

College of Saint Benedict and Saint John's University

DigitalCommons@CSB/SJU

Celebrating Scholarship & Creativity Day

Experiential Learning & Community
Engagement

4-27-2017

High intensity versus resistance exercise on postprandial triglycerides in healthy college students

Tyler J. Bruinsma

College of Saint Benedict/Saint John's University, tjbruinsma@csbsju.edu

Follow this and additional works at: https://digitalcommons.csbsju.edu/elce_cscday

Recommended Citation

Bruinsma, Tyler J., "High intensity versus resistance exercise on postprandial triglycerides in healthy college students" (2017). *Celebrating Scholarship & Creativity Day*. 124.

https://digitalcommons.csbsju.edu/elce_cscday/124

This Presentation is brought to you for free and open access by DigitalCommons@CSB/SJU. It has been accepted for inclusion in Celebrating Scholarship & Creativity Day by an authorized administrator of DigitalCommons@CSB/SJU. For more information, please contact digitalcommons@csbsju.edu.

High intensity versus resistance exercise on postprandial triglycerides in healthy college students

Tyler J. Bruinsma, Amy C. Olson, Ph.D., R.D.N., L.D.

Department of Nutrition, College of Saint Benedict and Saint John's University

May 5, 2017

Abstract

Elevated postprandial triglycerides contribute to the development of cardiovascular disease. Prior exercise is a well-established method to lower postprandial triglycerides; however, most exercise protocols involve prolonged aerobic exercise of sixty to ninety minutes which is not attainable by the general population. **PURPOSE:** The present study investigates the effect of high intensity interval training (HIIT) of twenty minutes and resistance training of thirty minutes on postprandial triglycerides. **METHODS:** Approval for this study was obtained from the Institutional Review Board of the University and signed informed consents were provided by all participants. Thirty healthy college-age students (5 males, 25 females) were recruited from nutrition and exercise science courses. All subjects completed the control, HIIT, and resistance protocols one week apart. Subjects performed the exercise protocols 12-16 hours prior to an oral fat tolerance test (milkshake, 1 g of fat per kg of body weight). Each exercise session was supervised; high intensity interval training consisted of four 30 second sprints with 4 minutes of walking recovery and the low-volume resistance training consisted of six machine-based lifts, 2 sets of 8 repetitions at 75% of one repetition maximum. Postprandial triglycerides were measured at baseline and three hours following consumption of the milkshake using the CardioChek PA blood analyzer. Data was analyzed using a three way repeated measures ANOVA statistical test. **RESULTS:** The means are as follows for each treatment and time point (baseline, and postprandial respectively): Control 107 +/- 49, 140 +/- 73 mg/dL; HIIT 93 +/- 34, 122 +/- 59 mg/dL; and Resistance 108 +/- 47, 144 +/- 64 mg/dL. A significant effect of treatment was noted indicating that HIIT TG values were lower than control ($p=0.027$); however, the treatment and time interaction was not significant ($p=0.699$). **CONCLUSION:** Twenty minutes of HIIT provided a sufficient energy deficit to significantly lower fasting and postprandial triglyceride values in a healthy, college-age population. Thirty minutes of low volume resistance training did not alter fasting or postprandial triglycerides in comparison to control values.

Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States, accounting for 23.5% of deaths in the most recent CDC survey [1]. Elevated serum cholesterol has historically been blamed as the predominant pathology of CVD [2]. Newer research demonstrates the importance of triglycerides in the pathophysiological processes implicating elevated postprandial triglycerides as an independent marker of CVD risk [2-4]. Postprandial triglycerides are of interest as a majority of modern life is spent in a postprandial state. Past research demonstrates the effectiveness of prior exercise at lowering postprandial triglycerides and therefore risk of CVD.

Prolonged aerobic exercise is the most studied form of exercise for lowering postprandial triglyceride responses. The majority of studies involving prolonged aerobic exercise use walking or other low to moderate intensity aerobic exercises, such as cycling for sixty to ninety minutes [5]. Past studies noted significantly lower postprandial triglyceride values and triglyceride area under the curve with prior exercise as compared to control with attenuations as high as 30% [6-9]. Prolonged aerobic exercise is effective in both healthy and hyperlipidemic populations [6, 8]. Implementing prolonged exercise protocols such as 60-90 minutes of walking is difficult due to the significant time commitment. ACSM guidelines recommend a minimum of thirty minutes of exercise per day, far less than the 60-90 minute sessions that were effective [10]. Twenty to thirty minutes of exercise at or above 65% of maximum heart rate lowered postprandial triglyceride area under the curve by 30% and postprandial triglyceride peak; however, maintaining high intensity levels for twenty to thirty minutes is difficult for many populations, particularly untrained individuals who are at a higher risk for hyperlipidemia and CVD [11, 12].

High intensity interval training (HIIT) consists of short bouts of high intensity activity (sprints) followed by a recovery period consisting of low to moderate activity. This cycle is repeated multiple times in a single training session and is typically performed on a cycle ergometer/bike or by running/walking. HIIT for twenty to thirty minutes is as effective as prolonged aerobic exercise at improving multiple markers of aerobic fitness [13-17]. The effect of acute HIIT sessions on postprandial triglycerides is equally comparable, though not as well studied [18-21]. HIIT provides an alternative, low time commitment exercise protocol that doesn't require prolonged bouts of high intensity exercise and is increasingly recommended to all populations. Elucidating the minimum time and intensity required for effective triglyceride lowering is an important next-step in determining the utility of HIIT [22].

Resistance exercise contributes to health maintenance by increasing muscle and bone strength. The American College of Sports Medicine (ACSM) recommends two to three, 30 minute sessions of resistance exercise per week [10]. Resistance training of sixty to ninety minutes lowers postprandial triglycerides with similar effectiveness to prolonged aerobic and HIIT exercise [5, 23-26]. Both high and moderate intensity protocols lowered postprandial triglyceride area under the curve equally well, by an average of 30% [25]. The prolonged nature and moderate to high intensity of the previously employed protocols is unattainable for the general population and exceeds ACSM minimum recommendations [10]. Further examination of lower intensity and shorter resistance exercise protocols for inducing lower postprandial triglycerides may lead to more realistic training interventions for the general population.

The purpose of the present study is to further examine the triglyceride lowering effects of shorter and lower intensity HIIT and resistance exercise protocols that are more widely attainable. The exercise protocols used in the present study are consistent with minimum ACSM exercise recommendations, allowing for the validation of these recommendations in the realm of triglyceride and CVD risk lowering. Confirming the effectiveness of HIIT and low volume and intensity resistance exercise is clinically relevant as providers seek to prescribe reasonable exercise interventions to patients at risk for CVD.

Methods

Subjects

Subjects were recruited from nutritional biochemistry and exercise science and sports studies courses offered at the College of Saint Benedict and Saint John's University. All were healthy and between the ages of 18 and 23. Subjects signed an informed consent and the study was approved by the Institutional Review Board of the College of Saint Benedict and Saint John's University.

Study Design

The study was cross-over in nature with subjects acting as their own controls. All subjects participated in three trials, a non-exercise control, HIIT, and resistance exercise (RE) with at least five days between each trial. Data was collected over two days with the exercise protocols being performed 12-16 hours prior to the oral fat tolerance test (OFTT). Subjects were instructed to consume similar diets, avoid alcoholic beverages, and refrain from exercise for 24 hours prior to the OFTT. Subjects provided a diet log to the research team including all foods consumed during this 24-hour period. Diet logs from all three trials were compared by research staff to ensure consistency in diet. Subjects were asked to fast for at least four hours prior to the OFTT. Fasting blood triglyceride levels were assessed immediately prior to the OFTT. Postprandial blood triglyceride levels were evaluated three hours following consumption of the OFTT. Subjects completed a post-survey regarding normal exercising habits, total fasting time prior to the OFTTs, and medications that could alter blood triglycerides. Participants were also asked if they consumed food between the exercise protocol and OFTT.

Exercise Protocols

All exercise sessions were supervised by the research team to ensure proper participation by all subjects. All sessions commenced with a dynamic warm-up consisting of a slow jog for 200 meters and light stretching. The HIIT protocol consisted of a thirty second sprint at maximal effort followed by four minutes of recovery consisting of a brisk walk. The HIIT protocol was similar to [21] but was performed on an indoor running track instead of a cycle ergometer. Subjects were introduced to the lifting protocol and

instructed in proper form forty-eight hours prior to the resistance exercise protocol. One-repetition maximums were also determined at this time, using a standard estimation chart. The resistance exercise protocol was developed with guidance from an NSCA certified strength and conditioning specialist and consisted of the following seven exercises performed on machines and in the indicated order: leg extension, seated chest press, seated row, leg press, latissimus dorsi pull down, triceps extension, sit ups. Two sets of eight repetitions at seventy-five percent of one repetition maximum were performed for all exercises except for sit-ups which were performed until failure. One minute of recovery was allowed between each set.

Oral Fat Tolerance Test

The OFTT consisted of heavy whipping cream (Kemps) and ice cream in equal parts. Lactose-free ice cream (Lactaid) was used for lactose-sensitive participants and Cookies N' Cream (Kemps) ice cream was used for all other participants. The resulting shake contained eight grams of fat per ounce and was administered to participants in a dosage to provide one gram of fat per kilogram of body weight.

Triglyceride Measurements

Blood triglyceride levels were measured using the CardioChek PA (PTS Diagnostics, Indianapolis, IN) which has been previously validated for diagnostic accuracy [27]. Blood for analysis was obtained by finger stick.

Statistical Analysis

A three by two, repeated measures ANOVA was performed to compare triglyceride levels. Correlational studies were performed to assess relationships between survey results and observed triglyceride data. Data analysis was completed using SPSS software (IBM).

Results

Thirty subjects were recruited, five males and twenty-five females. The average BMI of subjects was 24, and all were between the ages of 18 and 23 at the time of the study. Only one subject did not participate in regular exercise for at least one hour a week. Thirty-one percent of subjects exercised between one and three hours per week; sixty-six percent exercised at least four hours per week. Forty-two percent of subjects reported partaking in aerobic training, fifteen percent reported partaking in just resistance training and thirty-eight percent partake in both.

The triglyceride levels at baseline were, on average in the desirable range of less than 150 mg/dL. The mean and standard deviation for each treatment and time point are depicted in Figure 1. Statistical analysis revealed a significant main effect of treatment ($p=0.027$) and time ($p<0.001$); however, the interaction between treatment and time was not significant ($p=0.699$). The significant treatment effect indicates that HIIT lowered both fasting and postprandial triglyceride levels as compared to the non-exercise control. Resistance training did not lower fasting or postprandial triglycerides as compared to the control. The lack of a statistically significant interaction of treatment and time indicates that the exercise treatments did not affect the change in triglyceride values from fasting to postprandial. The changes in triglyceride values from fasting to postprandial were: Control: 33 mg/dL, HIIT: 29 mg/dL, Resistance: 36 mg/dL. No significant correlations were noted between fasting or change in triglycerides and: normal training time or type, length of fasting time, or energy replacement following exercise.

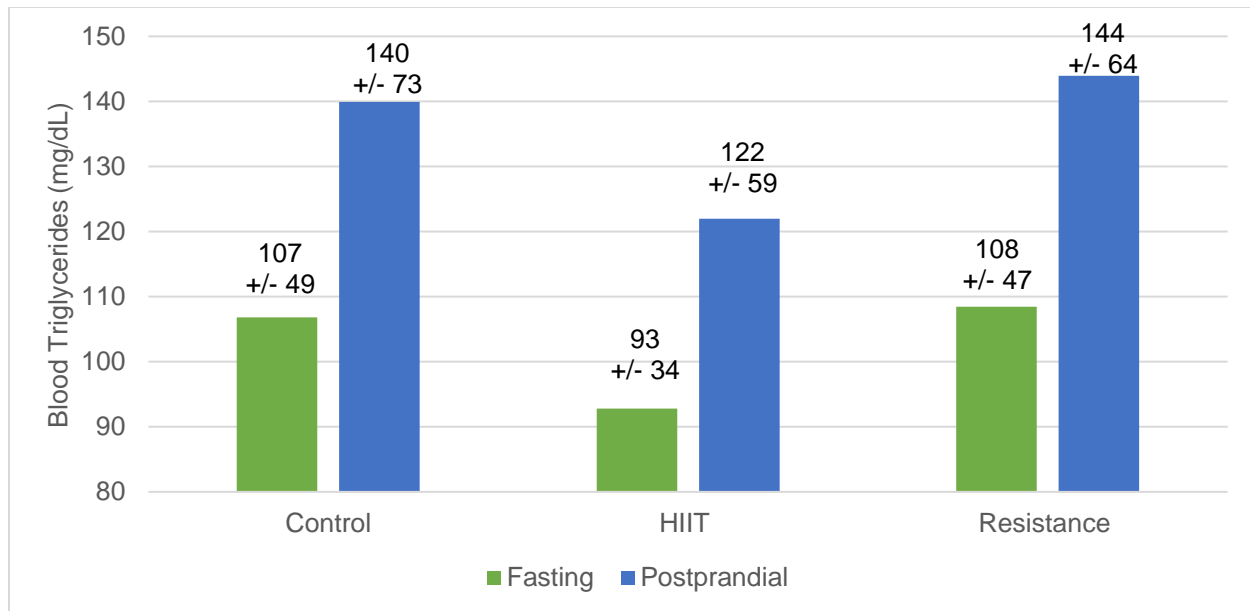


Figure 1: Mean triglyceride levels of college age students fasted and three hours postprandial for control, HIIT, and resistance trials. Average values \pm standard deviations are as follows (baseline, and postprandial respectively): Control 107 ± 49 , 140 ± 73 mg/dL; HIIT 93 ± 34 , 122 ± 59 mg/dL; and Resistance 108 ± 47 , 144 ± 64 mg/dL.

Surprisingly, some participant's (Control: 10%, HIIT: 20%, Resistance: 17%) blood triglycerides decreased from fasting to postprandial. Participants fasted for an average of eight hours prior to fasting triglyceride measurements during all trials. The diet logs revealed some inconsistency in meals between trials. A majority of subjects consumed similar food groups but exact meals varied from trial to trial. Sufficient information for complete macronutrient analysis was not provided by participants. Thirty-five percent of participants reported taking a medication that may affect blood triglycerides; however, all participants were consistent with medications during all three trials.

Discussion

A single bout of HIIT lowered both fasting and postprandial triglycerides in a healthy college-age population. HIIT produced significant results in a shorter time-frame, that is similar to ACSM exercise recommendations [10]. The low-volume resistance training did not lower fasting or postprandial triglycerides in this population.

Prior exercise elicits a decrease in postprandial triglycerides through various mechanisms. Increased activity of lipoprotein lipase (LPL), during and following exercise is one principal mechanism. LPL is responsible for hydrolyzing triglycerides from the blood, allowing cellular uptake of individual fatty acids. Resistance exercise does not increase LPL activity as effectively as aerobic exercise potentially explaining the lack of effect of the resistance trial [28, 29]

Skeletal muscle contractions induce increased LPL activity in skeletal muscle cells and LPL mRNA expression rises for up to eight hours following exercise, partially explaining the prolonged triglyceride lowering effect [30, 31]. Active recovery between sets in the resistance trial was not employed, limiting skeletal muscle contractions, which may have limited LPL activity.

Catecholamine release during and after exercise increases LPL activity in skeletal muscle [32]. Catecholamines are released more liberally in exercises of higher intensity and volume [33]. While the HIIT protocol provided high intensity bouts of exercise, the resistance protocol may have lacked sufficient intensity and volume to induce elevated catecholamine release.

Triglycerides and fatty acids are transported in the blood in very low density lipoproteins (VLDLs). LPL binds triglycerides in VLDLs for hydrolysis and uptake into cells. Circulating VLDL particles are larger and have a greater triglyceride load following exercise, increasing affinity for LPL which induces greater triglyceride clearance [34, 35]. Fewer, but more triglyceride dense VLDL particles are released from the liver the morning after aerobic exercise, further increasing affinity for LPL and triglyceride clearance [36]. Excess dietary carbohydrates are synthesized into fatty acids which are incorporated into triglycerides and transported in VLDLs. Exercise induces increased use of hepatic carbohydrates therefore lowering hepatic triglyceride and VLDL production and secretion, ultimately lowering blood triglycerides [37].

A sufficient energy deficit caused by exercise is necessary to experience a decrease in postprandial triglycerides. Multiple studies have partially or completely replaced exercise energy deficits and have found that the replacement eliminates the triglyceride lowering effect [6, 21, 38]. Both the present study and past research have noted lower fasting triglyceride levels in exercise trials further supporting an energy deficit as an important mediator [24, 25]. Fat oxidation, as indicated by respiratory exchange ratio, is elevated even at rest the morning following exercise. [24, 39]. Collectively, these results may indicate an acute period of negative energy balance that induces greater oxidation of fatty acids for cellular respiration and therefore more rapid clearance of triglycerides [40].

Pilot studies of the exercise trials in two subjects revealed that energy expenditure during the resistance protocol averaged 272 kcal while the HIIT protocol was 390 kcal. Past research indicates an energy expenditure range of 360 to 1,700 kilocalories is effective for attenuating postprandial triglycerides [24]. The resistance protocol employed is below this range indicating that a sufficient energy deficit may not have been produced to note the triglyceride lowering effect.

Hydrolyzed free fatty acids enter skeletal muscle cells and mitochondria by way of various fatty acid transport molecules including FAT/CD36 transporters [41]. FAT/CD36 is stimulated to translocate to the sarcolemma with less than two hours of moderate intensity exercise, increasing cellular uptake of free fatty acids [42]. Increased clearance of free fatty acids from the blood stream limits endogenous formation of triglycerides. Chronic exercise training (moderate intensity or HIIT) increases intracellular concentration of FAT/CD36 and mitochondria, increasing transmembrane transport of fatty acids and basal fat oxidation rates [41]. The test population was overwhelming well trained (97% participated in an hour or more of exercise per week) potentially explaining why we noted some decreases in triglycerides from fasting to postprandial however other mechanisms are most likely in effect.

ANOVA indicated a significant main effect of treatment (control vs. HIIT vs. resistance), but failed to show a significant interaction of treatment and time, meaning HIIT did not lower the difference between fasting and postprandial triglycerides. The change in triglycerides between fasting and postprandial for each trial was similar indicating that the exercise protocols had little effect on the interaction. HIIT did not significantly decrease the response to a fat load; instead, the postprandial triglycerides were lower because the fasting levels were lower.

All exercise was supervised to ensure proper execution by subjects. Scheduling difficulties led to variable timing of the exercise sessions and OFTT consumptions during the day which may have led to increased variability in triglyceride results. Some participants exercised in the early afternoon and consumed the OFTT early in the morning while others exercised in the late evening and consumed the OFTT at midday. Alcohol consumption greater than 24 hours prior to the OFTT may also have altered triglycerides and was not controlled between trials.

Future studies would benefit from a more thorough diet log, allowing for complete diet analysis of foods consumed twenty-four hours prior to the OFTT. Regulating alcohol intake for a greater period could decrease variability in fasting triglyceride levels. The peak in postprandial triglycerides may have been missed with only one postprandial measurement. Multiple postprandial sample collections would allow for greater confidence in peak determination and permit analysis of area under the curve, which has proven highly effective in previous research and may have better explained our results [43]. Measuring fasting

blood glucose would have been beneficial to ensure subjects compliance with fasting. Further research is necessary to determine why some subjects had decreased triglyceride levels from fasting to postprandial as this is not a previously reported anomaly.

Conclusion

HIIT training of four, 30 second sprints with four minutes of walking recovery between each sprint significantly lowered fasting and postprandial triglycerides before and after a high fat meal in a healthy college-age population. Low-volume resistance training totaling thirty minutes did not significantly alter fasting or postprandial response to a high fat meal in the same population. Future research into the minimum exercise duration and intensity required to elicit decreases in postprandial triglycerides in hyperlipidemic populations is necessary to provide easier exercise goals.

References

1. Kochanek, K.D., S.L. Murphy, J. Xu and E. Arias, (2014) Mortality in the United States, 2013. *NCHS Data Brief*, (178), 1-8.
2. Talayero, B.G. and F.M. Sacks, (2011) The role of triglycerides in atherosclerosis. *Current Cardiology Reports*, 13(6), 544-52. DOI: 10.1007/s11886-011-0220-3.
3. Sarwar, N., J. Danesh, G. Eiriksdottir, G. Sigurdsson, N. Wareham, S. Bingham, et al., (2007) Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. *Circulation*, 115(4), 450-8. DOI: 10.1161/CIRCULATIONAHA.106.637793.
4. Patsch, J.R., G. Miesenbock, T. Hopferwieser, V. Muhlberger, E. Knapp, J.K. Dunn, et al., (1992) Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state. *Arteriosclerosis Thrombus*, 12(11), 1336-45.
5. Maraki, M.I. and L.S. Sidossis, (2013) The latest on the effect of prior exercise on postprandial lipaemia. *Sports Medicine*, 43(6), 463-81. DOI: 10.1007/s40279-013-0046-9.
6. Burton, F.L., D. Malkova, M.J. Caslake and J.M. Gill, (2008) Energy replacement attenuates the effects of prior moderate exercise on postprandial metabolism in overweight/obese men. *International Journal of Obesity (London)*, 32(3), 481-9. DOI: 10.1038/sj.ijo.0803754.
7. Hurren, N.M., F.F. Eves and A.K. Blannin, (2011) Is the effect of prior exercise on postprandial lipaemia the same for a moderate-fat meal as it is for a high-fat meal? *British Journal of Nutrition*, 105(4), 506-16. DOI: 10.1017/S0007114510003995.
8. Zhang, J.Q., L.L. Ji, G. Nunez, S. Feathers, C.L. Hart and W.X. Yao, (2004) Effect of exercise timing on postprandial lipemia in hypertriglyceridemic men. *Canadian Journal of Applied Physiology*, 29(5), 590-603.
9. Lee, C.W., J.H. Wang, J.C. Hsieh, T.C. Hsieh and C.H. Huang, (2013) Effects of Combined Phase III and Phase II Cardiac Exercise Therapy for Middle-aged Male Patients with Acute Myocardial Infarction. *J Phys Ther Sci*, 25(11), 1415-20. DOI: 10.1589/jpts.25.1415.
10. Garber, C.E., B. Blissmer, M.R. Deschenes, B.A. Franklin, M.J. Lamonte, I.M. Lee, et al., (2011) American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Medical Science of Sports and Exercise*, 43(7), 1334-59. DOI: 10.1249/MSS.0b013e318213febf.
11. Miyashita, M. and K. Tokuyama, (2008) Moderate exercise reduces serum triacylglycerol concentrations but does not affect pre-heparin lipoprotein lipase concentrations after a moderate-fat meal in young men. *British Journal of Nutrition*, 99(5), 1076-82. DOI: 10.1017/S0007114507853438.
12. Sabaka, P., P. Kruzliak, D. Balaz, A. Komornikova, D. Celovska, G. Cammarota, et al., (2015) Effect of short term aerobic exercise on fasting and postprandial lipoprotein subfractions in healthy sedentary men. *Lipids Health and Disease*, 14, 151. DOI: 10.1186/s12944-015-0148-5.
13. Burgomaster, K.A., K.R. Howarth, S.M. Phillips, M. Rakobowchuk, M.J. Macdonald, S.L. McGee, et al., (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *Journal of Physiology*, 586(1), 151-60. DOI: 10.1113/jphysiol.2007.142109.

14. Gibala, M.J., J.B. Gillen and M.E. Percival, (2014) Physiological and health-related adaptations to low-volume interval training: influences of nutrition and sex. *Sports Medicine*, 44 Suppl 2, S127-37. DOI: 10.1007/s40279-014-0259-6.
15. Gibala, M.J., J.P. Little, M. van Essen, G.P. Wilkin, K.A. Burgomaster, A. Safdar, et al., (2006) Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *Journal of Physiology*, 575(Pt 3), 901-11. DOI: 10.1113/jphysiol.2006.112094.
16. Gillen, J.B., B.J. Martin, M.J. MacInnis, L.E. Skelly, M.A. Tarnopolsky and M.J. Gibala, (2016) Twelve Weeks of Sprint Interval Training Improves Indices of Cardiometabolic Health Similar to Traditional Endurance Training despite a Five-Fold Lower Exercise Volume and Time Commitment. *PLoS One*, 11(4), e0154075. DOI: 10.1371/journal.pone.0154075.
17. Little, J.P., A. Safdar, G.P. Wilkin, M.A. Tarnopolsky and M.J. Gibala, (2010) A practical model of low-volume high-intensity interval training induces mitochondrial biogenesis in human skeletal muscle: potential mechanisms. *Journal of Physiology*, 588(Pt 6), 1011-22. DOI: 10.1113/jphysiol.2009.181743.
18. Thackray, A.E., L.A. Barrett and K. Tolfrey, (2013) Acute high-intensity interval running reduces postprandial lipemia in boys. *Medical Science of Sports and Exercise*, 45(7), 1277-84. DOI: 10.1249/MSS.0b013e31828452c1.
19. Chu, A., Y.N. Boutcher and S.H. Boutcher, (2016) Effect of acute interval sprinting exercise on postprandial lipemia of sedentary young men. *Journal of Exercise and Nutritional Biochemistry*, 20(1), 9-14. DOI: 10.20463/jenb.2016.03.20.1.7.
20. Freese, E.C., N.H. Gist, R.M. Acitelli, W.J. McConnell, C.D. Beck, D.B. Hausman, et al., (2015) Acute and chronic effects of sprint interval exercise on postprandial lipemia in women at-risk for the metabolic syndrome. *Journal of Applied Physiology*, 118(7), 872-9. DOI: 10.1152/jappphysiol.00380.2014.
21. Freese, E.C., A.S. Levine, D.P. Chapman, D.B. Hausman and K.J. Cureton, (2011) Effects of acute sprint interval cycling and energy replacement on postprandial lipemia. *Journal of Applied Physiology*, 111(6), 1584-9. DOI: 10.1152/jappphysiol.00416.2011.
22. Allen, E., P. Gray, A. Kollias-Pearson, E. Oag, K. Pratt, J. Henderson, et al., (2014) The effect of short-duration sprint interval exercise on plasma postprandial triacylglycerol levels in young men. *Journal of Sports Science*, 32(10), 911-6. DOI: 10.1080/02640414.2013.865254.
23. Pafili, Z.K., G.C. Bogdanis, N.V. Tsetsonis and M. Maridaki, (2009) Postprandial lipemia 16 and 40 hours after low-volume eccentric resistance exercise. *Medical Science of Sports and Exercise*, 41(2), 375-82. DOI: 10.1249/MSS.0b013e31818703ec.
24. Pettitt, D.S., S.A. Arngrimsson and K.J. Cureton, (2003) Effect of resistance exercise on postprandial lipemia. *Journal of Applied Physiology*, 94(2), 694-700. DOI: 10.1152/jappphysiol.00377.2002.
25. Singhal, A., J.L. Trilk, N.T. Jenkins, K.A. Bigelman and K.J. Cureton, (2009) Effect of intensity of resistance exercise on postprandial lipemia. *Journal of Applied Physiology*, 106(3), 823-9. DOI: 10.1152/jappphysiol.90726.2008.
26. Zafeiridis, A., E. Goloi, A. Petridou, K. Dipla, V. Mougios and S. Kellis, (2007) Effects of low- and high-volume resistance exercise on postprandial lipaemia. *British Journal of Nutrition*, 97(3), 471-7. DOI: 10.1017/S0007114507336787.
27. Panz, V.R., F.J. Raal, J. Paiker, R. Immelman and H. Miles, (2005) Performance of the CardioChek PA and Cholestech LDX point-of-care analysers compared to clinical diagnostic laboratory methods for the measurement of lipids. *Cardiovasc J S Afr*, 16(2), 112-7.
28. Kiens, B. and H. Lithell, (1989) Lipoprotein metabolism influenced by training-induced changes in human skeletal muscle. *J Clin Invest*, 83(2), 558-64. DOI: 10.1172/JCI113918.
29. Kiens, B., H. Lithell, K.J. Mikines and E.A. Richter, (1989) Effects of insulin and exercise on muscle lipoprotein lipase activity in man and its relation to insulin action. *J Clin Invest*, 84(4), 1124-9. DOI: 10.1172/JCI114275.
30. Seip, R.L., T.J. Angelopoulos and C.F. Semenkovich, (1995) Exercise induces human lipoprotein lipase gene expression in skeletal muscle but not adipose tissue. *Am J Physiol*, 268(2 Pt 1), E229-36.

31. Seip, R.L., K. Mair, T.G. Cole and C.F. Semenkovich, (1997) Induction of human skeletal muscle lipoprotein lipase gene expression by short-term exercise is transient. *Am J Physiol*, 272(2 Pt 1), E255-61.
32. Burns, S.F., H. Corrie, E. Holder, T. Nightingale and D.J. Stensel, (2005) A single session of resistance exercise does not reduce postprandial lipaemia. *Journal of Sports Science*, 23(3), 251-60. DOI: 10.1080/02640410410001730142.
33. Kraemer, W.J., S.J. Fleck, C.M. Maresh, N.A. Ratamess, S.E. Gordon, K.L. Goetz, et al., (1999) Acute hormonal responses to a single bout of heavy resistance exercise in trained power lifters and untrained men. *Can J Appl Physiol*, 24(6), 524-37.
34. Al-Shayji, I.A., M.J. Caslake and J.M. Gill, (2012) Effects of moderate exercise on VLDL(1) and Intralipid kinetics in overweight/obese middle-aged men. *Am J Physiol Endocrinol Metab*, 302(3), E349-55. DOI: 10.1152/ajpendo.00498.2011.
35. Ghafouri, K., J. Cooney, D.K. Bedford, J. Wilson, M.J. Caslake and J.M. Gill, (2015) Moderate Exercise Increases Affinity of Large Very Low-Density Lipoproteins for Hydrolysis by Lipoprotein Lipase. *J Clin Endocrinol Metab*, 100(6), 2205-13. DOI: 10.1210/jc.2015-1196.
36. Harrison, M., N.M. Moyna, T.W. Zderic, D.J. O'Gorman, N. McCaffrey, B.P. Carson, et al., (2012) Lipoprotein particle distribution and skeletal muscle lipoprotein lipase activity after acute exercise. *Lipids Health Dis*, 11, 64. DOI: 10.1186/1476-511X-11-64.
37. Potts, J.L., R.M. Fisher, S.M. Humphreys, S.W. Coppack, G.F. Gibbons and K.N. Frayn, (1991) Peripheral triacylglycerol extraction in the fasting and post-prandial states. *Clin Sci (Lond)*, 81(5), 621-6.
38. Emerson, S.R., S.P. Kurti, B.S. Snyder, K. Sitaraman, M.D. Haub and S.K. Rosenkranz, (2016) 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*, 13, 26. DOI: 10.1186/s12970-016-0137-8.
39. Tsetsonis, N.V. and A.E. Hardman, (1996) Reduction in postprandial lipemia after walking: influence of exercise intensity. *Med Sci Sports Exerc*, 28(10), 1235-42.
40. Melby, C., C. Scholl, G. Edwards and R. Bullough, (1993) Effect of acute resistance exercise on postexercise energy expenditure and resting metabolic rate. *J Appl Physiol (1985)*, 75(4), 1847-53.
41. Smith, B.K., A. Bonen and G.P. Holloway, (2012) A dual mechanism of action for skeletal muscle FAT/CD36 during exercise. *Exerc Sport Sci Rev*, 40(4), 211-7. DOI: 10.1097/JES.0b013e31825eb263.
42. Bradley, N.S., L.A. Snook, S.S. Jain, G.J. Heigenhauser, A. Bonen and L.L. Spriet, (2012) Acute endurance exercise increases plasma membrane fatty acid transport proteins in rat and human skeletal muscle. *Am J Physiol Endocrinol Metab*, 302(2), E183-9. DOI: 10.1152/ajpendo.00254.2011.
43. Freese, E.C., N.H. Gist and K.J. Cureton, (2014) Effect of prior exercise on postprandial lipemia: an updated quantitative review. *J Appl Physiol (1985)*, 116(1), 67-75. DOI: 10.1152/japphysiol.00623.2013.