nutrición clínica y Dietética Hospitalaria

Artículo Original

Nutr. clín. diet. hosp. 2019; 39(2):97-103
DOI: 10.12873/392muñoz

Postprandial lipid profile in young colombian people. A comparison of two breakfasts

Muñoz Perez, Diana María[1]; Giraldo Guzmán, Cristian[1]; Astudillo Muñoz, Elcy Yaned[2]; Castañeda Gallon, Manuela[1]; González Correa, Clara Helena[1]

1 Nutrition, Metabolism and Food Safety Research Group, Universidad de Caldas. Colombia.
2 Universidad Libre Pereira. Colombia.

Recibido: 6/marzo/2019. Aceptado: 30/junio/2019.

ABSTRACT

The aim was to compare the postprandial lipid profile of university students who ate a regular breakfast of the Colombian Andean region, high in saturated fats and low in complex carbohydrates, compared with an experimental breakfast with low fat content and high in complex carbohydrates and its relation with anthropometric measurements. 75 university students consumed one of the two breakfasts after a 12-hour fast. A complete lipid profile was performed in a fasted state, three and six hours after breakfast ingestion. Of the 75 patients, 11 were withdrawn, 28 people consumed the experimental breakfast and 36 the usual one. There was no significant difference between the two groups; however there was a tendency to decrease the levels of all the components of the lipid profile in the experimental breakfast, except for High density lipoprotein (HDL). The Area under the Curve (AUC) did not show differences between breakfasts. The body mass index (BMI) and the waist / hip ratio (WHR) showed an inverse relationship with HDL and a direct relationship with Low density lipoproteins (LDL). In conclusion, there were not differences in the acute effect of both breakfasts, possibly due to factors such as smoking, exercise, sedentary lifestyle, type of food used in the diet, variables that were not discriminated in this study. The main contribution of this study is the description of the behavior over time of the lipid profile variables and their relationship with the anthropometric variables. It is possible that the effect of these diets is likely to be significant in the long term.

Correspondencia:
Diana María Muñoz Pérez
diana.2291424565@ucaldas.edu.co

KEYWORDS

Lipids, blood, postprandial, profile, healthy people.

LIST OF ABBREVIATIONS

BMI: Body mass index.

AUC: Area under the Curve.

WHR: Waist / hip ratio.

LDL: Low density lipoproteins.

Non-HDL: Non-high density lipoproteins.

HDL: High density lipoprotein.

TG: Triglyceride.

VLDL: Very low density lipoprotein.

apoA, apoB: Apoproteins A, B.

TC: Total Cholesterol.

INTRODUCTION

In recent decades the term postprandial lipemia has been used to represent the variation of triglycerides after the absorption of a high-fat meal[1]. In addition, the postprandial lipid profile allows to measure the different lipoprotein fractions after ingestion and absorption of food[2]. Studies show that a postprandial lipid profile provides more significant information compared with a fasting lipid profile, since in the postprandial state both hepatic and intestinal lipoproteins are found[2]. It has been shown that a fat-rich meal consumes 30 to 60 grams of lipids, after which the triglycerides in a healthy person show an elevation in the first 1-2 hours. Subsequently, a maximum peak between the 3rd and 4th hour is obtained with a return to the basal state between

the 6th and the 8th hour[3,4]. Very low density lipoproteins, or VLDL, increase after ingestion and remain elevated in blood around 3.6 hour[5]. In contrast, there may be a slight decrease in the low density lipoprotein (LDL), non-high density lipoprotein (NonHDL) and total cholesterol, especially in the first 4 hours after consuming the food but this has been associated with the intake of water that dilutes the blood components[6]. Postprandial lipemia is determined by the levels of preprandial triglycerides and also by the quality and quantity of lipids ingested[7,8]. A meal high in polyunsaturated fatty acids, generates a lower lipemia compared to an intake of saturated and monounsaturated fatty acids[8]. It is therefore the elevation of triglycerides and their respective transport lipoproteins (chylomicrons and VLDL), independent of other lipids, which determine postprandial lipemia as a risk factor for the generation of atherosclerosis, and the prediction of acute myocardial infarction[9,10].

The average person spends around 16 hours in the postprandial state and a high lipemia in this period can generate a decrease in high density lipoprotein (HDL) cholesterol, promote the accumulation of LDL, and generate pro-inflammatory and prothrombotic states[11]. People with metabolic syndrome have clearance of lipoproteins is diminished, In this situation, the probability that these begin to generate atherosclerotic processes, is greater because triglycerides begin to replace LDL in this physiopathological process[9]. In contrast, if we could develop better diets, implement them in healthy subjects, we could decrease not only the maximum peaks of lipemia, but the acceleration of the atherosclerotic disease. In this way you could have a valuable tool for the prevention of cardiovascular disease.

That is why the objective of this study was to compare the postprandial lipid profile of university students who ate a regular breakfast from the Andean region of Colombia, rich in saturated fats and low in complex carbohydrates, with the postprandial profile of young people who ate a breakfast with low fat content and rich in complex carbohydrates, in order to analyze the behavior of the different components of the lipid profile.

MATERIALS AND METHODS

Ethical Declarations

The experimental protocol was approved by the ethics committee of the Universidad de Caldas and was developed in accordance with the declaration of Helsinki (1964), revised in Tokyo (1975), Venice (1983) and Hong Kong (1989). In addition, Colombian resolution No. 008430 of October 4, 1993, which establishes the norms for health research, was taken into account. The proposal was classified in the research category with minimal risk. Before initiating the study, each participant signed an informed consent.

Subjects

The participants were volunteers, from the Universidad de Caldas and the Universidad Libre of Pereira, Colombia between the months of July to December 2016. They included university students between 18 and 35 years who were not consuming lipid-lowering drugs or had diagnoses of dyslipidemia, hyper or hypothyroidism, obesity or other chronic diseases. We also excluded people who in fasting had a triglyceride level greater than 150 mg / dl, or were women in pregnancy or lactation.

Study design

This is an experimental, parallel, randomized, controlled, single-blind study with convenience sampling. The participants went to the respective universities, after a 12-hour fast and were randomly assigned to consume one of two types of isocaloric breakfast (579.5 Kc). The experimental breakfast included foods with a high content of complex carbohydrates and low saturated fats. The usual breakfast was high in saturated fat and low in complex carbohydrates. The foods for each breakfast are listed below.

Experimental Breakfast: 100 g bananas, low fat milk (1%) 200 ml, fresh cheese 30 g, corn arepa 30 g, oat flakes 40 g, walnuts 10 g, peanut 10 g, panela 5 g, olive oil 3 ml.

Usual Breakfast: white sugar 10 g, whole milk (3.3%) 120 ml, scrambled egg 100 g, salt 1 g, butter 10 g, coffee 3 g, white bread 56 g, sausage 30 g. (Table 1) describes the percentage of adequacy of the macronutrients and the grams of the different types of lipids obtained through the software Nutritionist Pro®, licensed to the Universidad de Caldas.

Through interrogation, it was found that each student had complied with a 12-hour fast after which a first blood sample was collected. A second and third samples were obtained at 3 and 6 postprandial hours. In all three samples the lipid pro-

Table 1. Percentage adequacy of the macronutrients and type of lipids used in the diets.

Experimental Breakfast		Usual Breakfast	
Carbohydrates	56,1%	Carbohydrates	31,5%
Proteins	14,5%	Proteins	17,7%
Fat	29,4%	Fat	50.8%
MUFA	6,4 g	MUFA	11,4 g
PUFA	7.1 g	PUFA	3,8 g
SAFA	3.5 g	SAFA	13,9 g

MUFA - Monounsaturated fatty acids; PUFA - Polyunsaturated Fatty Acids; SAFA - Saturated Fatty Acids.

file was measured (LDL, HDL, total cholesterol, TG, VLDL and NonHDL).

Anthropometric measurements

Weight, height, abdominal and hip perimeters, and subcutaneous fat folds were measured according to the protocol established with Lohman *et al* 1988. With these variables were calculated the BMI, the waist / hip index and the percentage of fat bodily.

Lipid profile and laboratory tests

The samples were collected in vacuum tubes. The serum, separated from the erythrocytes by centrifugation (3500 RPM x 15 minutes at 4 ^{0}C) was stored at -80 ^{0}C for further analysis of the lipid profile. Total cholesterol, TG, LDL and HDL were analyzed in a COBAS 6000 from Roche. Non HDL cholesterol was obtained by subtracting HDL from Total cholesterol and VLDL was calculated with the TG / 5 ratio.

Statistic analysis

The analysis of the data was made with the statistical software SPSS® version 24.0, licensed to the Universidad de Caldas. The sample size calculation was obtained taking into account an alpha error of 5% and a beta error of 20%. The result indicated that a minimum of 10 individuals per group was required to obtain a power greater than 90%. All the descriptive statistics presented were expressed as mean +/- standard deviation (Table 2).

The normality or not of the anthropometric data was determined by means of the Shapiro-Wilk test. Subsequently, the Student's T test or the Mann-Whitney U test were applied for parametric or nonparametric data, respectively, in order to determine the differences between the two groups. In addition, a matrix of bivariate correlations was developed to investigate the relationship of these variables with laboratory tests.

Likewise, the normality of all the laboratory variables was verified: lipid profile (total cholesterol, LDL, Non HDL, HDL, triglycerides and VLDL) with the Shapiro-Wilk test. To verify that both groups entered the same conditions, the Student t test was applied to the fasting samples (Hour 0) of each variable. After each variable was applied an Analysis of variance for repeated measures (ANOVA), in order to establish the change in time (Hours 3 and 6) of each of the breakfast groups in the postprandial period.

In addition to the postprandial lipids that were measured at each point of time (0, 3 and 6 hours) the area under the

Table 2. Initial conditions of both groups according to breakfast.

	Units	Experimental Breakfast				Usual Breakfast				
		Mean +/- SD	Minimum	Maximum	CI 95%	Mean +/- SD	Minimum	Maximum	CI 95%	p value
n		**28**				**36**				
Age	**Years**	21,8 +/- 3	19	29	20,62 : 23,02	20,7 +/- 2,87	18	32	19,7 : 21,6	0,06
Weight	**kg**	62,5 +/- 8,3	45,6	77,2	59,2 : 65,7	61,1 +/- 11,6	41,4	83,9	57,1 : 65,06	0,59
Size	**m**	1,67 +/- 0,09	1,47	1,84	1,63 : 1,71	1,65 +/- 0,9	1,48	1,85	1,62 : 1,68	0,39
BMI	**kg/m^2**	22,33 +/- 2,6	16,6	28,2	21,3 : 23,3	22,3 +/- 3,37	16,04	29,6	21,1: 23,4	0,96
Fat	**%**	23,74 +/- 7,3	11,3	36,9	20,9 : 26,5	25,9 +/- 6,3	10,8	38,03	23,7 : 28,09	0,205
WHR		1,01 +/- 0,37	0,51	1,96	0,87 : 1,16	0,85 +/- 0,30	0,55	1,75	0,75 : 0,96	0,05
cTotal	**mg/dl**	164,2 +/- 23,5	119	217	155,07 : 173, 3	172,5 +/- 17,5	144	205	166,5:178,4	0,11
cHDL	**mg/dl**	49,4 +/- 10,9	32,8	76	45,2 : 53,7	49,9 +/- 10,6	28,1	74,6	46,2 : 53,5	0,88
cLDL	**mg/dl**	99,7 +/- 24,9	51,1	144	90,06 : 109,4	109,8 +/- 22,6	67,4	155,5	102,2: 117,5	0,09
cVLDL	**mg/dl**	15,9 +/- 5,3	7,6	25,4	13,9 : 18,03	16,05 +/- 4,1	7,6	26,6	14,6 : 17,4	0,94
cNoHDL	**mg/dl**	112,07 +/-22,6	64,8	156,5	103,2 : 120,8	112,7+/- 22,1	81	165,3	115,2 : 130,2	0,06
Tg	**mg/dl**	79,9 +/- 26,5	38	127	69,6 : 90,1	80,2 +/- 20,9	38	133	73,2 : 87,3	0,94

SD - standard deviation; BMI - body mass index; WHR - Waist / hip Ratio; CI – confidence interva.

curve (AUC) was measured through the trapezoidal method. The effect of the breakfasts on the AUC was compared with the Student t test for independent samples. A statistically significant p value of less than 0.05 was considered for all tests ($p < 0.05$).

RESULTS

Of the 75 initial participants, 3 withdrew during the course of the study and 8 because they had fasting triglycerides greater than 150 mg / dl. With the 64 resulting participants two groups were created, group 1 that received the experimental breakfast (Breakfast 1) and was composed of 28 people (14 men and 14 women), and group 2 that received the usual breakfast of the Andean region (Breakfast 2), had 36 people (13 men and 23 women). The variables size, weight, BMI and percentage % of fat presented a normal distribution, and no statistically significant difference was found between the two groups. The variables age and waist hip index were not normally distributed. There was no difference by age between both groups. The waist-hip index showed a marginal difference ($p = 0.050$) between the two groups (Table 3).

The laboratory tests taken on fasting showed a normal distribution and no significant differences were found in any of the lipid profile variables between the two groups at the beginning of the study (Table 2). In the ANOVA, there was a tendency to decrease total cholesterol, LDL, Non HDL, TG and VLDL in the group with the experimental breakfast of the Andean region compared with the usual breakfast of the Andean region, although it was not statistically significant. This trend was not observed for HDL.

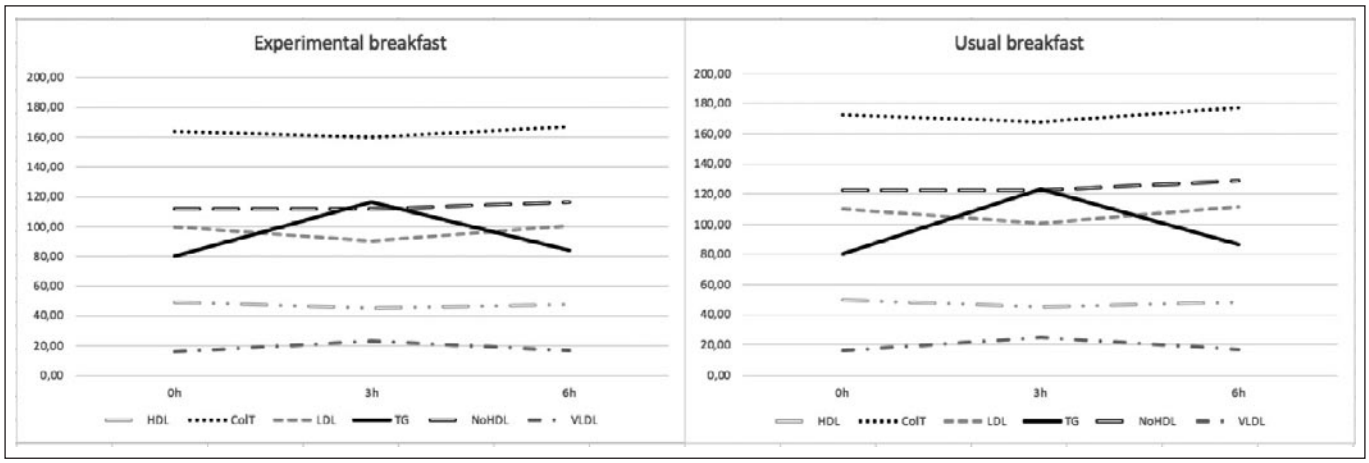
Figure 1 (A and B): shows the behavior over time of the levels of the laboratory variables of both breakfasts. There is a marked increase in TG and VLDL at 3 hours, with a decrease in baseline at 6 hours. With a difference in the average between breakfast (usual breakfast - experimental breakfast) TG (7.07 mg / dl (3h) and 2.45 mg / dl (6h)); VLDL (1.41 mg / dl (3h) and 0.49 mg / dl (6h)). On the contrary, a decrease in LDL, ColT and HDL was observed after 3 hours, with rise of them after 6 hours of breakfast. With a difference in the average between LDL breakfasts (10.30 mg / dl (3h) and 11.56 mg / dl (6h)); ColT (7.78 mg / dl (3h) and 10.26 mg / dl (6h)); HDL (0.08 mg / dl (3h) and 0.97 mg / dl (6h)). Particularly the No HDL shows a constant behavior in the 3 hours and then rise to 6 hours, with an average difference between breakfast of 10.71 mg / dl (3h) and 12.71 mg / dl (6h). No significant difference was observed in the area under the curve (AUC) of any of the lipid profile variables between the two groups.

Table 3. Anthropometric and laboratory variables according to sex at the beginning of the study.

	Units	Experimental Breakfast		Usual Breakfast	
		Men	Women	Men	Women
n		14	14	13	23
Age	**Years**	21,8 +/- 2,9	21,7 +/- 3,3	21,6 +/- 4	20,2 +/- 1,8
Weight	**kg**	66,7 +/- 6,2	58,2 +/- 8,2	70,2 +/- 11,8	55,95 +/- 7,8
Size	**m**	1,75 +/- 0,4	1,59 +/- 0,05	1,75 +/- 0,06	1,59 +/- 0,5
BMI	**kg/m²**	21,8 +/- 2,1	22,8 +/- 2,9	22,8 +/- 3,4	21,9 +/- 3,3
Fat	**%**	17,2 +/- 3,7	30,2. +/- 2,5	19,7 +/- 4,1	29,4 +/- 4,4
WHR		1,3 +/- 0,3	0,72 +/- 0,12	1,14 +/- 0,31	0,69 +/- 0,11
cTotal	**mg/dl**	155,05 +/- 15,5	173,3 +/- 27,01	172,9 +/- 16,03	172,3 +/- 18,7
cHDL	**mg/dl**	45,5 +/- 8,5	53,4 +/- 11,9	44,7 +/- 8,8	52,8 +/- 10,6
cLDL	**mg/dl**	93,9 +/- 19,9	105,4 +/- 28,6	114,6 +/- 24,5	107,2 +/- 21,5
cVLDL	**mg/dl**	17,1 +/- 4,05	14,8 +/- 6,2	18,3 +/- 3,9	14,7 +/- 3,8
cNoHDL	**mg/dl**	105,7 +/- 20	118,3 +/- 24,1	131,8 +/- 20,2	117,6 +/- 21,9
Tg	**mg/dl**	85,6 +/- 20, 2	74,1 +/- 31,2	91,7 +/- 19,6	73,8 +/- 19

BMI - Body mass index; WHR -waist / hip ratio.

Figure 1. It shows the behavior over time of each one of the lipid profile variables for both breakfasts.



In the correlation matrix, an inverse relationship was found between BMI and HDL (p = 0.049) and its respective AUC (p = 0.048), while the relationship was direct with total cholesterol (p = 0.009), LDL (p = 0.005), NoHDL (p = 0.005) and their respective AUC (p = 0.003), (p = 0.008), (P = 0.002). Likewise, the waist / hip index showed an inverse relationship with HDL (p = 0.012) and its AUC (p = 0.025) and a direct relation with fasting total cholesterol (p = 0.029). The percentage of fat had a direct relationship with fasting total cholesterol (p = 0.007) and its AUC (p = 0.02).

DISCUSSION

The results obtained in the study showed the change in time in each of the variables, especially in the TG. There was no significant difference between breakfasts in relation to the levels of the different components of the lipid profile. However, in the group that consumed the experimental breakfast there was a slight tendency to decrease circulating lipids (TG, VLDL, LDL, Non HDL, ColT) except HDL, as expected. This result may be due to the type of products used for the design of the diet, since the experimental breakfast, although it had complex carbohydrates, high PUFA and low SAFA, had less MUFA, important fatty acids in a healthy diet. The omega 3 fatty acids were not taken into account as well as Song *et al* [13]. Song and collaborators who in a study with 16 people compared 8 people with hypertriglyceridemia and 8 healthy people, with an average age of 53 and 46 years respectively, to whom one of two diets was applied, both with a PUFA / MUFA / SAFA ratio 1/1/1 but with different composition of fatty acids, one with high omega 3 and the other with low omega 3; found that in healthy people, TG had a maximum peak at 4 hours and a return to baseline at 8 hours, with an area under the incremental curve for lower TG in diets with omega 3 fatty acids, however, in the present study was carried out in young people, with an average age of 21 years, and with a much larger sample.

The polyphenol levels, which are important, were not taken into account in the study, since they influence the metabolism of the postprandial lipids, as they showed Annuzzi *et al*[14]. These authors evaluated, in people with signs of the metabolic syndrome, the response to different diets with omega 3 fatty acids and polyphenols. The authors found a significant decrease in the AUC of triglycerides and VLDL with diets rich in polyphenols. They also found that the isolated effect of them, by itself, was statistically significant. However, this was an 8-week intervention and did not evaluate the acute effect of them.

Few studies have evaluated the postprandial change in healthy people. In this study, samples were taken on fasting, at the third hour and at the sixth hour, as Sierra *et al* did[3] in a study in Colombia. They described that, in healthy people, it is in the third hour when the maximum triglyceride peak occurs and in the sixth hour it returns to its basal level. However, they did not take into account the behavior over time of the other components of the lipid profile.

The results obtained for triglyceride transporting lipoproteins (TG and VLDL) and total Cholesterol lipoproteins (LDL, HDL and ColT) were opposite. This could be because triglycerides are lipids of the exogenous diet and the first ones to be metabolized in the organism, after the metabolism of the chylomicrons, so they increase rapidly in the first three hours[15]. The lipoproteins cholesterol transporters, triglycerides and phospholipids, are manufactured mainly in the liver, from the proteins apoA and apoB in a second memento. These apoproteins have the function of carrying each of these lipids to be processed in the tissues in which they are metabolically active. Its subfractions have the ability to transport them differentially. This is how HDL3 are dense particles that are enriched with free cholesterol and phospholipids, while HDL2, which are less dense and relatively rich in proteins, are enriched with cholesterol esters and small amounts of triglycerides[16,17]. In the present study no quantifications of subfractions were made, which may be desirable in a future study.

The area under the curve evaluated the behavior over time of the different components of the lipid profile and no significant changes were found between breakfasts. A similar result was reported by Días *et al*[18] who bought the acute effect of a diet rich in saturated fats and a diet rich in omega-6 polyunsaturated fatty acids. These investigators did not observe significant differences in the iAUC (area under the incremental curve) for plasma TG, total cholesterol, LDL or HDL. From the statistical analysis of bivariate correlations, it was found that both the BMI and the waist / hip index presented an inverse relationship with the fasting HDL. There was also a direct relationship between BMI and LDL, total cholesterol and Non HDL. This indicates that a greater proportion of fat accumulated at the abdominal level, and the excessive consumption of carbohydrates and fats can reduce the production of HDL, since these transport lipids from the peripheral tissues to be metabolized by the liver. It also increases LDL, which are lipoproteins necessary to transport cholesterol to peripheral tissues[9]. Additionally, in this way, the direct relationship of fat percentage and hip waist index with fasting total cholesterol is explained. Similar results found by Navarrete *et al*[19] who described in a study with 3016 young participants, a direct association of BMI with cholesterol and triglycerides, as well as an inverse relationship with HDL levels, all statistically significant.

In the present study, there was no discrimination between smokers and non-smokers, which could be a factor of error for the study, since Sierra *et al*[20] demonstrated that the AUC of triglycerides was 21% higher in smokers, this could be key to explain our results.

This study has some limitations, this is how non-significant results can also be explained by the non-discrimination between athletes and sedentary people. The previous feeding of the members of each group was not evaluated either, since the postprandial metabolism usually adapts to the lifestyle and to the lipid load received daily[9]. Another important factor is the kind of food used in breakfasts, as these not only contain carbohydrates or fats, but also different concentrations of proteins and polyphenols, which interact in the postprandial metabolism[14]. There are also no known studies of the genetics of our population, which can also be a factor of confusion that influences the results found. On the other hand, the strengths of this study have to do with strict supervision and the rigor with which the study protocol was carried out. Additionally, the number of patients achieved an adequate adherence, with a loss of only 2% of the participants.

CONCLUSIONS

It was not possible to demonstrate that a diet rich in complex carbohydrates and mono and polyunsaturated fats reduced postprandial lipemia acutely compared to a diet high in saturated fatty acids and simple carbohydrates. Additional studies evaluating diets rich in these components are needed not only acutely but with a strict follow-up over time, as it would be expected that there would be a significant decrease in long-term interventions with a larger sample size.

ACKNOWLEDGMENT

Thanks to Universidad de Caldas for their financial support, to the volunteers who participated in the study and to Professor Carmen Dussán Luberth for her advice in the area of statistics.

REFERENCES

1. Alcala-Diaz JF, Delgado-Lista J, Perez-Martinez P, Garcia-Rios A, Marin C, Quintana-Navarro GM, et al. Hypertriglyceridemia influences the degree of postprandial lipemic response in patients with metabolic syndrome and coronary artery disease: from the CORDIOPREV study. PloS one. 2014;9(5):e96297.
2. Nordestgaard BG. A test in context: lipid profile, fasting versus nonfasting. *J Am Coll Cardiol*. 2017;70(13):1637-46.
3. Sierra ID, Pérez C, Mendivil O, Pinzón G, Mantilla G, Gámez A, et al. Evaluación del comportamiento del perfil de lípidos postingesta de una carga lipídica estandarizada en jóvenes sanos, Universidad Nacional de Colombia, 2000. *Rev Fac Med*. 2001; 49(3):141-6.
4. Jiménez-Gómez Y, Marín C, Peérez-Martínez P, Hartwich J, Malczewska-Malec M, Golabek I, et al. A low-fat, high-complex carbohydrate diet supplemented with long-chain (n-3) fatty acids alters the postprandial lipoprotein profile in patients with metabolic syndrome. *J Nutr*. 2010;140(9):1595-601.
5. Pérez CE, Mendivil CO, Sierra ID. Influencia de variables clínicas y lipídicas en la magnitud de la lipemia postpradial de sujetos con y sin hipertrigliceridemia. *Rev Fac Med*. 2007;55(1):4-13.
6. Nordestgaard BG, Langsted A, Mora S, Kolovou G, Baum H, Bruckert E, et al. Fasting Is Not Routinely Required for Determination of a Lipid Profile: Clinical and Laboratory Implications Including Flagging at Desirable Concentration Cutpoints-A Joint Consensus Statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine. *Clin Chem*. 2016;62(7):930-46.
7. Alipour A, Elte J, van Zaanen H, Rietveld AP, Cabezas MC. Novel aspects of postprandial lipemia in relation to atherosclerosis. *Atheroscler Suppl*. 2008;9(2):39-44.
8. Jackson KG, Poppitt SD, Minihane AM. Postprandial lipemia and cardiovascular disease risk: Interrelationships between dietary, physiological and genetic determinants. *Atherosclerosis*. 2012; 220(1):22-33.
9. Pirillo A, Norata GD, Catapano AL. Postprandial lipemia as a cardiometabolic risk factor. *Curr Med Res Opin*. 2014;30(8):1489-503.
10. Devaraj S, Wang-Polagruto J, Polagruto J, Keen CL, Jialal I. High-fat, energy-dense, fast-food–style breakfast results in an increase in oxidative stress in metabolic syndrome. Metabolism. 2008; 57(6):867-70.

11. Karupaiah T, Sundram K. Modulation of human postprandial lipemia by changing ratios of polyunsaturated to saturated (P/S) fatty acid content of blended dietary fats: a cross-over design with repeated measures. *Nutr J*. 2013;12(1):122.

12. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual: Human kinetics books; 1988.

13. Song Z, Yang L, Shu G, Lu H, Sun G. Effects of the n-6/n-3 polyunsaturated fatty acids ratio on postprandial metabolism in hypertriacylglycerolemia patients. *Lipids Health Dis*. 2013;12(1): 181.

14. Annuzzi G, Bozzetto L, Costabile G, Giacco R, Mangione A, Anniballi G, et al. Diets naturally rich in polyphenols improve fasting and postprandial dyslipidemia and reduce oxidative stress: a randomized controlled trial. *Am J Clin Nutr*. 2013;99(3):463-71.

15. Brunzell JD, Davidson M, Furberg CD, Goldberg RB, Howard BV, Stein JH, et al. Lipoprotein management in patients with cardiometabolic risk: consensus conference report from the American Diabetes Association and the American College of Cardiology Foundation. *J Am Coll Cardiol*. 2008;51(15):1512-24.

16. Nieto Cárdenas OA, García Cardona DM, Jiménez JA, Landázuri P. Efecto del ejercicio en subpoblaciones de lipoproteínas de alta densidad y en la presión arterial. *Rev. salud pública.* 2013;15, (1); 12-2.

17. Gidez LI, Miller GJ, Burstein M, Slagle S, Eder HA. Separation and quantitation of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. *J Lipid Res*. 1982; 23(8):1206-23.

18. Dias CB, Phang M, Wood LG, Garg ML. Postprandial lipid responses do not differ following consumption of butter or vegetable oil when consumed with omega-3 polyunsaturated fatty acids. *Lipids*. 2015;50(4):339-47.

20. Navarrete Mejía PJ, Alarico L, Jesús M, Velasco Guerrero JC, Huatuco Collantes ZA, Abregú Meza RA. Índice de masa corporal y niveles séricos de lípidos. *Horiz. Med*. 2016;16(2):13-8.

21. Sierra ID, Mendivil O, Pérez C, Pinzón AG, Mantilla G, Gámez Á, et al. Hábito de fumar y respuesta postprandial a una carga lipídicaestandarizada en jóvenes fumadores de la Universidad Nacional de Colombia, 2000. *Rev Fac Med*. 2001;49(1):251-6.