The Influence of Caffeine and Genistein Ingestion on Plasma Triglycerides Status in Male Soccer Players

Karol Mateusz Danielik (✉ karoldanielik@gmail.com)

University School of Physical Education in Wrocław: Akademia Wychowania Fizycznego we Wrocławiu
<https://orcid.org/0000-0002-1906-1853>

Karolina Łoźna

Wrocław University of Environmental and Life Sciences: Uniwersytet Przyrodniczy we Wrocławiu

Monika Bronkowska

Wrocław University of Environmental and Life Sciences: Uniwersytet Przyrodniczy we Wrocławiu

Research article

Keywords: caffeine, genistein, lipolysis, postprandial lipemia, soccer players

Posted Date: January 13th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-141830/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background

Scientific literature provides more and more information on compounds supporting fat metabolism. More research is needed to fully characterize the effects of compounds that increase concentration triglycerides. The aim of our study was to test the influence of caffeine and genistein consumption on triglyceride status in athletes.

Methods

Fourteen Polish adult sub-elite soccer players (23.1 ± 2.1 years; 179.6 ± 8.5 cm; 74.1 ± 8.5 kg; $12.4 \pm 3.8\%$ body fat) were accepted for research. The athletes for this randomized, double-blind study were joining fasted. After consuming a standardized milk meal, participants took caffeine (400 mg), genistein (120 mg), or placebo (400 mg of vitamin C). Athletes ate the compound if it saw a sequentially increase and decrease in triglyceride concentration after eating the test meal. Participants had their glucose levels measured every 15 minutes with a glucometer (Accu-Chek Active) and their triglyceride levels with CardioChek PA. The subject was terminating the study when he had sequentially increased and decreased triglyceride concentration after the compound. If no such reaction occurred, measurements were taken for up to 90 minutes. In order to determine statistically significant differences between the variables, non-parametric Kruskal-Wallis and U Mann-Whitney tests were used. The significance level of $\alpha = 0.05$ was adopted for both of these tests. The relationships between the variables were determined using the Spearman R correlation. The correlations were statistically significant at $p \leq 0.100$.

Results

A statistically significant difference was demonstrated between the initial and maximum triglyceride concentration after caffeine consumption compared to placebo ($p = 0.049$). After consuming caffeine, the athletes had a significant difference in triglyceride concentration at 30 min ($p = 0.018$) and 60 min ($p = 0.036$) compared to placebo. In the group taking genistein, only a statistically significant difference was noticed compared to placebo only in 60 min ($p = 0.029$).

Conclusion

Our research results show that after acute caffeine and genistein supplementation, triglyceride concentration increase in soccer players.

Background

The main energy substrates of athletes during exercise are carbohydrates and fats. In addition to the energy function, the right amount of these ingredients is designed to save the use of body proteins as an energy source. In the case of an insufficient amount of carbohydrates, free fatty acids (FFA) become the main energy substrate. They are released from fat cells and enter the plasma through the lipolysis

process. Lipolysis is defined as the process of enzymatic hydrolysis of triacylglycerols contained in fat cells [1]. Adipose tissue lipolysis takes place in order to maintain the supply of energy substrates in the state of energy deficit. This process is preceded by many biochemical reactions regulated by functional proteins [2]. The lipolysis reaction takes place in several stages. The first part of this process is the detachment of the fatty acid from the α position. The next step is to remove another acid molecule from the α 'position. At the very end of the hydrolysis, the last fatty acid residue from monoacylglycerol is separated from the β position [3]. The entire process is stimulated by enzymes. The activity of these fatty enzymes (lipases) is stimulated, inter alia, by glucagon, adrenaline and glucocorticosteroids. These compounds regulate adenylate cyclase. Its content in adipocytes directly affects the concentration of cyclic adenosine monophosphate (cAMP), which affects protein kinase (PKA). Thus, increasing or decreasing the level of cAMP regulates the lipolysis process [4,5]. There are many active chemical compounds in food that may affect adipose tissue by inducing, for example, lipolysis, or completely affect the apoptosis of adipocytes [6]. The results of scientific studies show the effect of given lipotropic factors based on *in vitro studies* conducted on murine models of adipose tissue [5,7–9] as well as *in vivo studies* [7,10–13]. Regulation of lipolysis occurs at many levels in response to changing metabolic conditions and nutrient intake. A relationship has been found between the rate of lipolysis and the body weight and size of fat cells [14, 15]. Chronic exposure to extreme physiological conditions such as obesity or starvation also induces metabolic adaptations that include changes in lipolysis. There is increasing evidence in the scientific literature that indicates that certain metabolically active nutrients in the diet can regulate lipolysis [16, 17]. There is a need to confirm the action of lipotropic agents *in vivo* and to provide information on the dose of a given component that produces a biological effect. More research is needed to fully characterize the effects of caffeine and genistein on the plasma triglyceride status of athletes. Therefore, the aim of this study was to examine the effects of caffeine and genistein consumption on changes in triglyceride concentrations.

Study Methods

Participants

The study in question was a randomized, double-blind study involving 14 Polish adult sub-elite soccer players (23.1 ± 2.1 years, 179.6 ± 8.5 cm, 74.1 ± 8.5 kg) from different teams. Anyone could participate in this study twice. A total of 23 trials were performed, of which 9 people took 400 mg of caffeine and 8 people took 120 mg of genistein. The placebo group (400 mg vitamin C) consisted of 6 men. Criteria for inclusion in the study:

- male gender,
- age ranging from 18 to 30 years,
- physically active soccer players,
- low or optimal level of adipose tissue for health (5 - 22% ^[18]).

Certain anthropometric features of the studied participants increased the homogeneity of the group. The characteristics of the group are presented in Table 1.

Study design

The study was conducted at the Department of Human Nutrition, Faculty of Biotechnology and Food Sciences at the University of Life Sciences in Wrocław. At each stage, the test was carried out in a sterile, closed room and in accordance with Good Hygiene Practice. Participants were informed about the purpose of the research and the possibility of opting out of the experiment at any stage. Consent to participate in the clinical trial was declared by a handwritten signature on the declaration. Laboratory tests were carried out in the period from May 14 to June 6, 2019.

Procedures

The study started after receiving the approval of the Bioethics Committee at the Medical University of Piastów Śląskich in Wrocław - 239/2019 of March 18, 2019.

Anthropometric measurements

The men were informed about the necessity to come on an empty stomach. Using the AccunIQ BC380 analyzer, basic anthropometric measures and body composition were determined, including : Total Fat Mass (PB), Viscellular Fat Area (VFA), Muscle Mass (SSM), Total Body Water (TBW).

24-hour dietary interview

On the day of the measurements, the participants were interviewed regarding food consumption in the last 24 hours. The analysis of 24-hour nutritional interviews took into account: energy value, protein content, carbohydrates, total fat, monounsaturated fatty acids, saturated fatty acids, polyunsaturated fatty acids, dietary fiber, vitamins B1, B2, B6, magnesium and calcium.

Consumption of a standardized milk meal

Studies [19–21] have shown that saturated fatty acids (SFA) have different effects on postprandial lipemia compared to monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA). The effect of increasing the concentration of triglycerides in the blood is influenced not only by the amount and type of dietary fat, but also by other dietary components, such as: fiber, glucose, starch and alcohol [22–24]. A standardized milk meal was prepared for the participants (based on the data from the literature) [19–21,23,24]. After initial measurements, the examined participants consumed a dairy product - 70 ml of cream, 18% UHT. It did not contain fiber, protein, carbohydrates or other ingredients, so that the effect was noticeable as soon as possible. The increase in fatty acid concentration after consuming the test meal was supposed to take place in a short time. Before eating meals, the concentration of glucose and triglycerides in the capillary blood of the athletes was measured.

The CardioChek PA device was used to test blood triglyceride concentrations. An Accu-Chek Active glucometer was used to measure glucose concentrations. Thereafter, the concentration of these two biochemical markers was monitored every 15 minutes. The participant was qualified to the next stage, compound consumption, if the athlete had been observed to have had, respectively, an increase and a decrease in triglyceride concentration. If the triglyceride concentration fluctuated, measurements were taken up to a maximum of 90 minutes.

Active Compound Ingestion

Study participants consumed one compound: caffeine, genistein, or placebo. Due to the small differences in body weight, the men took the same dose of the active compounds. In the case of caffeine, it was 400 mg, and in the case of genistein, 120 mg. The placebo was vitamin C at a dose of 400 mg. The concentration of glucose and triglycerides in the capillary blood was measured every 15 minutes. The study scheme is presented in Fig. 1. The participant finished the study when an increase and a decrease in triglyceride concentration, respectively, had been observed. If no such reaction occurred, measurements were taken up to 105 minutes.

Analysis of results

In the created database, the exact values of the concentrations of selected blood chemistry indicators were included and the percentage increase in relation to the initial values was calculated. This made it possible to test the effectiveness of these compounds depending on their type and the time during which this effect is noticeable.

The rate of increase in triglyceride concentration is a physical quantity representing the change in triglyceride concentration per unit time $[(\text{mg} / \text{dL}) / \text{min}]$. The triglyceride concentrations of all participants after consuming the cream and the compound are presented in Tables 2 and 3 consecutively. The following scheme was followed for each participant (example based on data obtained for 1 participant):

- Two graphs of the change in triglyceride concentration over time after consuming the test meal and after administration of the active compound (Figures 1 and 2).
- A trend line was drawn and described with a polynomial so that the curve fitting coefficient R^2 was close to 1. The more the polynomial plot fits the points marked on the coordinate system, the closer the R value is to 1.
- The tangent to the polynomial was determined at the point from which a rapid increase in triglyceride concentration was observed. A tangent is a geometric interpretation of the derivative of a polynomial function. In the graph, 1 tangent was assigned to the point x , equal to 0 min, and in the second graph to the point x , equal to 15 min.
- The rate of increase in triglyceride concentration was calculated. The rate of increase in triglyceride concentration was measured using the $\tan$ tangent, i.e., the angle of its inclination to the x axis.

Statistical analysis

The research results were collected in an Excel spreadsheet. The statistical analysis of the results was performed in Statistica 13.1 PL. In order to test the compliance of the data with the normal distribution, the Shapiro-Wilk test was used. The mean, standard deviation for parametric data, median and lower quartile (Q1) and upper quartile (Q3) for non-parametric data were calculated. In order to determine statistically significant differences between the variables, non-parametric Kruskal-Wallis and U Mann-Whitney tests were used. The significance level of $\alpha = 0.05$ was adopted for both of these tests. The relationships between the variables were determined using the Spearman R correlation. The correlations were statistically significant at $p \leq 0.100$.

Results

There were no statistically significant differences in the blood triglyceride growth rate ($p = 0.282$) or the difference in triglyceride concentrations after ingestion of genistein compared to placebo ($p = 0.228$). It was noticed in the study that there was a statistically significant difference in glucose concentration after the consumption of the compound ($p = 0.043$) (Table 2).

The difference in triglyceride concentrations after caffeine consumption compared to placebo was statistically significant ($p = 0.049$). No difference in rate was found concerning increase in triglycerides after caffeine consumption compared to placebo ($p = 0.775$) (Table 3)

In this study, it was shown that the physiological effect after ingestion of the compound is time dependent. Table 4 shows a comparison of caffeine and placebo with respect to the time in which the increase in triglyceride concentrations was observed after the consumption of compounds. It was observed that a statistically significant difference in the increase in triglycerides after caffeine consumption compared to placebo occurred at 30 ($p = 0.018$) and 60 ($p = 0.036$) minutes. On the other hand, ingestion of genistein resulted in a statistically significantly greater increase in triglyceride concentrations after consuming the compound at 60 minutes compared to the consumption of placebo ($p = 0.029$). In the remaining time values, no differences between these groups were observed (Table 5).

There was a statistically significant negative correlation between the amount of carbohydrates consumed on the day before the study and the time when the increase in triglyceride concentrations was observed after consuming the compound in the genistein group ($r = -0.912$) and placebo ($r = -0.626$).

Moreover, in the group taking genistein, it was shown that the consumption of polyunsaturated fatty acids the day before the study was statistically significantly correlated with the time during which the increase in triglyceride concentration was observed after the consumption of compounds ($r = -0.758$). The present study also showed a statistically significant difference in triglyceride concentrations after consumption of the test meal before genistein administration compared to caffeine ($p = 0.036$). In the group taking caffeine, the body weight of the participants was statistically significantly correlated with the rate of increase in blood triglyceride concentration after consuming the test meal (-0.683).

Discussion

The main factor influencing postprandial lipemia is the amount of fat present in a meal. Other variables such as the presence and type of carbohydrates, alcohol and other nutrients are widely described in the scientific literature [25–29]. The triglyceride response after a fatty meal is also influenced by gender, physiological status, and several other causes, the details of which are not yet fully understood [30]. In the author's own study, people taking caffeine showed a negative correlation between body weight and the rate of increase in triglyceride concentration after consuming a test meal. Similar conclusions were shown in the work of Tiihonen et al. [31]. The authors of this study showed that the concentration of triglycerides after a standardized meal was statistically significantly lower in the normolipidemic group compared to the obese or hyperlipidemic groups. In addition, the maximum concentration of free fatty acids in the blood was found much earlier in people with normolipidemia than in the other two groups.

Studies conducted among women showed a less intense postprandial lipid response than in men [32]. This proves that men are characterized by an increased ability to remove triglycerides from the cardiovascular system. Tolerance of fatty meals usually decreases with age but can also be related to weight gain or the phase of the menopause. Peddie et al. suggest that increased physical activity, such as running or cycling, and even walking, can significantly reduce fasting triglycerides and the postprandial triglyceride response [32]. In our study, people who consumed caffeine showed a 4-fold higher rate of triglyceride growth after consuming cream compared to placebo. Men in this group were characterized by a greater lipid response after consuming a fatty product. However, this result was not statistically significant ($p = 0.113$). It was observed that the amount of carbohydrate consumed the day before the study statistically significantly correlated with the increase in triglyceride concentration after consuming the compound in the placebo group (-0.626) and genistein (-0.912). After the results obtained, it can be suggested that a high intake of carbohydrates, possibly including fiber, may extend the time when genistein becomes active. Our study also showed that the consumption of polyunsaturated fatty acids influenced the time in which the increase in triglyceride concentration was observed after ingestion of genistein. In addition, no more statistically significant relationships were observed between the intake of macronutrients on the day before the study and the difference in triglyceride concentrations.

The lipolytic effects of caffeine and other tea- and coffee-derived methylxanthines are well recognized and reported in the scientific literature. These compounds stimulate lipolysis by increasing the concentration of cAMP in cells due to two main mechanisms. The first is the antagonism of A1-adenosine receptors [33]. These receptors dominate in differentiated mature adipocytes, where they inhibit the activity of adenylate cyclase and the lipolysis process [34]. Antagonism of A1 receptors results in inactivation of adenylate cyclase activity and increased lipolysis. Methylxanthines inhibit phosphodiesterase activity, preventing the breakdown of cAMP, and stimulate lipolysis in fat cells [35]. Caffeine is a widely used stimulant that has well-established benefits for athletic performance. Its ergogenic effect is attributed to the increased release of endorphins, improved neuromuscular function, improved alertness, and decreased perception of fatigue during exercise [36]. In recommendations for athletes, information on the consumption of caffeine in the amount of 3-6 mg of the compound for each

kilogram of body weight can be found. The concentration of caffeine increases within 15-45 minutes of being absorbed into the body. The maximum concentration is approximately 60 minutes after ingestion [37]. In experiments involving humans, it has been shown that caffeine increases lipid turnover and the concentration of non-esterified free fatty acids in the blood serum [7, 10, 11, 38]. Consumption of methylxanthines may also contribute to weight loss or maintenance [39]. In the work of Kobayashi-Hattori et al. [9] it was shown that administering caffeine to rats in a dose of 5 mg / kg for 21 days gradually decreased the weight and the percentage of adipose tissue in Sprague-Dawley rats. Based on the results obtained in this study, it can be suggested that in the case of a high-fat diet, caffeine consumption enhanced lipolysis mediated by catecholamines. In a study by Benowitz et al. [40], caffeine was administered in various doses to 12 healthy volunteers, 6 men and 6 women. Benowitz et al. Proved that the consumption of caffeine compared to placebo increased the amount of free fatty acids in a significant way. However, the authors of the same study did not show any statistically significant differences between doses of 2 mg / kg and 4 mg / kg of body weight. Another study showed an age difference in increasing the concentration of FFA in the blood plasma. Younger men were more susceptible to caffeine and significantly increased the concentration of FFA compared to the elderly [38]. Two studies have shown that the time when the increase in FFA concentration was observed after caffeine consumption was 30-60 minutes [11,40]. These data confirm the results obtained during our study. It is impossible to compare the results of our work with the results of the work of Benowitz et al. [40]. In this study, the concentration of FFA was monitored every 30 minutes.

Genistein isoflavone is also a compound that influences the release of free fatty acids from adipocytes into the plasma. It has been proved in animal models that soy isoflavones can regulate many pathways in the body, including the influence of 5'AMP-activated kinase. Affecting this compound may facilitate the process of reducing body fat [41]. In this study it was determined that a dose of 20-200 μ mol influences the condition of adipocytes leading to their apoptosis. This process is stimulated by inactivation of 5'AMP-activated kinase (AMPK). Mori et al. [42] proved that the consumption of soy isoflavones in a dose of 100 mg / day for nearly 6 months reduces body weight and reduces body fat. Weight loss has been observed in Asian people who consume fermented soy products. The participants of the study by Cha et al. and Cha et al. consumed two varieties of soybean paste: doenjang and kochujang for 3 months [43,44]. *In vitro* studies have shown that genistein also blocks adipogenesis by inactivating tyrosine kinase. The supply of genistein along with the diet may stimulate the lipolysis process in humans, potentially contributing to the reduction of adipose tissue mass [45]. Nogowski et al. [8] studied the effect of genistein on lipid metabolism in rats after ovariectomy. It was shown that genistein administered with feed increased the concentration of free fatty acids in the blood plasma. The effect was tested at a dose of 20-300 μ mol / l for a period of 14 days. The severity of the reaction was observed after increasing to a dose of 100 μ mol / l. The efficiency of genistein did not increase statistically significantly after the dose of 100 μ mol / l was exceeded. There is no information in the scientific literature regarding the influence of genistein supplementation on the lipolysis process *in vivo*. In our own study, genistein supplementation in a dose of 120 mg was tested. There were no statistically significant differences in triglyceride concentrations after ingestion of genistein compared to placebo.

The in-house study suggests that genistein increases free fatty acid concentrations and potentially affects body fat at 60 minutes compared to placebo. In our study, a strong correlation (-0.905) was found between body weight and the rate of increase in triglyceride concentration after ingestion of genistein. The strong correlation obtained shows that as body weight increases, genistein activity occurs later. Tiihonen et al. proved the correlation between body weight, fasting fatty acid concentration and postprandial lipemia similarly to this study [31].

In our own study, it was shown that there are no differences in the increase in triglyceride concentrations after caffeine consumption compared to genistein in each monitored sample. The lack of differences between caffeine and genistein consumption may be due to many factors affecting the postprandial lipid response. Our study showed a statistically significant difference in triglyceride concentrations after consuming a test meal before genistein administration compared to caffeine. A statistically significant difference in triglyceride concentrations after consumption of the test meal could have impaired the detection of differences in triglyceride concentrations after ingestion of the active compound.

Limitations

There are a few limitations of this study. The difficulty was that it was not possible to accurately estimate the ration of the meals consumed the day before the study. Another limitation in this study was the small sample size and the lack of diverse groups. Apart from methodological limitations, there were also difficulties for the participants. Participants had to consume a fatty test meal and endure about a 5-hour fast. More research is needed to include more people. Future studies should compare the effects of genistein, caffeine and placebo in each athlete separately. This study looked at the effect in a homogeneous group. Subsequent research should take into account other sports disciplines and the female gender.

Conclusion

In our own study, it was proved that caffeine and genistein influenced the process of increasing the concentration of triglycerides in the blood. These compounds increased the concentration of triglycerides in the capillary blood compared to placebo. Caffeine started its activity from 30 minutes. A significantly greater increase in the concentration of triglycerides in the blood plasma was observed at 30 and 60 minutes after the consumption of the compound compared to placebo. For genistein, the physiological effect was only noticed at 60 minutes compared to placebo. Based on the data from the nutritional history of consumption 24 hours before the study, it was proved that the amount of polyunsaturated fatty acids influenced the time in which the increase concentrations triglycerides was intensified. The amount of carbohydrates consumed on the day before the test had an influence on the increase in the concentration of free fatty acids after the consumption of the compound. In addition, a negative correlation was found between body weight and the rate of increase in the concentration of triglycerides in the blood plasma after consuming the test meal. More nutritional factors have not been associated

with the change in triglyceride concentrations and the lipid response following a meal with a specific fat content.

Abbreviations

cAMP: cyclic adenosine monophosphate

PKA: protein kinase

PB: Total Fat Mass

VFA: Viscellular Fat Area

SSM: Muscle Mass

TBW: Total Body Water

SFA: saturated fatty acids

MUFA: monounsaturated fatty acids

PUFA: polyunsaturated fatty acids

MAX: the highest triglyceride concentration

BEG: initial triglyceride concentration

FFA: free fatty acids

AMPK: 5'AMP-activated kinase

Declarations

Ethics approval and consent to participate

The subjects provided written informed consent to participate in the present study. This study was approved by the Bioethics Committee at the Medical University of Piastów Śląskich in Wrocław - 239/2019 of March 18, 2019.

Consent for publication

Not applicable.

Availability of supporting data

Please contact corresponding author for additional data, by emailing the corresponding author (karoldanielik@gmail.com)

Competing interests

The authors have no conflict of interest.

Funding

The study has been carried out as part of university research activity and there are no financial reasons which may lead to a conflict of interest.

Author's Contributions

This experiment was performed at the University of Life Sciences in Wrocław. K. D. and K. Ł. were responsible for the conception and design of the experiment. M. B. corrected the manuscript. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Acknowledgments

The authors would like to thank the soccer players involved in this research for their participation and cooperation.

Author information

Affiliations

Department of Biological and Motor Basis of Sport, University of Physical Education, Wrocław (Poland)

Karol Danielik

Department of Human Nutrition, Faculty of Biotechnology and Food Sciences at the University of Life Sciences in Wrocław

Karolina Łoźna, Monika Bronkowska

Corresponding author

Correspondence to Karol Danielik.

References

1. Kolditz CI, Langin D. Adipose tissue lipolysis: Current Opinion in Clinical Nutrition and Metabolic Care. 2010; 13: 377–81.

2. Zechner R, Zimmermann R, Eichmann TO, Kohlwein SD, Haemmerle G, Lass A, et al. FAT SIGNALS - Lipases and Lipolysis in Lipid Metabolism and Signaling. *Cell Metabolism*. 2012; 15: 279–91.
3. Bolsoni-Lopes A, Alonso-Vale, MIC. Lipolysis and lipases in white adipose tissue - An update. *Arch Endocrinol Metab*. 2015; 59: 335–42.
4. Holm C. Molecular mechanisms regulating hormone-sensitive lipase and lipolysis. *Biochemical Society Transactions*. 2003; 31: 5.
5. Duncan RE, Ahmadian M, Jaworski K, Sarkadi-Nagy E, Sul HS. Regulation of Lipolysis in Adipocytes. *Annu Rev Nutr*. 2007; 27: 79-101.
6. Kim J, Park J, Lim K. Nutrition Supplements to Stimulate Lipolysis: A Review in Relation to Endurance Exercise Capacity. *Journal of Nutritional Science and Vitaminology, J Nutr Sci Vitaminol*. 2016; 62: 141–61.
7. Ryu S, Choi SK, Joung SS, Suh H, Cha YS, Lee S, et al. Caffeine as a lipolytic food component increases endurance performance in rats and athletes. *J Nutr Sci Vitaminol*. 2001; 47: 139–46.
8. Nogowski L, Maćkowiak P, Kandulska K, Szkudelski T, Nowak KW. Genistein-Induced Changes in Lipid Metabolism of Ovariectomized Rats. *Ann Nutr Metab*. 1998; 42: 360–6.
9. Kobayashi-Hattori K, Mogi A, Matsumoto Y, Takita T. Effect of Caffeine on the Body Fat and Lipid Metabolism of Rats Fed on a High-Fat Diet. *Bioscience, Biotechnology, and Biochemistry*. 2005; 69: 2219–23.
10. Kim TW, Shin YO, Lee JB, Min YK, Yang HM. Effect of caffeine on the metabolic responses of lipolysis and activated sweat gland density in human during physical activity. *Food Sci Biotechnol*. 2010; 19: 1077–81.
11. Acheson KJ, Gremaud G, Meirim I, Montigon F, Krebs Y, Fay LB, et al. Metabolic effects of caffeine in humans: lipid oxidation or futile cycling? *The American Journal of Clinical Nutrition*. 2004; 79: 40–6.
12. Bloomer RJ, Fisher-Wellman KH, Hammond KG, Schilling BK, Weber AA, Cole BJ. Dietary supplement increases plasma norepinephrine, lipolysis, and metabolic rate in resistance trained men. *Journal of the International Society of Sports Nutrition*. 2009; 6: 10.
13. Dalbo VJ, Roberts MD, Stout JR, Kerkick CM. Acute effects of ingesting a commercial thermogenic drink on changes in energy expenditure and markers of lipolysis. *Journal of the International Society of Sports Nutrition*. 2008; 5: 6.
14. Laurencikiene J, Skurk T, Kulyté A, Hedén P, Aström G, Sjölín E, Rydén M, Hauner H, Arner P. Regulation of lipolysis in small and large fat cells of the same subject. *J Clin Endocrinol Metab*. 2011 Dec;96(12):E2045-9.
15. Cifuentes M, Albala C, Rojas CV. Differences in lipogenesis and lipolysis in obese and non-obese adult human adipocytes. *Biol Res*. 2008;41(2):197-204.
16. Arner P. Human fat cell lipolysis: Biochemistry, regulation and clinical role. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2005; 19: 471–82.

17. Langin D. Control of fatty acid and glycerol release in adipose tissue lipolysis. *Comptes Rendus Biologies*. 2006; 329: 598–607.
18. Turocy PS, DePalma BF, Horswill CA, Laquale KM, Martin TJ, Perry AC, et al. National Athletic Trainers' Association Position Statement: Safe Weight Loss and Maintenance Practices in Sport and Exercise. *J Athl Train*. 2011; 46: 322–36.
19. Jackson KG, Robertson MD, Fielding BA, Frayn KN, Williams CM. Olive oil increases the number of triacylglycerol-rich chylomicron particles compared with other oils: an effect retained when a second standard meal is fed. *The American Journal of Clinical Nutrition*. 2002; 76: 942–9.
20. Jackson KG, Robertson MD, Fielding BA, Frayn KN, Williams CM. Measurement of apolipoprotein B-48 in the Svedberg flotation rate (Sf) > 400, Sf 60–400 and Sf 20–60 lipoprotein fractions reveals novel findings with respect to the effects of dietary fatty acids on triacylglycerol-rich lipoproteins in postmenopausal women. *Clinical Science*. 2002; 103: 227–37.
21. Jackson KG, Wolstencroft EJ, Bateman PA, Yaqoob P, Williams CM. Greater enrichment of triacylglycerol-rich lipoproteins with apolipoproteins E and C-III after meals rich in saturated fatty acids than after meals rich in unsaturated fatty acids. *The American Journal of Clinical Nutrition*. 2005;81:25–34.
22. Cohen JC, Berger GM. Effects of glucose ingestion on postprandial lipemia and triglyceride clearance in humans. *Journal of lipid research*. 1990;31(4):597-602.
23. van Tol A, S. van der Gaag M, M. Scheek L, van Gent T, FJ Hendriks H. Changes in postprandial lipoproteins of low and high density caused by moderate alcohol consumption with dinner. *Atherosclerosis*. 1998;141:S101–3.
24. Williams CM. Dietary interventions affecting chylomicron and chylomicron remnant clearance. *Atherosclerosis*. 1998;141:S87–92.
25. Van de Wiel A. The effect of alcohol on postprandial and fasting triglycerides. *Int J Vasc Med*. 2012;2012:862504.
26. Sun L, Tan KWJ, Lim JZ, Magkos F, Henry CJ. Dietary fat and carbohydrate quality have independent effects on postprandial glucose and lipid responses. *Eur J Nutr*. 2018;57:243–50.
27. Dubois C, Armand M, Azais-Braesco V, Portugal H, Pauli AM, Bernard PM, et al. Effects of moderate amounts of emulsified dietary fat on postprandial lipemia and lipoproteins in normolipidemic adults. *The American Journal of Clinical Nutrition*. 1994;60:374–82.
28. Dubois C, Beaumier G, Juhel C, Armand M, Portugal H, Pauli AM, et al. Effects of graded amounts (0–50 g) of dietary fat on postprandial lipemia and lipoproteins in normolipidemic adults. *The American Journal of Clinical Nutrition*. 1998;67:31–8.
29. Purcell R, Latham S, Botham K, Hall W, Wheeler-Jones C. High-fat meals rich in EPA plus DHA compared with DHA only have differential effects on postprandial lipemia and plasma 8-isoprostane F2α concentrations relative to a control high–oleic acid meal: a randomized controlled trial¹²³⁴. *The American journal of clinical nutrition*. 2014;

30. Lopez-Miranda J, Williams C, Lairon D. Dietary, physiological, genetic and pathological influences on postprandial lipid metabolism. *Br J Nutr.* 2007;98:458–73.
31. Tiihonen K, Rautonen N, Alhoniemi E, Ahotupa M, Stowell J, Vasankari T. Postprandial triglyceride response in normolipidemic, hyperlipidemic and obese subjects - the influence of polydextrose, a non-digestible carbohydrate. *Nutrition Journal.* 2015;14:23.
32. Peddie MC, Rehrer NJ, Perry TL. Physical activity and postprandial lipidemia: Are energy expenditure and lipoprotein lipase activity the real modulators of the positive effect? *Progress in Lipid Research.* 2012;51:11–22.
33. Londos C, Cooper DM, Schlegel W, Rodbell M. Adenosine analogs inhibit adipocyte adenylate cyclase by a GTP-dependent process: basis for actions of adenosine and methylxanthines on cyclic AMP production and lipolysis. *Proceedings of the National Academy of Sciences.* 1978;75:5362–6.
34. Børglum JD, Vassaux G, Richelsen B, Gaillard D, Darimont C, Ailhaud G, Nègrel R. Changes in adenosine A1- and A2-receptor expression during adipose cell differentiation. *Mol Cell Endocrinol.* 1996 Mar 1;117(1):17-25.
35. DG Peers, JI Davies. Significance of the caffeine-like effect of various purines, pyrimidines and derivatives on adipose-tissue phosphodiesterase. *Biochemical Journal.* 1971;
36. Goldstein ER, Ziegenfuss T, Kalman D, Kreider R, Campbell B, Wilborn C, et al. International society of sports nutrition position stand: caffeine and performance. *Journal of the International Society of Sports Nutrition.* 2010;7:5.
37. Harpaz E, Tamir S, Weinstein A, Weinstein Y. The effect of caffeine on energy balance. *Journal of Basic and Clinical Physiology and Pharmacology.* 2017;28:1–10.
38. Arciero PJ, Gardner AW, Calles-Escandon J, Benowitz NL, Poehlman ET. Effects of caffeine ingestion on NE kinetics, fat oxidation, and energy expenditure in younger and older men. *American Journal of Physiology-Endocrinology and Metabolism.* 1995;268:E1192–8.
39. Westerterp-Plantenga MS, Lejeune MP, Kovacs EM. Body Weight Loss and Weight Maintenance in Relation to Habitual Caffeine Intake and Green Tea Supplementation. *Obesity Research.* 2005;13:1195–204.
40. Benowitz NL, Jacob P, Mayan H, Denaro C. Sympathomimetic effects of paraxanthine and caffeine in humans. *Clin Pharmacol Ther.* 1995;58:684–91.
41. Hwang JT, Park IJ, Shin JI, Lee YK, Lee SK, Baik HW, et al. Genistein, EGCG, and capsaicin inhibit adipocyte differentiation process via activating AMP-activated protein kinase. *Biochemical and Biophysical Research Communications.* 2005;338:694–9.
42. Mori M, Aizawa T, Tokoro M, Miki T, Yamori Y. Soy isoflavone tablets reduce osteoporosis risk factors and obesity in middle-aged Japanese women. *Clin Exp Pharmacol Physiol.* 2004;31 Suppl 2:S39-41.
43. Cha YS, Yang JA, Back HI, Kim SR, Kim MG, Jung SJ, et al. Visceral fat and body weight are reduced in overweight adults by the supplementation of *Doenjang*, a fermented soybean paste. *Nutr Res Pract.* 2012;6:520.

44. Cha YS, Kim SR, Yang JA, Back HI, Kim MG, Jung SJ, et al. Kochujang, fermented soybean-based red pepper paste, decreases visceral fat and improves blood lipid profiles in overweight adults. *Nutr Metab (Lond)*. 2013;10:24.
45. Harmon AW, Harp JB. Differential effects of flavonoids on 3T3-L1 adipogenesis and lipolysis. *American Journal of Physiology-Cell Physiology*. 2001;280:C807–13.

Tables

Table 1. Characteristics of the study group: anthropometric-training soccer players (n = 23).

Variable		± SD	Me	(Q1; Q3)
Age [years]	23.1	2.1	-	-
Body weight [kg]	74.1	8.5	-	-
Body height [cm]	179.6	8.5	179	174; 186
The level of adipose tissue [%]	-	-	12.1	11; 13.1
Muscle tissue content [%]	-	-	48.99	48.4; 49.6
Water content in the body [%]	-	-	64.23	63.5; 65.03

Table 2. Comparison of genistein effectiveness versus placebo

Variable	Genistein [median (Q1; Q3)]	Placebo [median (Q1; Q3)]	p
Rate of increase of triglycerides after cream consumption [(mg / dl) / min]	0.61 (0.31; 1.79)	1.28 (0.71; 1.86)	0.662
The rate of growth of triglycerides after ingestion of the chemical compound [(mg / dl) / min]	2.77 (1.40; 4.16)	2.21 (0.55; 3.08)	0.282
Difference in triglyceride concentration after consumption of the compound (MAX-BEG) [%]	29 (7; 60)	9 (6; 20)	0.228
Difference in triglyceride concentration after consumption of cream (MAX-BEG) [%]	8 (0; 19)	4 (0; 29)	0.852
Difference in glucose concentration after consumption of the compound (MAX-BEG) [%]	2 (0; 4)	6 (5; 9)	0.043
Difference in glucose concentration after consumption of cream (MAX-BEG) [%]	5 (3; 9)	6 (3; 13)	0.573

Mann-Whitney U test, **p <0.050**

Table 3. Comparison of the effectiveness of caffeine versus placebo

Variable	Caffeine [median (Q1; Q3]	Placebo [median (Q1; Q3]	p
Rate of increase in triglycerides after cream consumption [(mg / dl) / min]	5.12 (1.12; 8.74)	1, 28 (0.71; 1.86)	0.113
The rate of growth of triglycerides after ingestion of the chemical compound [(mg / dl) / min]	2.28 (0.43; 4.37)	2.21 (0.55; 3.08)	0.775
Difference in triglyceride concentrations after consuming the compound (MAX-BEG) [%]	36 (15; 94)	9 (6; 2)	0.049
Difference in triglyceride concentrations after consuming cream (MAX-EST) [%]	37 (18; 39)	4 (0; 29)	0.113
Difference in glucose concentration after consumption of the compound (MAX-EST) [%]	2 (0; 7)	6 (5; 9)	0.327
Difference in glucose concentration after consumption of cream (MAX-EST) [%]	8 (6; 9)	6 (3; 13)	0.688

Mann-Whitney U test, **p <0.050**

Table 4. Comparison of the effectiveness increase concentration by caffeine compared to placebo

Increase in triglyceride concentration after consumption of the compound over time [%]	Caffeine [median (Q1; Q3]	Placebo [median (Q1; Q3]	p
15min	5 (-11; 8)	4 (0; 16)	0.864
30 min	28 (8; 46)	2 (-9; 10)	0.018
45 min	15 (0; 31)	-7 (-26; 19)	0.224
60 min	7 (6; 12)	-13 (-23; 3)	0.036
75 min	7 (-0.09; 26)	-6 (-18; 17)	0.388
90 min	7 (-9; 26)	-9 (-19; 7)	0.145
105 min	-2 (-15; 10)	-3 (-20; 14)	0.776

Mann-Whitney U test, **p <0.050**

Table 5. Comparison of the effectiveness increase concentration triglyceride by genistein compared to placebo

Increase in triglyceride concentration after consumption of the compound over time [%]	Genistein [median (Q1; Q3)]	Placebo [median (Q1; Q3)]	p
15min	16 (4; 33)	4 (0; 16)	0.228
30min	23 (4; 52)	2 (-9; 10)	0.142
45min	2 (-2; 38)	-7 (-26; 19)	0.282
60 min	4 (-2; 29)	-13 (-23; 3)	0.029
75 min	6 (-1; 24)	-6 (-18; 17)	0.282
90 min	6 (-1; 24)	-9 (-19; 7)	0.081
105 min	4 (-3; 24)	-3 (-20; 14)	0.282

Mann-Whitney U test, **p <0.050**

Figures

Figure 1

Scheme of research.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Diagram1.jpg](#)
- [Diagram2.jpg](#)
- [Additionaltables.docx](#)