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Randomized Controlled Trial

Fasted exercise does not improve postprandial lipemia responses to different meals in lean and obese subjects: A crossover, randomized clinical trial

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SUMMARY

Introduction: Persistent episodes of postprandial hyperlipemia (PPL) and hyperglycemia (PPG) are considered risk factors for coronary heart disease (CHD) and premature death; whereas physical exercise improves lipid profile and glucose tolerance thus decreasing cardiovascular risks.
Objective: To investigate the effects of low-intensity fasted aerobic exercise on the magnitude of the PPL and PPG responses to meals with different energy content, in normal and obese subjects.
Methods: The study used a randomized crossover design. Twenty-one male (Lean: $n = 9$, BMI: 24.3 ± 2.2; and obese: $n = 12$, BMI 32.31 ± 2.1) volunteers aged 20–30 years, performed three interventions, separated by 7 days each: (i) 45 min at rest and isocaloric high-fat meal (60% lipids, 30% carbohydrates and 10% protein); (ii) fasted low-intensity aerobic exercise (50% VO_{2max}) for 45 min followed by an isocaloric or (iii) calorie deficit high-fat meal. Subjects were serially assessed for blood triglycerides, and glucose levels.
Results and conclusions: Low-intensity fasted aerobic exercise had no acute effect on PPL in lean and obese subjects. Glucose concentrations were reduced only in lean subjects. There is a significant difference in PPL values when comparing lean to obese subjects, implying that the nutritional status influences lipid and carbohydrate after fasted low-intensity aerobic exercise. Registered under ClinicalTrials.gov Identifier no. NCT00929890.

1. Introduction

Obesity and adiposity may cause profound metabolic alterations, including glucose and lipid metabolism thereby increasing the risk of coronary heart disease (CHD), hypertension, type 2 diabetes mellitus (T2DM), and some types of cancer [1–3]. In contrast, a physically active lifestyle and low-calorie energy intake are feasible and low-cost strategies for the prevention and management of obesity, dyslipidemia and other comorbidities [4].

Postprandial lipemia (PPL) is a condition in which triglycerides-rich chylomicron remnants are increased during the postprandial period and hypertriglyceridemia is prolonged, which has been suggested as a better metabolic biomarker of cardiovascular risk compared to fasting triglycerides (TG) [5]. High-fat diets exposes the vasculature to a marked increase in the production of proinflammatory cytokines, recruitment of neutrophils, and generation of oxidative stress damage [6–8], which are mechanistic contributors of the atherogenesis process [9,10]. Similarly, unhealthy diets resulting in persistent increments of blood glucose potentiating Postprandial Glucose (PPG) may activate toxic signaling cascades, which lead to cell damage and loss of function of several biological systems including the heart, peripheral nerves, and brain [11,12].

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It has long been known that acute exercise improves PPL after high-fat meal. These effects have been attributed to the increased TG mobilization and fatty acid oxidation mediated by catecholamines [13,14], the activity of lipase lipoprotein enzyme (LPL), and to the reduction of VLDL in the liver [15,16]. Since 1990s, a growing body of evidence has focused if exercise timing may influence postprandial metabolism and health [17]. In this way, fasted exercise has gained popularity as a strategy to increase fat oxidation and reduce fasting and postprandial lipemia [18]. However, nutritional status may affect fasted exercise-induced decreased on PPL, since there is an effect on lean but not on obese individuals [19–21]. Nevertheless, a plethora of current and classic studies demonstrate that subjects may vary in responsiveness to the same strategy involving exercise, diet, and other risk management components related to obesity [22–26].

Strikingly, exercise intensity and timing exerts a determinant role in decreasing PPL, and obese subjects could benefit from lower total energy expenditure to reduce PPL compared to lean subjects [27,28], albeit the magnitude of specific individual response in both lean and obese populations deserves scrutiny. Therefore, this study was designed to assess the acute effects of a low-intensity fasted exercise on PPL and PPG in untrained lean and obese subjects submitted to high-fat meals with different energy contents.

2. Methods

2.1. Subjects

This study is characterized as a randomized crossover trial (see flowchart, supplemental file). The sample size was calculated using Epi Info version 3.5.2 for a 95% confidence and 80% power, using TG area under the curve [16,19,21]. For this effect, we used 12 subjects per group. A total of twenty-one ($n = 21$) untrained men participated in this study, distributed in two experimental groups: Lean (body mass index between 20 kg/m^2 and 24.9 kg/m^2, and fat percentage under 30%) and Obese (body mass index between 30 kg/m^2 and 34.9 kg/m^2 and a fat percentage higher than 30%). In addition, all subjects were aged between 20 and 30 years and were not allowed to participate if they take part in exercise training programs and/or dietary intervention programs in the last six months. Also, subjects were excluded when at least one of the following conditions were present: smokers, dyslipidemic; using or used appetite suppressants or lipid-lowering drugs within the last 6 months prior to the study; alcoholism; diabetes mellitus; hypertension or chronic disease impairing the performance of aerobic exercise including angina, myocardial infarction within the previous 6 months, congestive heart failure, chronic obstructive pulmonary disease, uncontrolled asthma, cancer chemotherapy or radiotherapy, or any other chronic condition or medication were recommended do not participate in the study. Written informed consent was obtained from all participants. This study was approved by the ethics committee of research of Porto Alegre Clinicas Hospital (HCPA) under the number 110–649 and was conducted in accordance with the provisions of the Declaration of Helsinki. This study is part of a larger effort ("Additional Metabolic and Vascular Effects of Exercise in Patients on diet-based weight-loss programs"), registered in clinical-trials under number NCT00929890, Unique Protocol ID 08282.

2.2. Experimental design and protocols

Eligible Individuals were assessed for basal metabolic rate (BMR), body weight, height, body mass index (BMI), skinfold thickness, blood glucose (non-fasting), physical activity (Par-Q and You questionnaire), maximal and submaximal exercise test on a cycle ergometer with direct analysis of gases to determine the maximal oxygen consumption (VO_{2max}). Individuals who met the inclusion criteria were randomly assigned following simple randomization procedures (computerized random numbers) to different orders of three experimental protocols, using the tool available at www.randomization.com.

Volunteers performed the three protocols in a randomized order, separated an interval of 7 days. Subjects arrived at the laboratory at 07:30 h after carrying out 12 h of fasting and were assessed for basal metabolic rate and fasted blood collection. Subsequentially, subjects performed different protocols: a session of 45 min of rest in a seated position for the control protocol (CP), or a 45 min low-intensity (50% VO_{2max}) cycle ergometer exercise protocol for both caloric deficit (CDEF) or isocaloric protocols (ISO).

Afterward, subjects consumed a high-fat meal with different caloric values: equal to individual BMR coupled with 45 min of rest for CP, equal to individual BMR coupled with the energy expenditure of 45 min of exercise for ISO, and equal to individual BMR without considering energy expenditure of 45 min of exercise for CDEF. The high-fat meal was composed of 60% lipids, 30% carbohydrates, and 10% protein, and consisted of a paste-like drink made from skim milk, ice cream, and cream. Subjects had 10 min maximum to consume the meal. Water was available *ad libitum*. After the meal, blood samples were collected in the first hour and every 30 min, totaling 8 samples, 10 ml per tube. The timeline of the procedures is presented in Fig. 1.

2.3. Body composition

Body composition was calculated according to the five component methodology [29], and the data was collected by an experienced level III evaluator following the recommendations of the International Society for the Advancement of Kinanthropometry (ISAK). The skinfolds were measured using a caliper (Harpenden scientific model, Cescorf, Porto Alegre, Brazil), bone diameters by caliper and anthropometer (Cescorf, Porto Alegre, Brazil), girth measured using measuring tape (Sanny, São Bernardo do Campo, São Paulo), weight and height were measured by scale and stadiometer (Uranus, ref. OS-180, RS/Brazil). Body mass index was calculated by dividing the mass of the individual by the squared height.

2.4. Dietary control

All subjects were instructed to avoid alcoholic beverages and/or caffeine containing products for at least 48 h before the protocols. In the preliminary visit, subjects were individually instructed to fill two forms for 24 h dietary recalls (for two-days during the week), which were returned to the nutritionist for diet composition analysis. Each participant recorded all food and beverages consumed in the previous days before the protocols. Twenty-four hours prior the protocols, subjects repeated the same food consumption described on the first record. Data analysis used *Dietwin*® (Brubins) software, Professional version (2008).

2.5. Basal metabolic rate (BMR) and exercise energy expenditure assessment

Subjects were instructed to avoid performing physical activities on the day previous the test and to complete a 12 h fasting period with at least 8 h of sleep during the night, not drinking alcohol, caffeine or any kind of medication during this period without prior notice to the research team. The consumption of water was *ad libitum*. All BMR tests were performed between 07:30 and 08:30 h a.m. in a room temperature between 20 °C and 25 °C, with

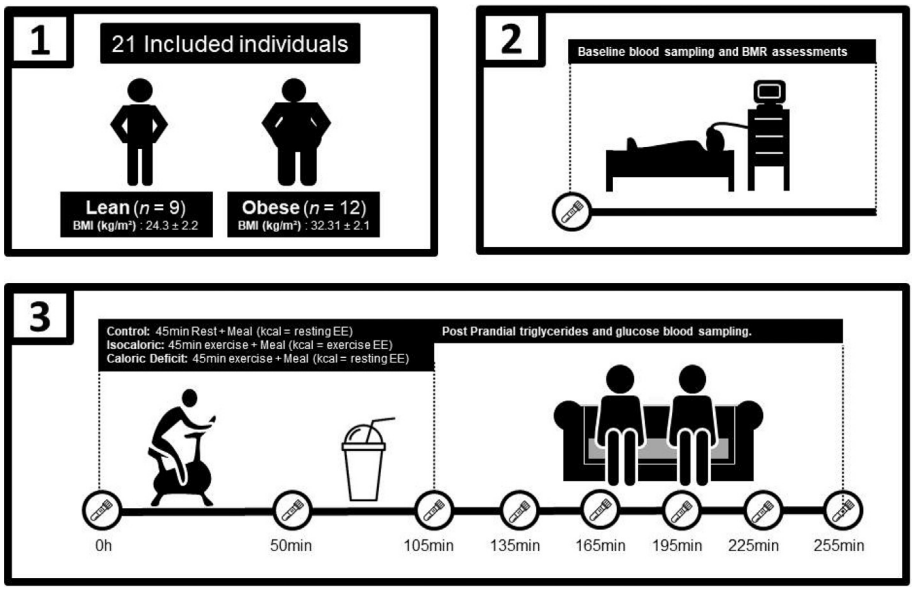

Fig. 1. A total of 21 Eligible Individuals (1 and 2) were assessed for basal metabolic rate (BMR), anthropometric measures, blood glucose (non-fasting), and maximal oxygen consumption (VO_{2max}) test on a cycle ergometer. Individuals were randomly assigned to different orders of three experimental protocols: a session of 45 min of rest in a seated position for the control protocol (CP), or a 45 min low-intensity (50% VO_{2max}) cycle ergometer exercise protocol for both caloric deficit (CDEF) or isocaloric protocols (ISO). Afterward, subjects consumed a high-fat meal with different caloric values. A total of 8 blood samples (Before the protocol, before the meal, 1 h after the meal, and 5 subsequent samples; 10 mL each) were obtained, and the total sampling time was 255 min.

controlled noise and low light. The protocol consisted of 10 min of rest on a stretcher in the supine position, followed by 30 min of gas capture. To determine VO_2 and VCO_2 values a gas analyzer was used (MedGraphics Cardiorespiratory Diagnostic Systems, CPX-D model) [30]. To calculate the BMR, the first 10 min of uptake were discarded and the mean values of VO_2 and VCO_2 ($L.min^{-1}$) of the subsequent 20 min were used. To obtain kcal/day values we used the equation proposed by Weir, 1949: $[(3.9 \times VO_2) + (1.1 \times VCO_2)] \times 1440$ [31]. For determination of the energy expenditure for the control, isocaloric, and caloric deficit protocols subjects used the same equipment, and seated on the cycle ergometer for 5 min before starting the protocols. Total energy expenditure was calculated as the sum of the absolute measured VO_2 ($l.^{min-1}$) for each minute during protocols (discarding the initial 5 min), multiplied by 3.5 (to obtain the metabolic equivalents), which were multiplied by 5.0 ($kcal.L^{-1}$) [32]. Post-exercise oxygen consumption was not considered for the determination of the caloric content of the meal. To report the energy expenditure of exercise, the mean of the two sessions is presented.

2.6. Maximal oxygen consumption test

The maximal oxygen consumption (VO_{2max}) was determined using an open circuit gas analyzer (MGC, CPX/D model). Maximum load tests were performed on a cycle ergometer (The Bike, Cibex, USA), The initial intensity was 25 W (W), and an increase of 25 W every minute (25 $W.min^{-1}$), maintaining a pedaling cadence of 70–80 revolutions per minute (rpm). A telemetric strap was positioned to continuously monitor the heart rate (HR) of the participants (S610, Polar Electro Oy, Finland). Subjects reported perceived exertion for every increase in intensity and were verbally encouraged to undertake maximum effort during the test. The test lasted for 8–12 min according to the recommendations of the American College of Sports Medicine [33] and ended when the participants reached one of the following criteria: (a) plateau in oxygen consumption; (b) heart rate $\geq$ predicted for age; (c) respiratory exchange rate value > 1.15; (d) perceived exertion >18 or when the subject voluntarily requested the test interruption [4].

2.7. Blood samples and biochemical analysis

Blood samples were obtained before BMR assessments using and hypodermic needle and syringe for a total of 5 mL from a vein of the antecubital region. Blood samples obtained during experimental protocols by placing a cannula (flexible polyvinyl chloride polymer, Teflon®) into a vein of the antecubital region. Saline was infused every 5 min to maintain the free access route for collection. A total of 8 blood samples (Before the protocol, before the meal, 1 h after the meal, and 5 subsequent samples; 10 mL each) were obtained, and the total sampling time was 255 min. This procedure was performed by a trained professional and using disposable material. The levels of triglycerides, total cholesterol, HDL-Cholesterol and plasma glucose were performed by enzymatic colorimetric method (Advia Bayer®), and LDL-C was estimated using the Friedewald formula [34]. The area under the curve (AUC) of post-prandial lipemia was calculated by the trapezoidal method as previously described [4].

2.8. Statistical analysis

The data were structured and analyzed using SPSS (*Statistical Package for Social Sciences*) version 26.0 for Windows. Normality of distribution for all the variables used Shapiro–Wilk test and the homoscedasticity of variances was evaluated with Levene's test. Experimental groups were compared before testing protocols for all variables using *Student's t* test for independent samples to determine whether there were differences at baseline between groups. Postprandial differences between different treatment groups was analysed using the two-way repeated measures ANOVA with post hoc of Bonferroni. We calculated incremental AUC for TG and glucose, which was compared within the same protocol between and within groups using two-way repeated measures ANOVA with Bonferroni's *post hoc* test. To isolate the effects of exercise and the

Table 1
General characteristics of the participants.

	Obese ($n = 12$)	Lean ($n = 9$)
Age (years)	25.6 ± 3.1	23.9 ± 2.0
Body mass (kg)	97.5 ± 12.8	73.03 ± 10.5*
Height (m)	1.73 ± 0.6	1.73 ± 0.8
BMI (kg/m^2)	32.34 ± 2.1	23.4 ± 1.5*
Waist circumference (cm)	99.7 ± 5.3	80.8 ± 6.6*
Sum of six Skinfolds (mm)	184.6 ± 34.1	84.23 ± 23.7*
VO_{2max} (mL.kg^{-1}.min^{-1})	27.2 ± 4.0	33.1 ± 4.1*
Adipose Mass (kg)	19.9 ± 4.00	34.8 ± 7.1*
Muscle Mass (kg)	34.0 ± 7.27	39.9 ± 4.9
Residual Mass (kg)	7.7 ± 1.58	9.9 ± 1.8
Bone Mass (kg)	7.6 ± 1.54	8.8 ± 1.0
Skin Mass (kg)	3.6 ± 0.56	3.9 ± 0.3
BMR (Kcal/dia)	1624.5 ± 207.7	1790.9 ± 350.0
Energy expenditure (Kcal)	277.75 ± 48.57	265.21 ± 50.33
HDL-Cholesterol (mg/dL^{-1})	36.83 ± 5.9	44.88 ± 12.92
LDL-Cholesterol (mg/dL^{-1})	101.9 ± 14.9	102.95 ± 22.6
Total Cholesterol (mg/dL^{-1})	161.91 ± 15.9	163.11 ± 31.0
Triglycerides (mg/dL^{-1})	115.58 ± 37.8	76.33 ± 22.7*

BMI = Body Mass Index; $VO_{2máx}$ = Maximal oxygen consumption. Data are shown as mean ± standard deviation. (*) Significative differences between groups, $p < 0.05$. ∑Skinfold were (Subscapular, Triceps, Abdominal, Supraspinal, Medial thigh and calf).

meal in both TG and glucose responses, we calculated the deltas: Postexercise − Baseline/Baseline, and 8 h Postmeal − Baseline/Baseline, expressed as percentuals. The results are expressed as mean and standard deviation (SD), and the significance level was $p > 0.05$.

3. Results

The characteristics of the 21 participants are presented in Table 1 (see also flowchart, supplemental file). As expected, subjects of the obese group presented significant higher body mass, BMI, waist circumference, sum of skinfolds, VO_{2max} and adipose mass. There were no differences between groups in fasting serum triglycerides, HDL, LDL, albeit we found significant higher total cholesterol for the obese group ($p = 0.008$). Notably, not all participants complete all trials, resulting in different subjects in different protocols as follows: Control (n): Obese = 12, and Lean = 9; Isocaloric (n): Obese = 12, and Lean = 7; Caloric Deficit (n): Obese = 9, and Lean = 9.

Figure 2 shows the results for serum triglycerides level in different time points representing the PPL response. During all protocols, we found no interaction effects, but significant effects regarding time, column and subject were observed (See Table 2). During the control protocol (Fig. 2 A) a significant difference was only observed in time-points 5 and 7 ($p = 0.0445$ and $p = 0.0483$, respectively). For the isocaloric (Fig. 2 B) significant differences were found between time-points 3 to 7 ($p < 0.04$). For the caloric deficit (Fig. 2 C) protocol we found a significant difference in all time-points ($p < 0.04$ for all points). Analysing the incremental area under the curve (iAUC) for triglycerides (Fig. 2 D), we found a significant statistical difference between lean and obese groups during the control, isocaloric and caloric deficit protocols ($p = 0.019$, $p = 0.006$, *and* $p = 0.001$, respectively). No differences were found between protocols within groups.

The glucose curve response to the protocols is presented in Fig. 3. During all protocols, we found significant effects regarding subject, and regarding column only in the isocaloric protocol (See Table 2). Similar glucose curve was observed for lean and obese subjects during the control, isocaloric, and caloric deficit protocols (Fig. 3, A, B, and C, respectively). Thus, when evaluating the iAUC for glucose, we found no significant differences between lean and obese subjects in the evaluated protocols (Fig. 3 D). However, iAUC was lower in lean subjects after the isocaloric and caloric deficit protocols when compared to the control protocol ($p = 0.006$, and $p = 0.0004$, respectively).

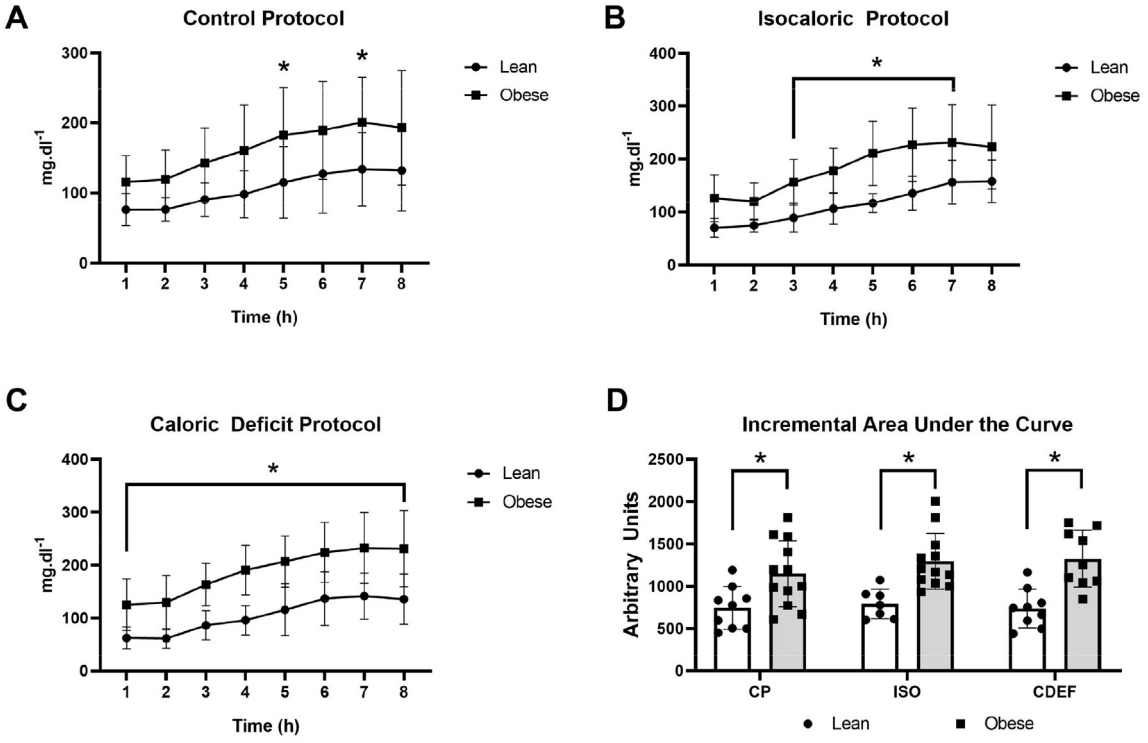

Fig. 2. Postprandial serum triglyceride levels of lean and obese subjects for the Control (A), Isocaloric (B) and Caloric Deficit (C). Incremental area under the curve (iAUC) of serum triglyceride shows a significant statistical difference (*) between groups obese and lean for all protocols (D).

Table 2
Two-Way Anova and Bonferoni's post-hoc results for Triglycerides and Glucose responses.

Triglycerides						
	Control		Isocaloric		Caloric Deficit	
	F (DFn, DFd)	*p*	F (DFn, DFd)	*p*	F (DFn, DFd)	*p*
Interaction	F (7, 133) = 0.8086	0.5816	F (7, 119) = 1.255	0.2784	F (7, 112) = 1.174	0.3236
Time	F (7, 133) = 23.18	**0.0001**[a]	F (7, 119) = 29.05	**0.0001**[a]	F (7, 112) = 42.91	**0.0001**[a]
Column	F (1, 19) = 7.181	**0.0148**[a]	F (1, 17) = 13.58	**0.0018**[a]	F (1, 16) = 18.74	**0.0005**[a]
Subject	F (19, 133) = 25.02	**0.0001**[a]	F (17, 119) = 13.62	**0.0001**[a]	F (16, 112) = 21.83	**0.0001**[a]
Glucose						
Interaction	F (7, 133) = 2.24	0.0342	F (7, 119) = 1.60	0.1402	F (7, 112) = 0.648	0.7147
Time	F (7, 133) = 2.06	0.0521	F (7, 119) = 1.52	0.1670	F (7, 112) = 1.84	0.0855
Column	F (1, 19) = 3.21	0.0890	F (1, 17) = 4.62	**0.0461**[a]	F (1, 16) = 1.95	0.1809
Subject	F (19, 133) = 10.73	**0.0001**[a]	F (17, 119) = 2.73	**0.0008**[a]	F (16, 112) = 2.58	**0.0019**[a]

Values in bold are statistically significant.
[a] Significant differences.

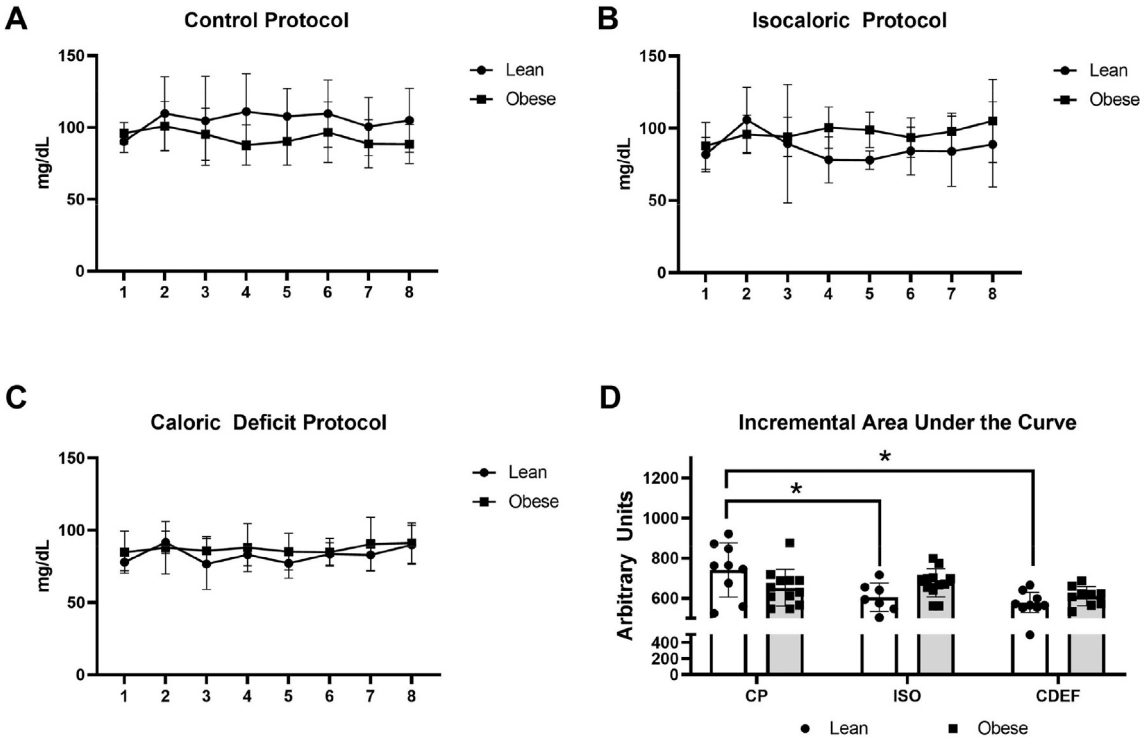

Fig. 3. Postprandial glucose levels of lean and obese subjects for the Control (A), Isocaloric (B) and Caloric Deficit (C). Incremental area under the curve (iAUC) of glucose is significantly lower (*) following the Isocaloric and Caloric Deficit when compared to the control protocol only in the lean group (D).

The individual percentual variation for triglycerides and glucose responses after the exercise and meals are presented in Fig. 4. Specific effects are described in Table 3. A significant difference was found between groups following the meal for the glucose curve after the control protocol, with obese presenting lower percentual variation when baseline values were taken as reference (Fig. 4 B; $p = 0.015$).

4. Discussion

The present study investigated the acute effects of a low-intensity fasted exercise on PPL and PPG in untrained lean and obese subjects submitted to high-fat meals with different caloric contents. The main finding of this study was low-intensity fasted aerobic exercise did not reduce PPL in both lean and obese groups when compared to the rest condition. However, through iAUC analysis we found that lean subjects had significantly lower AUC compared to obese relative to all protocols, albeit no differences were found within groups comparing different protocols. This result indicated that there is no beneficial short-term effect of Low-intensity fasted aerobic exercise on PPL and PPG responses following a high-fat meal, regardless of caloric content. However, as we analyzed individual responses, we identified a heterogeneous pattern in subjects from both groups. Not all lean subjects positively responded to the exercise protocol used in this study, as not all obese negatively responded to the same stimuli. Thus, this opens up an interesting discussion on responders and non-responders in both populations.

Prior exercise attenuates the PPL curve after a high-fat meal Factors such as exercise intensity and timing, body composition, energy expenditure, energy and macronutrients content of the meal may significantly influence the rate of blood TG removal and/

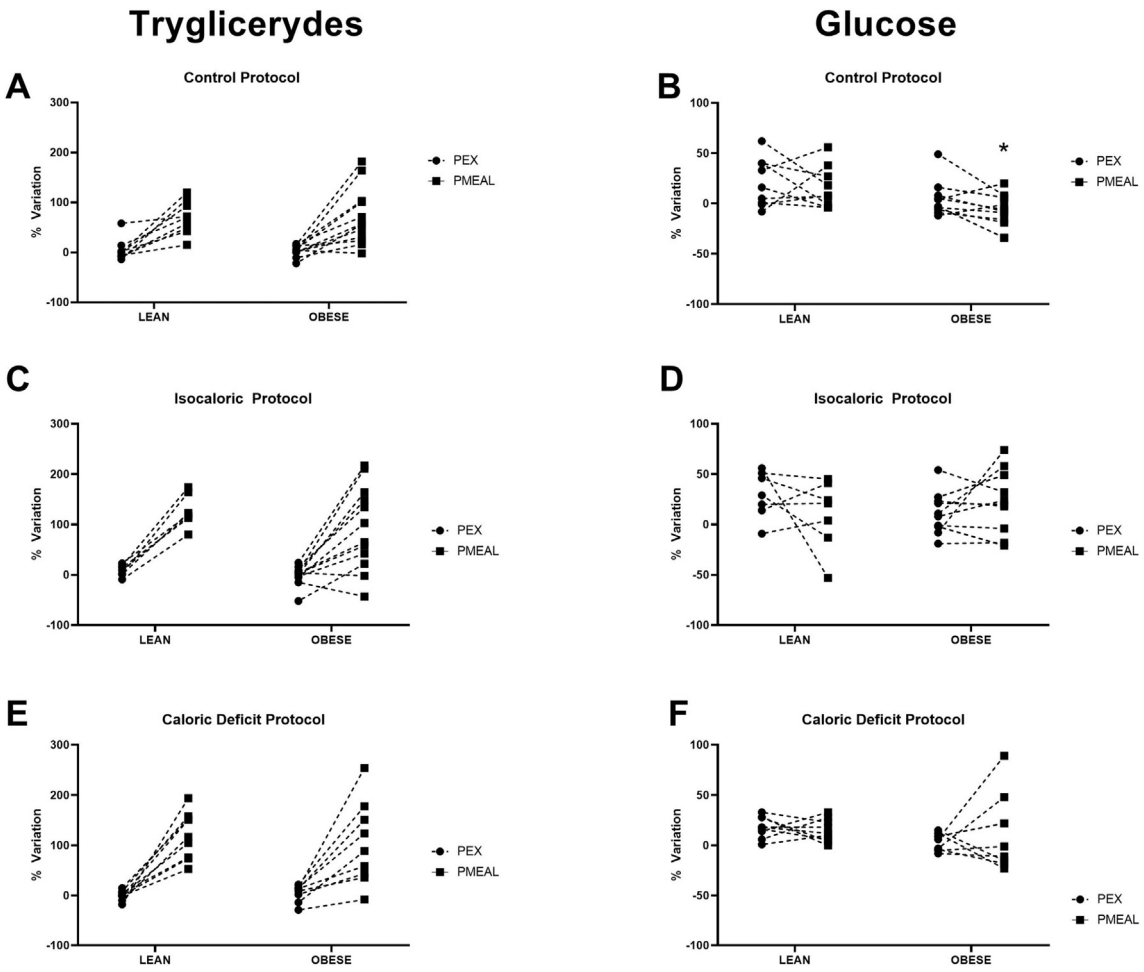

Fig. 4. Individual variation of triglycerides (A, C, and E) and glucose responses (B, D, and F). Obese individuals showed significant (*) lower percentual variation when baseline values were taken as reference for glucose responses during the control protocol (B).

Table 3
Two-Way Anova and Bonferoni's post-hoc results for individual Triglycerides and Glucose responses.

Triglycerides						
	Control		Isocaloric		Caloric Deficit	
	F (DFn, DFd)	*p*	F (DFn, DFd)	*p*	F (DFn, DFd)	*p*
Time	F (1, 16) = 0.17	0,6808	F (1, 12) = 1.67	0,2204	F (1, 32) = 0.12	0,7213
Column	F (1, 22) = 27.21	**0.0001**[a]	F (1, 22) = 27.28	**0.0001**[a]	F (1, 32) = 45.91	**0.0001**[a]
Time x Column	F (1, 16) = 0.02	0,8669	F (1, 12) = 0.34	0,5701	F (1, 32) = 0.48	0,4896
Glucose						
Time	F (1, 16) = 12.58	**0,0027**[a]	F (1, 34) = 0.10	0,7427	F (1, 32) = 1.52	0,2256
Column	F (1, 22) = 1.6	0,2105	F (1, 34) = 0.26	0,6093	F (1, 32) = 0.19	0,6637
Time x Column	F (1, 16) = 0.55	0,4688	F (1, 34) = 2.54	0,1197	F (1, 32) = 0.79	0,3779

Values in bold are statistically significant.
[a] Significant differences.

or VLDL secretion by the liver [16,35]. Our results are in line with other studies demonstrating that a single exercise session with or without a calorie deficit does not significantly reduce the PPL in lean and obese subjects [36,37]. Several studies have investigated the effect of exercise at intensities ranging from 25 to 70% VO_{2max}, on the PPL [37–41]. Katsanos et al. showed that exercise at a moderate intensity (65% VO_{2max}) significantly decreased PPL, whereas lower intensity (25% VO_{2Max}) has no effect in young male subjects [38]. Tsetsonis et al. [37] reported that low-intensity aerobic exercise (3 h, 32% VO_{2max}) and moderate-intensity (1.5 h, 63% VO_{2max}) reduced TG AUC, implying that the intensity does not influence PPL [37]. Taking together, these studies indicate that the volume (duration) of aerobic exercise is the most important factor to reduce PPL in lean subjects.

Our findings show that fasted low-intensity exercise (50% VO_{2max}) performed for 45 min, which promotes an energy expenditure lower than 300 kcal/session (277.75 + 48.57 kcal/session and 265.21 + 50, 33 kcal/session for the lean and obese group,

respectively) is not effective in reducing PPL in lean and class I obese subjects, corroborating with a recent study suggesting that 60 min of moderate aerobic exercise (50% VO_2) reduced the PPL in a different population [41]. Heden et al. [42] demonstrated that aerobic exercise performed the night prior to a mixed meal of approximately 600 kcal reduced postprandial TAG and improved insulin responses in obese compared to lean subjects. It is noteworthy that we submitted the subjects to a high-fat meal soon after the protocols and we used different exercise duration (45 min) and intensity (50% VO_2), which could account for some differences comparing to the present study. In addition, Ferguson et al. (1998), suggested that the attenuation of PPL needs aerobic exercise sessions with a caloric expenditure higher than 600 kcal [43]. Our results support the theory that the benefit of aerobic exercise on PPL is dependent on the total caloric expenditure of exercise.

Moreover, we found a difference in the PPL between the lean and obese subjects for all protocols (Fig. 2, D). Obese showed a higher TG curve compared to the lean group, independent of the caloric deficit as indicated by the iAUC. We can speculate some plausible mechanisms such as the decrease in the skeletal muscle and adipose tissue LPL, insulin resistance, high apoC-III levels, reduction in fatty acid oxidation, increased secretion or decreased removal of VLDL-TG from the plasma [16,35,44]. Nevertheless, this intra-group difference is in accordance to the findings of Burton et al. [45], reporting that the replacement of the exercise-induced energy expenditure lowered postprandial insulinemia and increased fat oxidation, albeit the exercise-induced energy deficit augmented both effects and decreased PPL in obese subjects.

Metabolic flexibility is the ability to shift from glucose to fat oxidation during different physiological conditions, such as exercise in the fasted state, and is influenced by training status and fat mass [46]. Our results showed PPG following fasted exercise were reduced in lean but not on obese subjects. This suggests that low-intensity fasted aerobic exercise may not be sufficient to attenuate the glucose curve after a high-fat meal on grade I obese subjects. We confirmed the findings of Prior et al. (2014) showing that obese adult subjects may present impairment in glucose uptake during exercise [47]. Importantly, Oberlin et al. (2014) showed a decrease in the glycemic curve in obese diabetic subjects who underwent sixty minutes of moderate-intensity aerobic exercise [48].

Trying to provide an individual profile regarding the effect of short-term exercise protocols on PPL, we identified different responses to the isocaloric and caloric deficit protocols. Considering that these obese subjects were not previously engaged in exercise or diet programs, we speculate that the genetic background could account for this effect. As demonstrated in a classic study [22] involving heredity, exercise, and health risks, there is an important variation between subjects from the same and different families regarding responses to training, ranging from 5 to 50 percent of gains between subjects of the same family (siblings) and up to 250 percent among subjects from different families. Remarkably, there are a percentage of respondents and nonresponders to concurrent training for several health outcomes [24]. Accordingly, sedentary overweight/obese insulin-resistant women can also differentially respond to high-intensity interval training and resistance training to significantly reduce glucose, insulin, and HOMA-IR values [49]. We also identified that not all lean subjects presented the same pattern of responses in the percentual variation of glucose responses. This individual analysis suggests a rapid metabolic adaptation to a lipid overload, which is more prominent in lean subjects. Additionally, future studies could address the long-term adaptations to low-intensity exercise in obese subjects.

The present findings have limitations. We assessed lean and obese individuals without obesity-related co-morbidities, aiming to minimize influential factors besides body composition differences. Therefore, we cannot out rule different results in a clinical population. In addition, our fasting period was 12-hours and longer periods could induce different results. Thus, to achieve the benefits of exercise on the glucose control of obese individuals it may be necessary a higher intensity, longer duration, and/or more than one exercise session. Moreover, future studies comparing diet composition and volume, and intensity of exercise leading to higher caloric deficits should be performed to establish wide precise strategies for individuals under high metabolic risk of cardiovascular events.

5. Conclusion

The present study shows that low-intensity fasted aerobic exercise has no acute effect on PPL responsiveness in lean and obese subjects. PPG following fasted exercise were reduced only in lean subjects. Body composition may influence PPL and PPG curve after a high-fat meal. Suggesting that one session of exercise is not sufficient to improve postprandial metabolism in obesity. Future studies should address the effects induced by higher duration or intensities of exercise on PPL.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clnesp.2020.11.013.

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