides < 200 mg/dL), as it is known that these parameters can influence postprandial metabolism.

Exclusion criteria were: clinical manifestations of cardiovascular disease (acute myocardial infarction, aorto-coronary bypass, coronary angioplasty, angina pectoris and positive exercise stress test, stroke or intermittent claudication in the last 10 years), congestive heart failure (NYHA ≥ II), oxygen therapy, liver or kidney disease, active cancer disease, hypothyroidism (TSH > 10 mU/mL), smoking habit and consumption of >30 g alcohol per day.

The institutional ethics committee approved the study protocol (2012/077). All subjects signed a written informed consent.

3 | METHODS

3.1 | Clinical and anthropometric parameters

An experimented researcher carried out a complete medical history and physical examination in all the subjects. Blood pressure was measured in the sitting position after a 10-minutes rest, with two separated measurements; BMI was calculated as the weight in kilograms divided by height in metres squared; abdominal circumference was measured in centimetre in the midpoint between the

iliac crest and the lowest rib. The same researcher performed all measurements.

3.2 | Biochemical parameters

Blood samples were drawn from an antecubital vein following an overnight 12 hours fasting period. Total cholesterol and triglycerides levels were measured by standard enzymatic techniques. High-density lipoprotein cholesterol (HDL-C) was measured after precipitation of apoB-containing lipoproteins with polyanions and VLDL cholesterol (very low-density lipoprotein cholesterol) after separation of VLDL ($d < 1.006$ g/mL) by ultracentrifugation. The low density lipoprotein cholesterol (LDL-C) was calculated by subtraction of VLDL and HDL cholesterol from total cholesterol. Total plasma apoB was measured by immunoturbimetry. The coefficients of variation for lipids and lipoproteins were <7%. Glucose was determined using enzymatic methods. Insulin was determined using a standardised ELISA. The homoeostasis model assessment (HOMA) index which is defined as fasting glucose (mmol/L) × fasting insulin (mIU/L)/22.5. IR was considered when HOMA index was ≥3.2. All procedures were standard as previously described.[9]

3.3 | Oral fat load test

All the individuals were submitted to an OFLT with a commercial liquid preparation of long-chain triglycerides (Supracal, SHS International, Liverpool, UK). Each 100 mL contains 50 g of fat (450 kcal) of which 9.6 g are saturated, 28.2 g are monounsaturated (MUFA) and 10 g are polyunsaturated (PUFA), and the $\omega 6/\omega 3$ fatty acid ratio is greater than 20:1. The fatty acid content and complete composition can be obtained from the SHS International Ltd., Nutricia web site.

AO patients and healthy subjects consumed a high-fat meal containing 50 g fat per m^2 body surface. MO subjects consumed a 60 g high-fat meal (125 mL of Supracal), as performed by other investigation groups, since the calculated dose by m^2 body surface in these patients seems disproportionate.[10]

The study started at 8.30 AM after a 12 hours overnight fast. A cannula for venous blood sampling was placed and subjects rested for 30 minutes before the first blood sample extraction. After that, the OFLT was initiated. Participants remained sitting or supine during the next 8 hours and were only allowed to drink mineral water. Peripheral blood samples were obtained in sodium EDTA before and at regular 2-hours time intervals for 8 hours after the OFLT.

3.4 | Statistical analysis

Power analysis was performed to calculate the sample size using GPower 3.1 programme. The calculated sample size was 54 individuals (18 at each group with similar age and gender distribution) with given α (0.05), power (0.9) and effect size (0.5 based on previous data because postprandial situation should duplicate the differences expected on the fasting state).

Data were analysed using the Statistical Package for the Social Sciences (SPSS 20 for Windows; SPSS Chicago, IL, USA). Results are expressed as mean ± standard deviation. The *P*-values were two-tailed and a *P*-value of less than .05 was considered significant.

TABLE 1 Clinical and biochemical characteristics of the studied subjects

	Healthy subjects (n = 20)	Abdominal obese (n = 20)	Morbid obese (n = 20)
Age (years)	38.8 ± 10.1	39.5 ± 10.8	41.3 ± 11.9
Gender (M/F)	9/11	9/11	9/11
Waist circumference (cm)	86.7 ± 8.9	105.0 ± 11.9*	132.5 ± 14.3*,**
BMI (kg/m^2)	24.2 ± 2.7	32.3 ± 2.5*	45.8 ± 4.5*,**
SBP (mm Hg)	116.9 ± 13.3	121.7 ± 15.1	128.8 ± 14.4*
DBP (mm Hg)	70.5 ± 9.3	80.6 ± 10.2*	78.8 ± 13.3*
Fasting glucose (mg/dL)	89.7 ± 6.1	90.8 ± 6.1	87.5 ± 11.1**
Fasting insulin (μU/mL)	5.2 ± 2.8	16.8 ± 8.9*	19.7 ± 11.6*
HOMA index	1.2 ± 0.64	3.8 ± 2.3*	4.4 ± 3.0*
Total cholesterol (mg/dL)	178.3 ± 27.9	211.4 ± 23.1*	162.7 ± 43.4**
HDL-C (mg/dL)	60.3 ± 11.3	53.7 ± 10.2	48.4 ± 10.2*,**
LDL-C (mg/dL)	105.5 ± 23.2	132.3 ± 15.7*	114.9 ± 41.5**
Triglycerides (mg/dL)	68.6 ± 21.4	138.0 ± 71.5*	117.7 ± 42.5*
ApoB (mg/dL)	79.2 ± 17.6	97.7 ± 12.9*	88.5 ± 21.8*

Abbreviations: Apo, apolipoprotein, BMI, body mass index, DBP, diastolic blood pressure, HDL-C, high-density lipoprotein cholesterol, HOMA, homoeostasis model assessment, LDL-C, low-density lipoprotein cholesterol, SBP, systolic blood pressure.

*$P < .05$ versus healthy; **$P < .05$ versus abdominal obesity.

TABLE 2 AUC and changes during the OFLT of triglyceridaemia, glycaemia, insulinaemia and HOMA index according to the grade of obesity

	Healthy subjects (n = 20)	Abdominal obese (n = 20)	Morbid obese (n = 20)
Glucose (mg/dL)			
0 hour	89.7 ± 6.1	90.8 ± 6.1	87.5 ± 11.1**
2 hours	87.1 ± 7.1***	91.9 ± 6.6	86.5 ± 8.1**
4 hours	86.1 ± 7.8***	89.6 ± 8.8	82.5 ± 7.8**,***
6 hours	84.3 ± 5.3***	86.9 ± 7.1	79.2 ± 7.7**,**,***
8 hours	84.1 ± 6.6***	86.4 ± 5.5**	77.3 ± 8.7**,**,***
Insulin (μU/mL)			
0 hour	5.2 ± 2.8	16.8 ± 8.9*	19.7 ± 11.6*
2 hours	5.1 ± 2.6	15.2 ± 7.9*	11.9 ± 10.0*,***
4 hours	4.4 ± 2.4	12.8 ± 7.4*,***	9.2 ± 7.6*,***
6 hours	3.6 ± 2.8***	8.6 ± 4.0*,***	9.1 ± 6.1*,***
8 hours	3.1 ± 1.9***	7.9 ± 5.3*,***	8.4 ± 4.6*,***
HOMA index			
0 hour	1.2 ± 0.6	3.8 ± 2.3*	4.4 ± 3.0*
2 hours	1.1 ± 0.6	3.5 ± 1.8*	2.6 ± 2.2*,***
4 hours	0.9 ± 0.6	2.9 ± 1.7*,***	1.9 ± 1.6*,***
6 hours	0.7 ± 0.6***	1.8 ± 0.8*,***	1.8 ± 1.2*,***
8 hours	0.6 ± 0.4***	1.7 ± 1.1*,***	1.6 ± 0.9*,***
Triglycerides (mg/dL)			
0 hour	68.7 ± 21.4	138.0 ± 71.5*	117.7 ± 42.5*
2 hours	107.8 ± 49.1***	180.4 ± 75.4*,***	122.6 ± 43.7**,***
4 hours	121.9 ± 59.4***	219.6 ± 81.3*,***	138.2 ± 53.9**,***
6 hours	95.1 ± 66.4***	151.5 ± 52.6*	136.8 ± 57.8*,***
8 hours	65.6 ± 20.4	115.1 ± 40.1*	118.3 ± 47.6*
AUC			
AUC glucose (mg × $dL^{-1} \times h^{-1}$)	688.8 ± 47.6	713.9 ± 49.2	661.2 ± 58.5**
iAUC glucose (mg × $dL^{-1} \times h^{-1}$)	37.9 ± 24.6	39.9 ± 33.3	51.3 ± 40.5
AUC insulin (μU × $mL^{-1} \times h^{-1}$)	34.4 ± 16.4	97.9 ± 47.8*	88.6 ± 49.2*
iAUC insulin (μU × $mL^{-1} \times h^{-1}$)	15.2 ± 13.2	44.1 ± 32.6	71.5 ± 63.6*
AUC HOMA (h^{-1})	7.4 ± 3.7	21.8 ± 10.7*	18.6 ± 10.7*
iAUC HOMA (h^{-1})	3.6 ± 3.3	10.3 ± 7.8	17.1 ± 18.1*
AUC triglycerides (mg × $dL^{-1} \times h^{-1}$)	784.1 ± 330.5	1356.3 ± 486.2*	1031.3 ± 381.1**
iAUC triglycerides (mg × $dL^{-1} \times h^{-1}$)	258.7 ± 192.1	336.9 ± 191.8	149.4 ± 128.3**

Abbreviations: AUC, area under the curve; HOMA, homoeostasis model assessment; iAUC, incremental area under the curve; OFLT, oral fat load test.

*$P < .05$ versus healthy; **$P < .05$ versus abdominal obesity; ***$P < .05$ versus basal at the same group.

Because of the sample size and the measurement of variables that do not fulfill the criteria of normality, non-parametric tests were used. The Mann-Whitney test was used to assess differences in measured parameters at various time intervals after the OFLT between groups. The Wilcoxon test was used for comparison of data before and after the OFLT in the same subject. Fisher's exact test was used to analyse the differences in qualitative parameters. Spearman's correlation was used to assess the degree of association between two quantitative variables.

A linear regression analysis was used to assess multiple associations. Triglycerides values at 4 hours after OFLT, as expression of postprandial

TABLE 3 Clinical and biological characteristics of the studied subjects according to the presence or absence of insulin resistance

	Healthy subjects (n = 20)	Obese non-IR (n = 18)	Obese with IR (n = 22)
Age (years)	38.85 ± 10.1	42.21 ± 10.5	41.4 ± 12.6
Gender (M/F)	9/11	11/7	11/11
Waist circumference (cm)	86.7 ± 8.93	115.6 ± 19.9*	124.7 ± 17.8*,**
BMI (kg/m^2)	24.25 ± 2.67	38.1 ± 6.8*	42.2 ± 8.0*,**
SBP (mm Hg)	116.95 ± 13.33	121.9 ± 13.2	128.3 ± 15.8*,**
DBP (mm Hg)	70.50 ± 9.29	76.9 ± 9.7	81.5 ± 13.2*,**
Fasting glucose (mg/dL)	89.70 ± 6.1	88.1 ± 9.4	89.5 ± 9.7
Fasting insulin (μU/mL)	5.20 ± 2.76	10.7 ± 3.2*	25.0 ± 10.2*,**
HOMA index	1.10 ± 0.64	2.21 ± 0.6*	5.6 ± 2.8*,**
Total cholesterol (mg/dL)	178.30 ± 27.97	172.1 ± 38.3	190.3 ± 46.7*
HDL-C (mg/dL)	60.30 ± 11.34	47.4 ± 12.3*	42.2 ± 12.6*
LDL-C (mg/dL)	105.50 ± 23.16	111.4 ± 25.6	130.4 ± 38.7*
Triglycerides (mg/dL)	68.65 ± 21.39	98.3 ± 27.3*	148.3 ± 63.5*,**
ApoB (mg/dL)	79.20 ± 17.63	88.0 ± 17.3*	95.6 ± 20.2*

Abbreviations: Apo, apolipoprotein; BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; HOMA, homoeostasis model assessment; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure.

*$P < .05$ versus healthy; **$P < .05$ versus obese non-IR.

lipaemia,[11] were used as dependent variable and anthropometric (BMI and waist circumference) and metabolic variables (HOMA index and fasting triglycerides) were used as independent variables.

4 | RESULTS

Clinical and biochemical characteristics in fasting state of healthy, abdominal obese and morbid obese subjects are shown in Table 1. Comparing the three groups, there were no significant differences in age or gender distribution. However, as expected, we found differences regarding BMI and waist circumference. Fasting plasma triglycerides, insulinaemia and HOMA index were significantly higher in both groups of obesity compared with healthy subjects.

Postprandial values of the evaluated parameters in the three groups are shown in Table 2. During the OFLT we found an increase in postprandial triglycerides achieving the maximum at 4 hours and recovering fasting values after 8 hours. At 2 and 4 hours, triglycerides values were significantly higher in AO subjects compared with MO. In addition, the AUC of triglycerides was highest in AO subjects compared with MO and healthy subjects. No significant changes of total cholesterol, LDL-C, HDL-C and apo B values were found in the postprandial estate.

Postprandial glycaemia and insulinaemia showed a significant decrease in all the groups. Significantly greater decreases in insulin levels were found in both obese groups compared with the decrease observed in healthy subjects (52.8% AO and 57.3% MO vs 40.3% controls, $P < .01$; Table 2).

In addition, the iAUC of triglycerides was highest in AO subjects compared with MO subjects.

When we divided the subjects with obesity according to the presence of IR, we found that those with IR showed the worst metabolic profile (Table 3). During the OFLT (Table 4 and Figure 1) we found an increase in postprandial triglycerides achieving the maximum at 4 hours and recovering fasting values after 8 hours. Significantly greatest decrease in insulin levels was found in the group of obese with IR (59.2% in obese with IR vs 45.8% in obese without IR and 40.3% in controls, $P < .01$). Moreover, the AUC of triglycerides was higher in subjects with IR compared with the other two groups. However, although iAUC of triglycerides was higher in obese subjects with IR, there were not significant differences with obese without IR.

We also performed a correlation analysis (Table 5). The maximum triglycerides values observed at 4 hours and AUC of triglycerides, both as expression of postprandial hyperlipaemia,[11] significantly correlated with HOMA index and fasting triglyceridaemia. However, there was not association with BMI or waist circumference.

Finally, to determine independent correlates of peak triglyceridaemia (triglycerides at 4 hours after the OFLT), forward step-wise linear regression analysis was performed, including as covariates the univariate correlates identified in the Spearman test. In regression analysis, HOMA index and fasting triglyceridaemia emerged as positive determinants ($P < .05$). However, BMI and waist circumference were not independent predictors of postprandial triglycerides response.

TABLE 4 AUC and changes during the OFLT of triglyceridaemia, glycaemia, insulinaemia and HOMA index according to the presence or absence of insulin resistance

	Healthy subjects (n = 20)	Obese non-IR (n = 18)	Obese with IR (n = 22)
Glucose (mg/dL)			
0 hour	89.7 ± 6.1	88.1 ± 9.4	89.5 ± 9.7
2 hours	87.1 ± 7.1***	89.1 ± 8.6	88.5 ± 7.3
4 hours	86.1 ± 7.8***	85.5 ± 11.3	85.3 ± 6.6***
6 hours	84.3 ± 5.3***	82.8 ± 9.8***	81.9 ± 7.2***
8 hours	84.1 ± 6.6***	81.6 ± 10.5***	80.5 ± 7.3***
Insulin (μU/mL)			
0 hour	5.2 ± 2.8	10.7 ± 3.2*	25.0 ± 10.2*,***
2 hours	5.1 ± 2.6	7.5 ± 5.5*	17.9 ± 9.2*,***,***
4 hours	4.4 ± 2.4	6.3 ± 4.9*,***	14.2 ± 7.8*,***,***
6 hours	3.6 ± 2.8***	6.8 ± 2.3*,***	10.5 ± 6.5*,***,***
8 hours	3.1 ± 1.9***	5.8 ± 2.3*,***	10.2 ± 5.5*,***,***
HOMA index			
0 hour	1.2 ± 0.6	2.2 ± 0.6*	5.6 ± 2.8*,***
2 hours	1.1 ± 0.6	2.3 ± 1.3***	4.7 ± 1.5*,***,***
4 hours	0.9 ± 0.6	1.9 ± 1.4***	4.0 ± 1.5*,***,***
6 hours	0.7 ± 0.6***	1.4 ± 0.7*,***	2.1 ± 0.8*,***,***
8 hours	0.6 ± 0.4***	1.0 ± 0.8*,***	1.1 ± 0.9*,***,***
Triglycerides (mg/dL)			
0 hour	68.7 ± 21.4	98.3 ± 27.3*	148.3 ± 63.5*,***
2 hours	107.8 ± 49.1***	127.2 ± 45.3***	160.9 ± 74.0*,***
4 hours	121.9 ± 59.4***	141.4 ± 56.4***	194.8 ± 83.8*,***,***
6 hours	95.1 ± 66.4***	122.0 ± 35.33*,***	159.6 ± 63.6*,***
8 hours	65.6 ± 20.4	102.6 ± 30.7*	128.9 ± 50.4*,***
AUC			
AUC glucose (mg × dL^{-1} × h^{-1})	688.8 ± 47.6	684.6 ± 74.1	681.4 ± 48.8
iAUC glucose (mg × dL^{-1} × h^{-1})	37.9 ± 24.6	46.5 ± 38.5	46.7 ± 37.9
AUC insulin (μU × mL^{-1} × h^{-1})	34.4 ± 16.4	57.9 ± 21.3	120.4 ± 45.9*,***
iAUC insulin (μU × mL^{-1} × h^{-1})	15.2 ± 13.2	31.3 ± 14.6	84.4 ± 63.4*,***
AUC HOMA (h^{-1})	7.4 ± 3.7	12.4 ± 5.7	25.7 ± 10.0*,***
iAUC HOMA (h^{-1})	3.6 ± 3.3	6.6 ± 2.8	20.3 ± 17.8*,***
AUC triglycerides (mg × dL^{-1} × h^{-1})	784.1 ± 330.5	982.1 ± 297.4	1307.0 ± 504.5*,***
iAUC triglycerides (mg × dL^{-1} × h^{-1})	258.7 ± 192.1	203.8 ± 187.5	241.3 ± 176.8

Abbreviations: AUC, area under the curve; HOMA, homoeostasis model assessment; iAUC, incremental area under the curve; OFLT, oral fat load test.

*$P < .05$ versus healthy; **$P < .05$ versus obese non-IR; ***$P < .05$ versus basal at the same group.

5 | DISCUSSION

Our results show that non-diabetic normolipidaemic subjects with abdominal or morbid obesity had higher fasting and postprandial triglyceridaemia values during the OFLT compared with controls. There is enough evidence indicating that the decrease in insulin action in IR states increases lipolysis and promotes higher fasting and postprandial triglycerides response.[10] Our results are according to these observations. In fact, when we divide the group of obesity according to the presence of IR, the obese subjects with IR show significantly higher postprandial lipaemia while healthy controls and obese non-insulin resistant do not show significant differences between them.

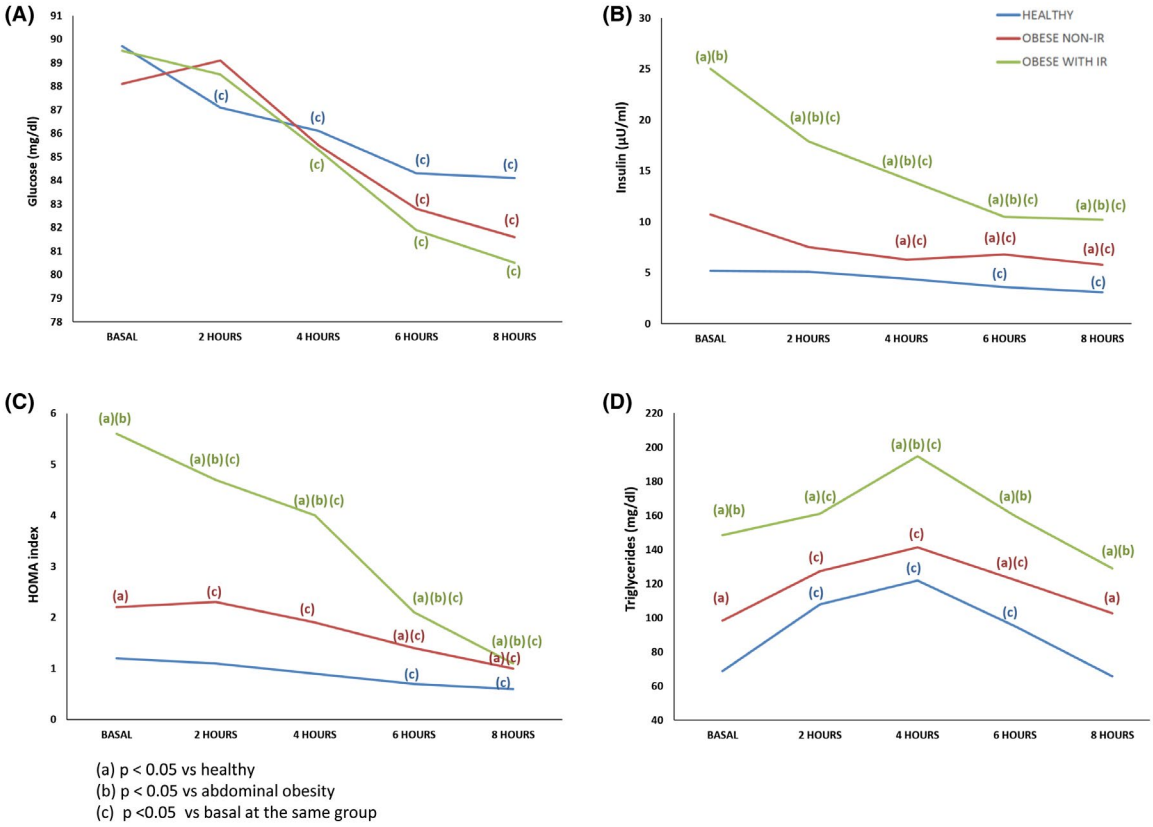

FIGURE 1 Plasmatic changes during the oral fat load test in control subjects and subjects with abdominal obesity and morbid obesity. A, Glycaemia during the oral fat load test. B, Insulinaemia during the oral fat load test. C, HOMA during the oral fat load test. D, Triglyceridaemia during the oral fat load test

Furthermore, postprandial lipaemia (peak of triglycerides and iAUC of triglycerides)[11] was significantly correlated with HOMA index and triglycerides, but not with BMI or waist circumference in subjects with obesity. In the same line, linear regression analysis showed that only triglycerides and HOMA were predictors of postprandial lipaemia. Thus, the most important factor in obesity which influences the triglycerides homoeostasis is the metabolic phenotype. It is in the line of the concept of obese metabolically healthy.[12] In the last years, it has been suggested that not all obese subjects will develop cardio-metabolic complications, paying more attention to the different metabolic phenotypes of obesity. In this sense, one of the characteristics of the metabolically unhealthy obesity is the presence of IR.[13]

Overweight and obesity have been associated with exaggerated postprandial lipaemia.[14-16] Taking into account that postprandial lipaemia has been associated with atherogenic factors,[17] it would be of great interest to detect those individuals with higher risk. Previous studies have found that BMI and abdominal circumference were correlated with postprandial triglyceridaemia.[18,19] On the contrary, in contrast to the commented studies, our results in MO and AO subjects showed no correlation between anthropometric parameters (BMI and abdominal circumference) and peak of postprandial triglycerides (4 hours after OFLT), nor with the iAUC of triglycerides. However, we found that postprandial triglyceridaemia was independently associated with a higher fasting IR state (Tables 4 and 5). Similar results were found by van Wijk et al [20] Thus, our study shows that IR has a higher impact on postprandial triglycerides response after an acute OFLT than BMI. In the same line, Perez-Martinez et al found that metabolically healthy patients displayed lower postprandial response. [21]

Our study has also shown a significant reduction in postprandial glycaemia and insulinaemia in abdominal and morbidly obese subjects. A greater decrease in glucose (12% at 8 hours) and insulin values (58% at 8 hours) after the OFLT was found in MO subjects compared with the AO subjects. Previous studies have also demonstrated that unsaturated fat improved fasting and postprandial IR. [22-25] Furthermore, other dietary interventional studies have also demonstrated that enriched meals with unsaturated fat improved fasting and postprandial triglyceridaemia.[26,27] Taken together all these data, and considering that IR determines daylong triglyceridaemia, playing a determinant role in development of postprandial lipid abnormalities, it could be suggested that the reduction of IR induced by unsaturated fat could be useful to reduce postprandial triglyceridaemia.

However, our study has some limitations. We have used an OFLT. It is an acute intervention. Thus we cannot extrapolate long-term beneficial effects of this fat on postprandial metabolism. In addition, this is not a physiological intake of this macronutrient. Furthermore, we do not have a different model to compare whether different kind

TABLE 5 Correlation coefficients among postprandial lipaemia indicators and different variables in subjects with obesity

	Peak of triglycerides (4 h) (mg/dL)	iAUC triglycerides (mg × dL^{-1} × h^{-1})
Waist circumference (cm)	*r* −0.291 *P* = .077	*r* −0.254 *P* = .123
BMI (kg/m^2)	*r* 0.206 *P* = .203	*r* 0.289 *P* = .071
Fasting Glucose (mg/dL)	*r* 0.259 *P* = .106	*r* 0.426 *P* = .006
Fasting insulinaemia (μU/mL)	*r* 0.445 *P* = .004	*r* 0.242 *P* = .133
HOMA index	*r* 0.494 *P* = .001	*r* 0.323 *P* = .044
Triglycerides (mg/dL)	*r* 0.797 *P* < .001	*r* 0.334 *P* = .035

Abbreviations: AUC, area under the curve; BMI, body mass index; HOMA, homoeostasis model assessment; iAUC, incremental area under the curve.

of fat exerts different results than unsaturated fat. Finally, we cannot exclude the influence of genetic and environmental factors in the modulation of postprandial lipaemia.[28]

In summary, after an OFLT using predominantly unsaturated fat the postprandial triglycerides response was higher in obese subjects with IR. However, despite the increase in triglycerides, there was a significant decrease in glycaemia and insulinaemia in healthy normoweight subjects, and in non-diabetic normolipidaemic AO and MO subjects. The decrease was higher in subjects with IR. Moreover, postprandial triglyceridaemia significantly correlated with IR parameters and not with anthropometric parameters in obese groups. The exact mechanism to explain these results is unknown. More studies are necessary to confirm our results. In that case, it could be useful to change dietetic recommendations in case of obesity, because nowadays the general recommendation is to reduce general fat ingestion.

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DISCLOSURE

The authors declare no conflict of interests regarding the publication of this paper.

AUTHOR CONTRIBUTIONS

Beatriz Moreno-Pérez, Esther Benito and Miguel Civera were involved in patient recruitment and reviewing the manuscript. Blanca Alabadi and Marta Peiro were involved in data collection and reviewing the manuscript. Sergio Martinez-Hervas was involved in conducting the study, data analysis, writing the manuscript and reviewing the manuscript. Herminia González-Navarro, Laura Piqueras and Maria Jesús Sanz were involved in biochemical analysis and reviewing the manuscript. Juan F Ascaso was involved in design and reviewing the manuscript. Jose T. Real was involved in design, conducting the study, writing the manuscript and reviewing the manuscript.

DATA AVAILABILITY STATEMENT

The datasets used and analysed during the current study cannot be public available because of the individual privacy of the subjects included in the study. However, the data generated and included in the current study are available from the corresponding author upon reasonable request.

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