

Physical exercise and reduction of postprandial lipemia: the influence of caloric expenditure

Exercício físico e redução da lipemia pós-prandial: a influência do gasto calórico

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RESUMO | INTRODUÇÃO: Embora o efeito redutor de uma única sessão de exercício físico (EF) sobre a lipemia pós-prandial (LPP) seja controverso, estudos apontam que o gasto calórico é o principal determinante de redução da LPP. **OBJETIVO:** Testar a hipótese de que uma sessão de EF, baseado no gasto calórico modifica a LPP. **MÉTODOS:** Estudo de intervenção prospectivo controlado, tendo como população 18 homens com idade média de $22 \pm 1,3$ anos e IMC de $21 \pm 4,2 \text{ kg/m}^2$. Todos eram estudantes irregularmente ativos, com valores de triglicérides (TG) de jejum menor ou igual a 150mg/dl. Foram submetidos a dois testes de LPP: Basal (LPPB) e Exercício (LPPE). Amostras sanguíneas foram coletadas nos tempos 0 (jejum) e após a ingestão de um composto lipídico (25g) em 180 e 240 minutos para a dosagem dos TG. No teste da LPPE foi aplicado logo após a coleta de 120 minutos, uma sessão de EF em esteira ergométrica alcançando um gasto energético de 500kcal. **ESTATÍSTICA:** Foram comparadas as medianas da variação (delta) entre os valores dos TG dosados entre o jejum e 120min ($\Delta 1$), jejum e 240min ($\Delta 2$) e entre 120 e 240min ($\Delta 3$). Utilizou-se o teste de Wilcoxon bidirecional para comparação dos deltas. **RESULTADOS:** As medianas dos TG do $\Delta 1$ respectivamente para LPPB e LPPE foram de 63 Vs 60 ($p=0,95$); $\Delta 2$ 102 Vs 25 ($p=0,02$) e $\Delta 3$ 32 Vs -10 ($p<0,01$). **CONCLUSÃO:** Neste estudo, uma sessão de exercício físico baseado no gasto calórico de 500kcal, após a ingestão lipídica, reduziu o pico da LPP em jovens saudáveis.

PALAVRAS-CHAVE: Metabolismo. Lipídeos. Lipoproteínas. Medicina física e reabilitação. Dislipidemias.

ABSTRACT | INTRODUCTION: Although the reductive effect of a single physical exercise (PE) session on postprandial lipemia (PPL) is controversial, studies indicate that caloric expenditure is the main determinant of PPL reduction. **OBJECTIVE:** Test the hypothesis that a PE session based on caloric expenditure modifies PPL. **METHODS:** This is a prospective controlled intervention study, including a cohort of 18 men with average age of 22 ± 1.3 years and BMI of $21 \pm 4.2 \text{ kg/m}^2$. All were irregularly active students, with fasting triglyceride (TG) values of less than or equal to 150mg/dL. They were submitted to two PPL tests: Basal (PPLB) and Exercise (PPE). Blood samples were collected at time 0 (fasting) and after ingestion of a lipid compound (25g) at 180 and 240 minutes for TG dosing. In the PPE test, a PE session on treadmill was applied shortly after the collection at 120 minutes, achieving an energy expenditure of 500kcal. **STATISTICS:** The medians of the variation (delta) between the TG values between fasting and 120min ($\Delta 1$), fasting and 240min ($\Delta 2$) and between 120 and 240min ($\Delta 3$) were compared. The bi-directional Wilcoxon test was used to compare deltas. **RESULTS:** The TG medians of $\Delta 1$ respectively for PPLB and PPE were 63 Vs.60 ($p=0.95$); $\Delta 2$ 102 Vs. 25 ($p=0.02$) and $\Delta 3$ 32 Vs. -10 ($p<0.01$). **CONCLUSION:** In this study, a physical exercise session based on caloric expenditure of 500kcal, after lipid intake, reduced the PPL peak in healthy youngsters.

KEYWORDS: Metabolism. Lipids. Lipoproteins. Physical medicine and rehabilitation. Dyslipidemias.

Introduction

After a meal the elevation of triglycerides and lipoproteins rich in triglycerides in the bloodstream is observed, which is denominated postprandial lipemia (PPL)¹. Such condition is measured both in amplitude, corresponding to the time required for triglyceride values to return to baseline levels, and in magnitude, related to the moment at which triglycerides reach their highest value after lipid overload².

In the initial phase of PPL, lipid elevation occurs until it reaches the peak of plasma concentration, with a mean duration of 3 to 4 hours. The plateau represents the next step, in which the lipids remain stable after reaching the maximum level, in a time interval of 1 to 2 hours. Finally, the final stage corresponds to the decrease of the lipids until they return to basal values, in an average period of 2 to 3 hours³.

PPL is a very sensitive marker of the onset of atherosclerosis, being even more than fasting lipid values⁴. The more intense the magnitude and amplitude of PPL, the greater the feedback on insulin resistance, endothelial dysfunction and the risk of atheromatosis^{5,6}.

It is known that cardiovascular diseases, led by the synthesis of the atherosclerotic plaque, are among the main causes of death worldwide, so it is important to develop non-drug preventive and therapeutic measures that minimize these events⁷. Physical exercise, in a chronic way, is an important tool in PPL control⁸. Physical exercise can be defined as any body movement produced as a result of muscle contraction, being a necessarily planned, structured and repetitive activity^{9,10}. However, the reducing effect of a single exercise session on PPL is still the subject of several scientific discussions⁹. The duration, the time that the exercise is performed, the daily rest period and the caloric expenditure per session for a single physical exercise session to reduce PPL acutely are still questions not fully clarified by science.

More specifically, in a review conducted by Petto et al⁹. the authors pointed out that the main factor responsible for the reduction of PPL after a single physical exercise session is the caloric expenditure. Thus, the present study aimed to test the hypothesis

that a session of aerobic exercise, based on caloric expenditure, modifies the magnitude of PPL.

Materials and methods

Experimental design and sample

A prospective controlled intervention study, including a cohort of 18 men with an average age of 22 ± 1.3 years and a BMI of 21 ± 4.2 kg/m² was carried out. All were students of the Social Faculty, BA, Brazil, irregularly active, with fasting triglyceride values less than or equal to 150 mg/dL. Initially, 21 individuals were selected; of these, three did not attend the exercise protocol.

To determine if the individual was irregularly active, the International Short Physical Activity Questionnaire (IPAQ-long), developed by the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC) and validated in Brazil¹¹.

The following were excluded from the study: obese individuals that presented physical and clinical conditions incompatible with physical exercise, that use food supplements, anabolic, hypo or hyperlipid diet, lipid-lowering agents, corticoids, diuretics, beta-blockers, and that presented a history of alcoholism, hypothyroidism, parenchymal kidney disease or diabetes mellitus.

The cohort was asked to respond a standard questionnaire and underwent a subsequent physical examination, both having the function of collecting general information about the characteristics of the sample. Physical examination consisted in measuring Heart Rate (HR) and resting Blood Pressure (BP), total body mass, height and waist circumference.

For HR measurement, a Polar pulse heart rate monitor (model FT2) was used. Regarding the BP verification, the recommendations of the VII Brazilian Guidelines of Arterial Hypertension¹² were followed, using a sphygmomanometer for the average adult, duly calibrated by the National Institute of Metrology (Instituto Nacional de Metrologia - INMETRO) and a stethoscope duo-sonic, both of the brand BD.

Height was measured with the aid of a professional Sanny stadiometer (accuracy of 0.1 cm) with measurement performed while the subjects were barefoot and with the buttocks and shoulders supported in vertical abutment. The total body mass was verified through a Filizola digital scale (maximum capacity of 150kg), measured by INMETRO, with its own certificate specifying a margin of error of ± 100 g. For the measurement of the waist circumference, the lowest localized curvature between the ribs and the iliac crest was used, without compressing the tissues, using a metallic and flexible Starrett tape measure, with a measurement definition of 0.1 cm. When it was not possible to identify the slightest curvature, the measurement was obtained two centimeters above the umbilical scar¹³.

BMI was calculated with the measurements of mass and height, according to the following equation: $BMI = \text{mass (kg)} / \text{height}^2 \text{ (cm)}$. The BMI cutoff points adopted were those recommended by the IV Brazilian Guidelines on Dyslipidemias and Prevention of Atherosclerosis Department of Atherosclerosis of the Brazilian Society of Cardiology, that is, low weight (BMI <18.5); eutrophy (BMI 18.5-24.9); overweight (BMI 25-29.9) and obesity (BMI ≥ 30)¹⁴.

Ethical criteria

After sample's selection, the research steps were detailed in a language accessible to those selected, highlighting the study objectives, risks and benefits involved in the procedures. The informed consent form was signed before the candidates actually became volunteers. The project was approved by the Research Ethics Committee of the BAHIANA - School of Medicine and Public Health, under the protocol number 050/2008, and all procedures followed Resolution 466/12 of the National Health Council.

Collection protocol

On the day after the completion of the standard questionnaire and physical examination, the selected volunteers underwent a cardiopulmonary exercise treadmill test at the Cardiovascular Research Laboratory of the Teaching Assistance Unit of the Bahia School of Medicine and Public Health (Escola Bahiana de Medicina e Saúde Pública - EBMSp). The tests were applied by the same team, composed of

one cardiologist and two auxiliaries, using the ramp protocol and the VO2000 ergospirometer from Inbrasport (Porto Alegre, RS - Brazil).

Forty-eight hours after the cardiopulmonary test, the volunteers performed the PPL test in a specialized laboratory. The volunteers were instructed not to change their diet and not to carry out any physical exertion other than what is normal, one week before the test, as well as not to drink alcoholic beverages 24 hours before the laboratory evaluation.

For the collection, the studied population was submitted to a fast of twelve hours. The antecubital vein was cannulated from where blood samples were collected at zero times in which triglycerides, HDL, total cholesterol and blood glucose were measured by the enzymatic method. LDL was calculated by the Friedewald equation¹⁵. After the fasting blood collection, the volunteers ingested a diet cereal bar and a lipid compound containing 25g. Two and four hours after the ingestion of the lipid compound, the triglycerides were again dosed, while the subject was in a laboratory environment under resting conditions. This stage was termed baseline postprandial lipemia (PPLB) and served as a reference of post-exercise postprandial lipemia analysis.

The lipid compound used was provided by Tecnovida (São Paulo, SP, Brazil). Of the 25g of lipids, 15g were monounsaturated, 8g polyunsaturated and 2g saturated, corresponding to 45% of the daily fat intake recommended for a diet of 2,000 Kcal. The cereal bar contained 1.2g of proteins and 0.8g of lipids, but no carbohydrate. The bar was administered so that the intake of the lipid compound was more palatable and did not cause gastric discomfort.

Seven days after the PPLB test, the volunteers were submitted to the PPL with exercise test (PPLE). In this test, just like the previous, after a fast of 12h the triglycerides, total cholesterol and HDL were collected while LDL was calculated. Then, the volunteers ingested the same 25g lipid compound and the diet cereal bar (Flow chart 1). Once again triglycerides were dosed after 2h and 4h of the lipid compound intake. However, on this day, after the second hour, the volunteers underwent continuous exercise for approximately 50 minutes, with 75-80% of reserve heart rate (reserve HR = rest HR

+% x (HRmax obtained – HR at rest), on a treadmill (Johnson Health Tech T 707 Treo brand), which corresponds to the interval between the first and second ventilatory thresholds, that is, a moderate effort intensity (Flow chart 2). The average speed developed during training was between six and eight km/h and necessarily reaching a caloric expenditure of 500 kcal at the end of the session. Maximum HR was obtained in the cardiopulmonary test and HR at rest with the supine volunteer after five minutes.

Calculated caloric expenditure was measured by the same heart rate meter used to verify HR, based on the following variables: sex, height, mass, mean HR at rest and during exercise.

Statistical analysis

Before the analyses, symmetry and kurtosis tests were performed to identify the normality of the data and to test the assumptions of the analyses. The bidirectional paired Student's t test was used to compare the averages of the variables with normal distribution. To compare medians, the Wilcoxon test (Signed-Rank-Test) was used.

The deltas (Δ) reflecting the PPL, that is, the triglycerides variation between the fasting collection and the points 120min ($\Delta 1$) and 240min ($\Delta 2$) and ($\Delta 3$)

triglycerides variation between the 120 and 240min points, did not present a parametric distribution, being described in medians and interquartile intervals. Therefore, the Wilcoxon test was used to compare $\Delta 1$, $\Delta 2$ and $\Delta 3$. All analyses were performed in the statistical package (Statistical Package for the Social Sciences) SPSS version 13.0. The level of significance assumed was 5%.

In order to calculate the sample adequacy, alpha = 0.05 (bidirectional) and beta = 0.80 were considered, adopting as significant a difference of 20% between the deltas of the two days of collection. Considering that the laboratory variation coefficient of the triglycerides dosage is 5% and that a difference four times greater than expected cancels out the bias of this coefficient, 18 volunteers were then required. The sample calculation was performed in GraphPad StatMate 2.0 for Windows.

Results

The mean maximal heart rate obtained on the day of the cardiopulmonary test was 189 ± 4 bpm with mean test duration time of 8 ± 1 minutes.

The values of the fasting lipid profile and blood glucose on the days of PPLB and PPLE test did not present statistical difference as described in Table 1.

Table 1. Lipid profile and blood glucose on the two days of baseline and exercise collection. (n = 18)

Variables	Basal Test	Test Exercise	p* value
Triglycerides (mg/dL)	88±28	90±37	0.71
High density lipoprotein (mg/dL)	38±7	39±4	0.85
Low density lipoprotein (mg/dL)	121±24	123±10	0.62
Total cholesterol (mg/dL)	178±23	186±14	0.74
Glycemia (mg/dL)	85±10	84±9	0.98

* Bidirectional Student's t test for dependent sample.

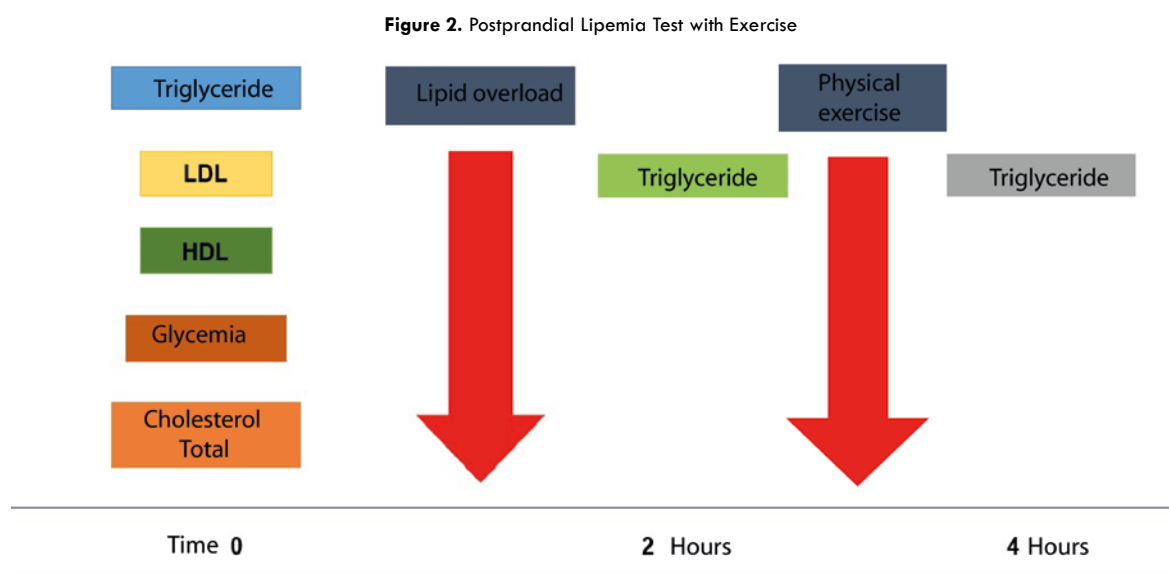
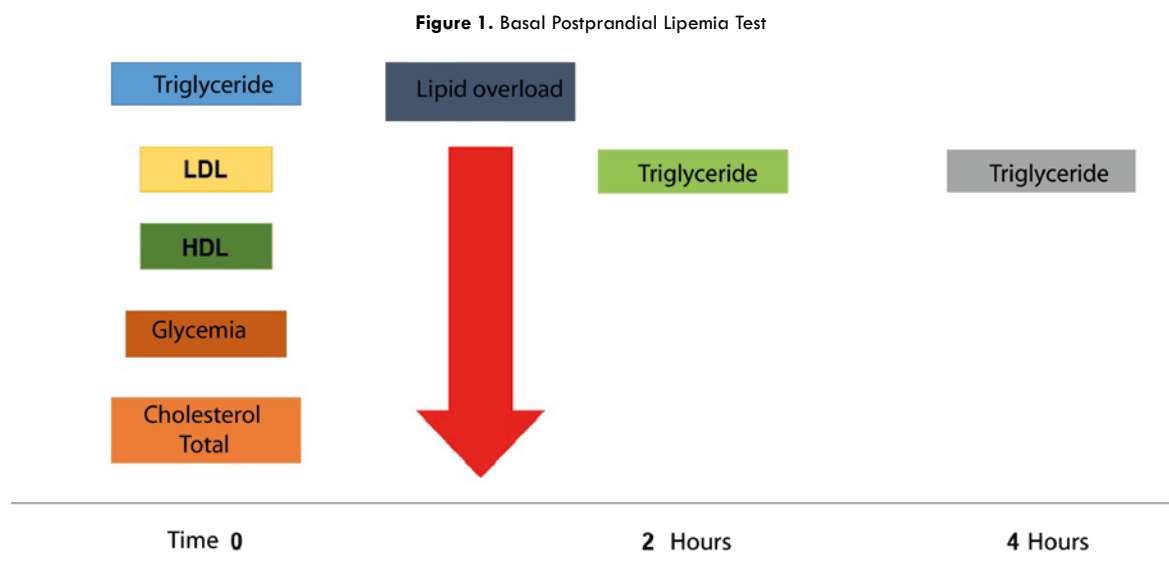
The values of triglycerides (mg/dL) at 2 and 4h after the ingestion of the lipid compound for the PPLB and PPLE test days were respectively 148 ± 72 vs. 148 ± 78 ($p=0.99$) for 2h and 179 ± 80 vs. 139 ± 82 ($p=0.04$) for 4h showing a significant difference.

Table 2 shows the comparison of deltas (reflecting PPL), identifying a difference between $\Delta 2$ and $\Delta 3$ of LPP, that is, PPL on the day of exercise was significantly lower than the baseline, between the 2nd and 4th hour.

Table 2. Values of triglyceride delta (mg/dL) corresponding to postprandial lipemia on the two days of collection. (n = 18)

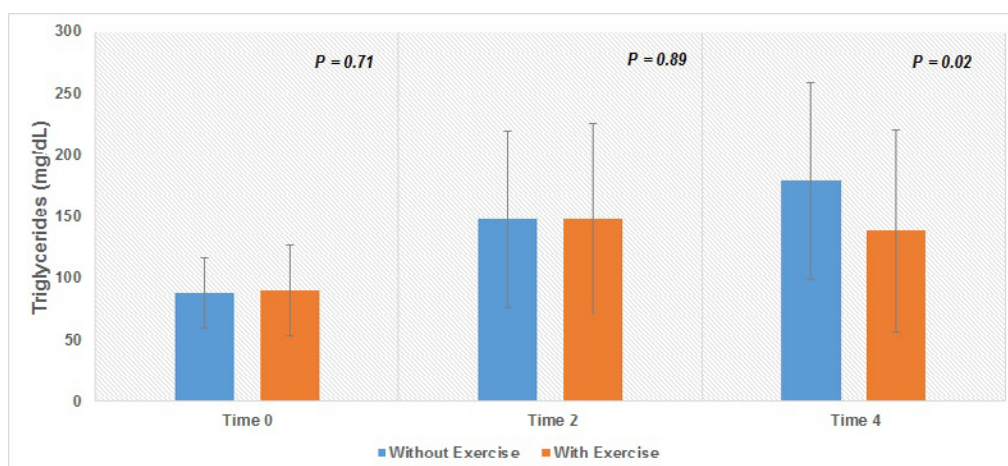
	Basal Test	Exercise Test	p* value
$\Delta 1$	63 (28-82)	60 (28-72)	0.95
$\Delta 2$	102 (52-122)	25 (15-73)	0.02
$\Delta 3$	32 (31-49)	-10 (-26-1)	< 0.01

$\Delta 1$ – Difference between fasting triglyceride values and those 2h after the ingestion of the lipid compound; $\Delta 2$ - Difference between fasting triglyceride values and those 4h after the ingestion of the lipid compound; $\Delta 3$ – Difference between triglyceride values, 2h and 4h after the ingestion of the lipid compound. *Wilcoxon test.



Graph 1 shows the mean values of the triglycerides at the collection points in the day of the PPLB and PPLE test.

Graph 1. Exercise and PPL



Discussion

According to the results obtained in the present study it is possible to infer that a session of aerobic physical exercise, based on caloric expenditure, reduces PPL in young normolipemic patients.

In unconformity with the present study, an investigation carried out by Wagmacker et al.¹⁶, in 2012, showed that moderate intensity exercise with a duration of 45 minutes, performed just before lipid overload, did not affect the PPL peak in subjects with central obesity. In another study Emerson et al.¹⁷ submitted 12 irregularly active overweight volunteers to a protocol of moderate intensity (60% of peak VO₂) on a treadmill with a duration of 30 and 60 minutes. After exercise, a 12-hour fast followed a meal rich in fat. Blood was then collected immediately before and 1, 2, 4, 6 and 8h after the meal, for triglyceride dosing. The results showed that neither 30 nor 60 minutes of moderate intensity physical exercise were able to reduce PPL or postprandial inflammation¹⁷. In a similar protocol, although with a longer duration, Bodel et al.¹⁸ verified that a 90-minute session of physical exercises also did not reduce PPL in overweight men.

It is interesting to observe similarities and differences between the 3 studies. The time of application of the exercise (performed before the intake of the lipid compound) and the intensity were similar in the three situations. As differences, the duration of physical exercise and the profile of the population

evaluated can be observed. However, regardless of these factors, no PPL reductions were found in any of these studies. Comparing these protocols with that of this study, it is important to note the moment of application of the exercise and the concern with the caloric expenditure. In the three studies mentioned above, the exercise was applied before the lipid overload, whereas in this study the exercise was applied 2h after lipid intake. Another point was that, in the last two, the protocols were based on intensity and duration and not on caloric expenditure. Although Wagmacker et al.¹⁶ have also based their work on caloric expenditure (250kcal), this was lower than the expenditure proposed for the reduction and used in this study (500kcal).

Some studies have also sought to increase the intensity variable, in order to observe the effects on PPL. In the analysis of the effects of high intensity interval exercise (80% of peak VO₂) and short duration (20 minutes), Petto et al.⁹ did not show PPL reductions of irregularly active youngsters. As justification, the authors pointed out the fact that there was probably no significant metabolic expenditure, due to the reduced treadmill time, despite the accentuated intensity.

On the other hand, Gabriel et al.¹⁹ detected a significant reduction in postprandial triglyceride levels after intermittent high-intensity exercise. As a difference between the studies, the physical activity in the first case is highlighted after the lipid overload and preceding the ingestion of lipids in the second

example. In both cases the duration of the session was similar.

In research that shows beneficial effects of physical exercise with PPL, a significant variable presents a significant metabolic imbalance in its protocols. This imbalance, however, has no effect when mediated only by the restriction of the previous caloric supply, and there is a need for a significant energy expenditure mediated by physical exercise²⁰.

Ferreira et al.²¹ in 2011 showed that physical exercises of high or moderate intensity, performed before fat intake, are able to reduce the levels of postprandial lipids, since they present a consumption of 500kcal during the session. This information also corroborates the results of the present study, which, although performed after fat intake, stipulated the same energy expenditure and also detected PPL reduction.

The caloric expenditure generated by the exercise is therefore one of the pillars for PPL reduction, since even when performed at lower intensity, it can still be more effective in comparison to the high-intensity exercise. Logically, a combination with the other variables involved in physical exercise may or not guarantee the expected results. By illustrating this possibility, we can cite the research carried out by Emerson et al.¹⁷ where the group that performed physical exercises for a period of 60 minutes with a caloric expenditure of 582 kcal did not present a reduction in PPL. Thus, it cannot be said that energy consumption is sovereign and guarantees a reduction in PPL. As an example, interestingly, in the study conducted by Gabriel et al.¹⁹, it could be seen that the protocol that promoted lower energy expenditure reduced the triglyceride-rich lipoproteins of the blood stream more effectively.

In the present study, an approximate increase of 31mg/dL in triglyceride values could be observed from the second to fourth hour in the PPLB test, whereas in the PPLE test there was a reduction of 9mg/dL in the triglycerides. The mechanisms responsible for this difference are not fully explained by the literature, however, the increase in fat uptake by peripheral tissues and the reduction in the release of very low-density lipoproteins by the liver, both caused by exercise, seem to be the main responsible²².

Some researches usually provide a calorie-dense meal to address the energy deficit, both in adults and adolescents, and sometimes this “replacement” has been pointed out as being responsible for not reducing PPL^{17,23,24}. In these cases, even if a physical exercise session is performed at high intensity (> 80% peak VO₂) and with high energy expenditure (> 500kcal), it is still possible to have no positive impact on PPL.

Thackray et al.²⁵, in 2016, affirmed that a variable, which until then was little debated in relation to the others, can be determinant as regards the success or not of the physical exercise/PPL relation: prolonged rest in the days previous to the evaluation. This study verified that even in activities with a duration of 1h at moderate intensity (67% VO₂max) and significant energy consumption (730kcal) the reduction of PPL can be verified or not. The group in which the positive outcome was observed had been encouraged to be active daily on the two days prior to the physical exercise session and assessment of lipid levels; while the group that remained inactive (approximately 14.3h of daily rest/two days) had higher PPL levels²⁵.

It is worth noting that in the study conducted by Kim et al.⁸, for the “active” classification, the use of ladders and long walks was encouraged in detriment to the use of elevators and automotive vehicles in the daily transport. A device quantifying the number of steps taken during the day was also used, which ensured that the “active” group did about 15,500 steps more than the inactive group⁸.

The periods of immobility need to be longer lasting to overcome the beneficial effects of physical exercise in PPL. Another study carried out by Kim et al.⁶ promoted post-meal lipid reduction both in the active and inactive group, which in this case was subjected to a fixed daily inertia of 8h/day.

This prolonged daily rest can promote adverse effects on cardiovascular health and even generate accidental mortality. Since it does not reduce PPL, inactivity for most of the day may contribute to the formation of the atherosclerotic plaque regardless of practicing physical exercise, thus causing a series of undesirable ischemic changes⁴.

It should be noted that, with the exception of the studies conducted by Thackray et al.⁸, none of the other studies previously cited mentioned the variable prolonged daily rest. This observation could probably justify a series of failures of other studies, since a variable as important as this one was disregarded in overweight and obese populations as well as in irregularly active individuals.

The present study counted on university students who, for the most part, work, performing dynamic activities during the day that are incompatible with idleness. This fact allied to the protocol with stipulated caloric expenditure of 500kcal and the moment of exercise application (2h after the intake of the lipid compound) are preponderant factors for the reduction of PPL after physical exercise. The active lifestyle may also have been the differential for the positive effects of exercise in protocols developed in low intensity, which justifies, until then, unlikely results²⁰.

Therefore, it is hypothesized that in addition to the variables discussed and analyzed in this work, a combination that involves exercises of higher intensities, durations and energy expenditures allied to an active routine, is the rite that promotes greater attenuation of PPL and possibly provides greater reduction of atherosclerosis and of the clinical conditions attributed to it.

Conclusion

In this study, a physical exercise session based on caloric expenditure of 500kcal, after lipid intake, reduced the PPL peak in healthy youngsters. Although the positive results are attributed to the stipulated energy expenditure, one cannot point out this variable as being the only one responsible for the reducing effect of PPL.

Author contributions

Petto J, Gomes VA, Santos, ACN, Ladeia AMT participated in the study conception and design. Petto J, Sacramento MS, Gomes VA and Santos CAN participated in the data collection. Petto J, Sacramento, MS, Gomes VA, Santos CAN e Ladeira AMT participated in the writing and critical review and approval of content.

Conflict of interests

Dr. Ladeia, Dr. Gomes, Dr. Andrade, Dr. Petto, Dr. Santos, and Dr. Sacramento reports grants from Fundação de Amparo à Pesquisa da Bahia (FAPESB), during the conduct of the study; grants from Fundação de Amparo à Pesquisa da Bahia (FAPESB), outside the submitted work.

References

1. Zilvermit DB. Atherogenesis: a postprandial phenomenon. *Circulation*. 1979;60:473-85. doi: [10.1161/01.CIR.60.3.473](https://doi.org/10.1161/01.CIR.60.3.473)
2. Valdés PA, Ramos DC, Mehta R, Hernandez LM, Bautista IC, Mendez OP et al. Factors associated with postprandial lipemia and apolipoprotein A-V levels in individuals with familial combined hyperlipidemia. *BMC Endocr Disord*. 2014;14:90. doi: [10.1186/1472-6823-14-90](https://doi.org/10.1186/1472-6823-14-90)
3. Petto J, Wagmacker DS, Andrade ALS, Tenorio MCC, Ladeia AMT. Variação das lipoproteínas plasmáticas durante a lipemia pós-prandial em repouso e pós-exercício físico. *Revista Brasileira de Fisiologia do Exercício*. 2014;13(4):217-223.
4. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med*. 2001;161(12):1542-1548. doi: [10.1001/archinte.161.12.1542](https://doi.org/10.1001/archinte.161.12.1542)
5. Nakamura K, Miyoshi T, Yunoki K, Ito H. Postprandial hyperlipidemia as a potential residual risk factor. *J Cardiol*. 2016;67(4):335-9. doi: [10.1016/j.jicc.2015.12.001](https://doi.org/10.1016/j.jicc.2015.12.001)
6. Kim IY, Park S, Trombold JR, Coyle EF. Effects of moderate- and intermittent low-intensity exercise on postprandial lipemia. *Med Sci Sports Exerc*. 2014;46(10):1882-1890. doi: [10.1249/MSS.0000000000000324](https://doi.org/10.1249/MSS.0000000000000324)
7. Dahlof B. Cardiovascular disease risk factors: epidemiology and risk assessment. *Am J Cardiol*. 2010;105(supl 1):3A-9A. doi: [10.1016/j.amjcard.2009.10.007](https://doi.org/10.1016/j.amjcard.2009.10.007)
8. Kim IY, Park S, Chou TH, Trombold JR, Coyle EF. Prolonged sitting negatively affects the postprandial plasma triglyceride-lowering effect of acute exercise. *Am J Physiol Endocrinol Metab*. 2016;311(5):E891-E898. doi: [10.1152/ajpendo.00287.2016](https://doi.org/10.1152/ajpendo.00287.2016)
9. Petto J, Pereira JA, Britto RP, Sá CK, Souza LAP, Ladeia AMT. Efeito agudo imediato de uma sessão de exercício físico sobre a lipemia pós-prandial em jovens irregularmente ativos. *Rev Bras Cardiol*. 2013;26(2):100-105

10. Caspersen CJ, Kriska AM, Dearwater SR. Physical activity epidemiology as applied to elderly populations. *Baillieres Clin. Rheumatol.* 1994;8(1):7-27. doi: [10.1016/S0950-3579\(05\)80222-5](https://doi.org/10.1016/S0950-3579(05)80222-5)
11. Matsudo SM, Matsudo VR, Araújo T, Andrade D, Andrade E, Oliveira L et al. Nível de atividade física da população do Estado de São Paulo: análise de acordo com o gênero, idade, nível socioeconômico, distribuição geográfica e de conhecimento. *Rev Bras Cien Mov.* 2002;10(4):41-50.
12. Malachias MVB, Souza WKS, Plavnik FL, Rodrigues CIS, Brandão AA, Neves MFT et al. 7ª Diretriz Brasileira de Hipertensão Arterial. *Arq Bras Cardiol* 2016; 107(3Supl.3):1-83. doi: [10.5935/abc.20160153](https://doi.org/10.5935/abc.20160153)
13. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. Geneva: World Health Organization; 2000.
14. Sposito AC, Caramelli B, Fonseca FA, Bertolami MC, Afíune Neto A, Souza AD et al. IV Diretriz Brasileira Sobre Dislipidemias e Prevenção da Aterosclerose. Departamento de Aterosclerose da Sociedade Brasileira de Cardiologia. *Arq Bras Cardiol.* 2007;88(supl 1):2-19. doi: [10.1590/S0066-782X2007000700002](https://doi.org/10.1590/S0066-782X2007000700002)
15. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem.* 1972;18(6):499-502.
16. Wagemacker DS, Souza KK, Graça JV, Barreto GC, Macêdo PCS, Ferreira TF et al. Acute effect of moderate intensity physical exercise on postprandial hyperlipaemia of individuals with central obesity. *Health.* 2012;4(12A):1546-1550. doi: [10.4236/health.2012.412A221](https://doi.org/10.4236/health.2012.412A221)
17. Emerson SR, Kurti SP, Snyder BS, Sitaraman K, Haub MD, Rosenkranz SK. Effects of thirty and sixty minutes of moderate-intensity aerobic exercise on postprandial lipemia and inflammation in overweight men: a randomized cross-over study. *Journal of the International Society of Sports Nutrition.* 2016;13:26. doi: [10.1186/s12970-016-0137-8](https://doi.org/10.1186/s12970-016-0137-8)
18. Bodel NG, Gillum T. 90 Minutes of Moderate-Intensity Exercise does not Attenuate Postprandial Triglycerides in Older Adults. *Int J Exerc Sci.* 2016;9(5):677-684
19. Teixeira M, Kasinski N, Izar MCO, Barbosa LA, Novazzi JP, Pinto LA et al. Efeitos do exercício agudo na lipemia pós-prandial em homens sedentários. *Arq Bras Cardiol.* 2006;87(1):3-11. doi: [10.1590/S0066-782X2006001400002](https://doi.org/10.1590/S0066-782X2006001400002)
20. Gabriel B, Ratkevicius A, Gray P, Frenneaux MP, Gray SR. High-intensity exercise attenuates postprandial lipaemia and markers of oxidative stress. *Clin Sci (Lond).* 2012;123(5):313-321. doi: [10.1042/CS20110600](https://doi.org/10.1042/CS20110600)
21. Gill JMR, Hardman AE. Postprandial lipemia: effects of exercise and restriction of energy intake compared. *Am J Clin Nutr.* 2000;71(2):465-71. doi: [10.1093/ajcn/71.2.465](https://doi.org/10.1093/ajcn/71.2.465)
22. Ferreira AP, Ferreira CB, Souza VC, Córdova COA, Silva GCB, Nóbrega OT et al. The influence of intense intermittent versus moderate continuous exercise on postprandial lipemia. *Clinics (São Paulo).* 2011;66(4):535-41. doi: [10.1590/S1807-59322011000400003](https://doi.org/10.1590/S1807-59322011000400003)
23. Gill JMR, Hardman AE. Exercise and postprandial lipid metabolism: an update on potential mechanisms and interactions with high-carbohydrate diets. *J. Nutr. Biochem.* 2003;14(3):122-132. doi: [10.1016/S0955-2863\(02\)00275-9](https://doi.org/10.1016/S0955-2863(02)00275-9)
24. Thackray AE, Barrett LA, Tolfrey K. High-Intensity Running and Energy Restriction Reduce Postprandial Lipemia in Girls. *Med Sci Sports Exerc.* 2016;(48)3:402-411. doi: [10.1249/MSS.0000000000000788](https://doi.org/10.1249/MSS.0000000000000788)
25. Thackray AE, Barrett LA, Tolfrey K. Energy replacement diminishes the effect of exercise on postprandial lipemia in boys. *Metabolism.* 2016;65(4):496-506. doi: [10.1016/j.metabol.2015.12.001](https://doi.org/10.1016/j.metabol.2015.12.001)