

The Effect of Caffeine Supplementation on Blood Lactate and Glucose after 800 and 1500 meter Run

ASGHAR KESHAVARZ SIAHPOOSH and AMIN NESAEI

Graduated from Islamic Azad University of Qazvin, Iran.
*Corresponding author E-mail: a.keshavarz1313@yahoo.com

<http://dx.doi.org/10.13005/bpj/914>

(Received: February 11, 2016; accepted: April 08, 2016)

ABSTRACT

The purpose of the present research is to study the effect of caffeine consumption with different doses on blood lactate and glucose levels after middle-distance running (1500 and 800-meter run). The participants of the present research include 15 middle-distance runners of Tehran province, Iran, who had participated in the national tournaments. The height, weight, and body fat of the subjects were measured on the first day of the research. In the first stage, subjects received placebos at three intervals (30 minutes before, 5 minutes before, and immediately after the 1500 and 800-meter run). In the second and third stages (with a 5-day interval between each two stages), all the subjects received caffeine gum with two different doses (180 and 300 mg) at three intervals (30 minutes before, 5 minutes before, and immediately after the 1500 and 800-meter run). Blood glucose and lactate of the subjects were measured in all three stages, 5 minutes before and immediately after performing the 1500 and 800-meter run. Gum and placebo were given to the participants in a double-blind fashion. Descriptive statistics, analysis of variance with repeated measures, and Tukey's post hoc test were used for data analysis ($p \leq 0.05$). The results of the present research indicated that none of the doses of caffeine had a significant effect on blood lactate and glucose levels both before and after the middle-distance exercise and during the 1500 and 800-meter run any judgment regarding consumption of caffeine by middle-distance runners, especially in the form of gums, requires further investigations.

Key words: Caffeine gum, Middle-distance running, Blood glucose, Blood lactate.

INTRODUCTION

Caffeine is an alkaloid compound which occurs naturally in the seeds, leaves, and fruits of many plants such as coffee beans, tea leaves, cocoa beans, and so on¹ and it also exists in a number of foods and drinks². Caffeine is readily distributed throughout the body after ingestion. The hydrophobic property of caffeine allows it to pass through all the biological membranes³. Human studies have shown that there are no physiological barriers hindering the passage of caffeine through tissues⁴⁻⁵. Understanding the ergogenic effects of caffeine requires an insight into the mechanisms of its function at the cellular level. Three major cellular

mechanisms have been proposed to explain the ergogenic potential of caffeine during exercises: (1) increased myofilament affinity for calcium and/or increased release of calcium from the sarcoplasmic reticulum in skeletal muscle, (2) cellular actions caused by accumulation of cyclic adenosine monophosphate (cAMP) in various tissues including skeletal muscle and adipocytes; and (c) cellular actions mediated by competitive inhibition of adenosine receptors in the central nervous system and somatic cells. Based on these three cellular mechanisms, caffeine has become prevalent among athletes as a nutritional supplement.

Studies carried on caffeine show that caffeine consumption stimulates the central nervous system and lead to the release of free fatty acids from adipose tissues. This is thought to increase the absorption of free fatty acids by the muscle and its oxidation in support of energy production; further, it is believed that muscle glycogen utilization is reduced through this mechanism. However, recent studies have cast a doubt on the validity of this hypothesis, especially in trained athletes⁶.

In most studies caffeine consumption led to increased concentration of free fatty acids in blood plasma, but this issue did not result in increased lipid oxidation or reduced glycogen utilization⁷. Despite these observations, caffeine did improve performance in most of these studies. Other observations support the theory that caffeine affects sport performance through mechanisms other than alterations in fat and carbohydrate metabolism and that caffeine can lead to better performance in events that last for 1 to 30 minutes. This duration is limited and cannot have a significant effect on the level of muscle glycogen. Thus, there must be other reasons for improved performance as a result of caffeine consumption⁸⁻¹⁰.

Caffeine has a stimulating effect on the central nervous system. Therefore caffeine may improve performance through influencing the processes that determine the stimulation of the neuromotor system. Research has shown that caffeine has positive effects on exercises such as 1500-meter swimming, one-hour time trial cycling, and on the capacity to perform a task during a two-hour pedaling test¹¹. Some studies carried out have focused on the effect of caffeine on brief performance with high intensity. The results of these studies are not as consistent as those studying endurance exercises^{9, 12, & 29}.

Although the results of some studies have not confirmed the effect of caffeine on sport performance, most studies have observed the ergogenic effect of caffeine in sub-maximal endurance exercises. That is because in endurance exercises, the intensity is kept constant while the duration increases. The effect of caffeine on muscle glycogen storage has been the focus of attention of

many studies. Caffeine, fructose, and glucose (individually or combined) have a considerable effect on muscle glycogen storage during cycling activity¹³. Similarly, caffeine and sucrose considerably stimulate endurance, but do not have a similar effect on performance. Previous studies, ingestion of caffeine at one hour before exercise decreases muscle glycogen utilization up to about 55% during the first 15 minutes of exercise. Therefore, glycogen stored in the next stages of exercise will become available and delays the onset of exhaustion. This is an important mechanism, for depletion of muscle glycogen can be responsible for much of the fatigue observed during endurance tests⁸. Therefore, increased utilization of intramuscular triglycerides or muscle fatty acids following caffeine consumption can inhibit decomposition of glycogen. However, increased concentration of plasma free fatty acids due to caffeine is not always accompanied by alterations in substrate utilization by the muscle. The concentration of free fatty acids in blood is a product of hepatic discharge and the amount utilized by other tissues that can widely change with a change in experimental conditions. Finally, in high altitudes, caffeine increases endurance through a mechanism other than mobilization of fatty acids, for it does not increase the concentration of plasma carnitine. The mechanism for this effect is yet unknown⁸.

It appears that during anaerobic exercises, caffeine increases the production of lactate by the muscle¹⁴⁻¹⁵. This phenomenon can reflect the increased production of muscle glycogenolysis by caffeine or increased release of lactate which does not necessarily reflect lactate production¹⁵. The focus of previous studies has been mainly on comparing different forms and doses of caffeine (capsule, oral solution, gum) to identify which of these forms is healthier, more secure, and more quickly absorbable. Noam and colleagues came to the conclusion that the rate of caffeine absorption was faster when consumed as chewing gums in comparison with a standard pill¹⁶. Moreover, research has shown that almost 85% of caffeine is released by 5 minutes of chewing gum¹⁶. This will lead to a faster onset of the stimulating effects of caffeine¹¹. Kamimori and colleagues studied the rate of absorption and bioavailability of caffeine in

chewing gums versus capsules¹¹. 84 male participants received placebo or 50, 100, and 200 mg of caffeine as chewing gum or capsule. The results showed that both gum and capsule formulations provide near comparable amounts of caffeine to the systemic circulation, but the rate of absorption was significantly faster from the gum formulation¹¹. Another research by Syed and colleagues studied caffeine gums in various doses. 48 participants consumed placebos or 50, 100, and 200 mg of caffeine chewing gum. The results showed that the maximum caffeine concentration was similar to the study that compared gum and capsule formulations. Moreover, the rate of absorption from the gum formulation was similar to the previous study signifying that the level and rate of absorption is constant when a certain dose is taken¹⁷.

Generally, a review of the results of research studies indicate that caffeine consumption in different doses and forms has different effects on the performance of athletes in different sports; yet the results of most of these studies suggest that caffeine consumption one hour before exercise increases the mobilization of free fatty acids and dependence on lipid catabolism; this will reduce muscle glycogen utilization which in turn delays

fatigue and has the greatest effect in prolonged endurance exercises and caffeine consumption in the form of chewing gums has the greatest effectiveness due to its fast rate of absorption. However, there is little research regarding the effect of caffeine in mid-endurance, sprint, and strength exercises and there has not yet been any definite conclusion regarding the positive effects of caffeine in these types of activity. Reviewing the literature, since caffeine is more quickly absorbed in the form of chewing gums and so far the effect of caffeine consumption on mid-endurance exercises has not been studied, carrying out a research in this regard seems necessary. Accordingly, the present research aimed to study the effect of caffeine gum consumption with different doses on blood glucose and lactate after a 1500 and 800-meter run as well as the time of this exercise in mid-endurance athletes.

Methodology

The present research is quasi-experimental in which 10 men participated with a mean and standard deviation of 24.8±1.6 years of age, 174.5±3.9 cm of height, 65.5±4.7 kg of weight, and 13±2.7% body fat. The subjects appeared at the place of testing and explanations were provided regarding the purpose of the research and test

Table 1: Mean and standard deviation of the level of blood lactate before and after performing the 1500-meter run and after consumption of different doses of caffeine and placebo and the results of the post hoc test

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Before 1500-meter run	3.3±1.60*	4.35±2.67*	4.45±2.19*
After 1500-meter run	15.93±2.29*	15.59±2.16*	12.32±2.26*

Notes: *significant difference before and after the 1500-meter run ($P \leq 0.05$)

Table 2: Mean and standard deviation of the level of blood lactate before and after performing the 800-meter run and after consumption of different doses of caffeine and placebo and the results of the post hoc test

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Before 800-meter run	3.5±1.46*	3.4±2.13*	3.76±2.34*
After 800-meter run	17.39±3.93	16.19±2.30	16.22±2.61

Notes: *significant difference before and after the 800-meter run ($P \leq 0.05$)

procedures and they filled out the consent forms. 15 subjects were selected from the middle-distance runners of Tehran province, Iran, who had participated in the national tournaments. The participants had no record of caffeine consumption. The height, weight, and body fat of subjects were measured in the beginning day of the research.

Seca height and weight measuring device was used for precise measurement of the height and weight of the subjects. Harpenden Skinfold Caliper was used in order to collect data related to body composition and body fat. Body fat was measured using Jackson-Pollock three-site Formula. The correlation of this method with underwater weighting is 0.9 for the seven-site formula and 0.89 for the 0.89 formula.

First, using Harpenden caliper the subcutaneous fat thickness of the chest, abdomen, and thigh of the subjects was measured and the sum of it was replaced in Jackson-Pollock formula (1978) in order to calculate the density; then, the body fat percentage was estimated by replacing the obtained density in Siri equation ($S = \text{chest} + \text{abdomen} + \text{Thigh}$)

$$\text{Body Fat Percentage} = \frac{4.95}{d} - 4.50 \times 100$$

A glucometer device (Bioneme GM300, made in Sweden with a precision of \pm mg/dl) was

used in the present research for measuring blood glucose. Further, Lactate Scout (made in Canada with a precision of \pm mm/l; SN: 1-800-462-2876) was used for measuring blood lactate and Robic Stopwatch with an error level of 0.06 seconds was used for recording the time of 1500-meter run.

In the first stage, all the subjects received placebos at three intervals (30 minutes before, 5 minutes before, and immediately after the 1500 and 800-meter run). In the second and third levels (with a 5-day interval between each two stages), all the subjects received caffeine gum with three different doses (180 and 300 mg) at three intervals (30 minutes before, 5 minutes before, and immediately after the 1500 and 800-meter run). The participants were asked to chew the gum for 5 minutes. Gum and placebo were given to the participants in a double-blind fashion. Blood glucose and lactate of the subjects were measured in all four stages, 5 minutes before and immediately after performing the 1500 and 800-meter run. Descriptive statistics (for calculating mean and standard deviation and drawing diagrams and tables), analysis of variance with repeated measures, and Tukey's post hoc test (for determining the effect of different doses of caffeine on blood glucose and lactate as well as the time of the 1500 and 800-meter run) were used for data analysis. All the data analyses were done using SPSS 16 and EXCEL.

Table 3: Mean and standard deviation of the level of blood glucose before and after performing the 1500-meter run and after consumption of different doses of caffeine and placebo and the results of the post hoc test

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Before 1500-meter run	89.3 \pm 13.19	118.3 \pm 12.34	117 \pm 9.46
After 1500-meter run	96.8 \pm 9.34	112.5 \pm 13.36	119.22 \pm 14.46

Table 4: Mean and standard deviation of the level of blood glucose before and after performing the 800-meter run and after consumption of different doses of caffeine and placebo and the results of the post hoc test

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Before 800-meter run	114.3 \pm 21.09	115.3 \pm 16.39	117.36 \pm 9.46
After 800-meter run	121.8 \pm 14.64	123.5 \pm 11.06	121.20 \pm 12.06

Findings

The results from analysis of variance with repeated measures (table 1) indicate the significant effect of different measurement stages on blood lactate. Moreover, Tukey's post hoc test indicated a significant difference between the levels of blood lactate before and after performing the 1500-meter run, after taking placebos, and after taking 180 mg and 300 mg of caffeine, while no significant difference was observed between the levels of blood lactate between consumption of different doses of caffeine and placebo before and after performing the 1500-meter run ($F=86.35$, $p \leq 0.05$).

The results from analysis of variance with repeated measures (table 2) indicate the significant effect of different measurement stages on blood lactate. Moreover, Tukey's post hoc test indicated a significant difference between the levels of blood lactate before and after performing the 800-meter run, after taking placebos, and after taking 180 mg and 300 mg of caffeine, while no significant difference was observed between the levels of blood lactate between consumption of different

doses of caffeine and placebo before and after performing the 1500-meter run ($F=124.53$, $P \leq 0.05$).

Moreover, the results of analysis of variance with repeated measures suggested the lack of a significant effect of different measurement stages on blood glucose ($F=14.342$, $P \leq 0.248$; table 3).

Moreover, the results of analysis of variance with repeated measures suggested the lack of a significant effect of different measurement stages on blood glucose ($F=6.214$, $P \leq 0.089$; table 4).

The results of analysis of variance with repeated measures suggested the lack of a significant effect of different doses of caffeine and placebo on the time of 1500-meter run ($F=6.098$, $P \leq 0.108$; table 5).

The results of analysis of variance with repeated measures suggested the lack of a significant effect of different doses of caffeine and placebo on the time of 800-meter run ($F=11.32$, $P \leq 0.095$; table 6).

Table 5: Mean and standard deviation of the time of 1500-meter run after consumption of different doses of caffeine and placebo

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Time (min)	4.22±0.23	4.17±0.18	4.18±0.36

Table 6: Mean and standard deviation of the time of 800-meter run after consumption of different doses of caffeine and placebo

	Placebo	180 mg of Caffeine	300 mg of Caffeine
Time (min)	2.04±0.43	2.03±0.41	2.02±0.29

DISCUSSION

The purpose of the present research was to study the effect of caffeine consumption with different doses on the levels of blood glucose and lactate after a middle-distance exercise (1500 and 800-meter run). The results showed that the level of blood glucose does not change with consumption of different doses of caffeine chewing gum. This

finding is consistent with the results of Van Soeren and Graham¹⁸, Bertram and colleagues¹⁹, and Bangsbo and colleagues²⁰. Van Soeren and Graham studied six recreational athletes and came to the conclusion that caffeine has no effect on the levels of blood glucose during an exhausting exercise. In another study carried out by Bertram and colleagues, they examined the effect of caffeine on glucose kinetics in 12 active men and

found that caffeine has no effect on endogenous glucose production. Bangsbo and colleagues studied the acute and chronic responses to caffeine and exercise in healthy adults. They found that the levels of blood glucose do not change with caffeine consumption.

However, there are studies that have found caffeine to cause a rise in blood glucose²¹⁻²³. Graham and Spriet²³ examined exercise responses to different doses of caffeine in endurance athletes and came to the conclusion that blood glucose concentration increases during exercise. The reason why blood glucose did not increase with caffeine gums in the present research could be due to the nature of the sport. While Graham and Spriet observed a rise in the level of blood glucose in endurance exercises, the present research studied mid-endurance exercises (1500 and 800-meter run). Another reason could be the low-dose caffeine ingestion by the participants of the present research. The doses used in the present research may have been insufficient for creating a significant change in the level of blood glucose during running, although the rate of absorption of caffeine in gum formulation is higher.

The results of the present research also showed a significant difference between the levels of blood lactate before and after performing the 1500 and 800-meter run, after consumption of placebo and 180, 240, and 300 mg of caffeine, while no significant difference was observed between the levels of blood lactate between consumption of different doses of caffeine and placebo before and after performing the 1500 and 800-meter run. The reason for the significant difference in the level of blood lactate before and after performing the 1500 and 800-meter run and after taking placebo and three doses of caffeine is due to the mid-endurance nature of the exercise. However, the results show that blood lactate does not change by taking different doses of caffeine. Research regarding the effect of caffeine on blood lactate during exercise has not been discussed much and in those few cases that do, the findings regarding the glycogen sparing property of caffeine are contradictory. Most researchers have shown that the concentration of blood lactate increases

after caffeine consumption²³⁻²⁶. This increase in blood lactate can indicate lactate production by active muscles or decreased blood clearance²³. Our findings are contrary to those of Van Soeren and Graham, Jackman and colleagues, Graham and Spriet, and Sukman^{23, 25, 26, & 30}. Van Soeren observed the metabolic effects of caffeine after withdrawal and reported that blood lactate increases in response to physical activity and caffeine consumption²⁸. Jackman and colleagues also studied the effect of caffeine during short intense exercise and found that the concentration of blood lactate increases during the exercise and after caffeine consumption²⁵. Moreover, Graham and Spriet found that blood lactate increase during exercise with caffeine ingestion²³. Studies that have reported an increase in blood lactate after caffeine consumption are inconsistent with the theory that caffeine is glycogen sparing. If glycogen sparing occurs, lactate concentration should not increase, since free fatty acids are the main source of fuel during exercise. Many factors can be the reasons behind the inconsistency of the results of the present research with those of other studies. The first reason is the low-dose caffeine consumption by the participants of the present research. The second is the fitness of the participants and the third is the type of exercise used in the research.

Considering the fact that this study is among the first to examine the effect of caffeine chewing gums on mid-endurance exercises, more investigations are required to identify the precise mechanisms for the ergogenic effect of caffeine in these exercises.

CONCLUSION

The results of the present research showed that consumption of 180 and 300 mg of caffeine does not affect performance and the levels of blood glucose and lactate after a 1500 and 800-meter run. Considering these results, any judgment regarding consumption of caffeine by mid-endurance athletes, especially in the form of gums, requires further investigations. This research is among the first studies on the use of caffeine gum and can thus be used as a stepping stone for future research.

REFERENCES

1. Cox G, Desbrow B, Burke L. *Journal of Applied Physiology*, **93**: 990-999 (2002).
2. Arnaud MJ. *Progress in Drug Research*, **31**: 273-313 (1987).
3. Fredholm BB, Battig K, Holmen J, Nehlig A, Zvartau EE. *Pharmacological Reviews*, **51**: 81-125 (1999).
4. Bonati M, Garattini S. Berlin: Springer-Verlag, (1984).
5. Yesair DW, Branfman AR, Callahan MM. Human disposition and some biochemical aspects of methylxanthines. In G. A. Spiller (Ed.), New York: Alan R. Liss. 215-234 (1984).
6. Melinda M, Nanna LM, Janice T. Human Kinetics. 2nd Edition (2009).
7. Graham TE, Spriet LL. *Journal of applied physiology* (Balitimore, Md.), **71**(6):2292-2298 (1991).
8. Essig D., Costill, and P.J. Vanhandel. *Int. J. Sports Med.* **1**: 86-90 (1980).
9. Rall, T. W. New York: Macmillan. 589-603 (1985).
10. Doherty, M., Smith, P., Hughes, M., Davison, R. *J. Sports Sci.* **22**(7): 637-43 (2004).
11. Kamimori GH, Karyekar CS, Otterstetter R, Cox DS, Balkin TJ, Belenky GL, Eddington ND. *International Journal of Pharmaceutics*, **234**: 159-167 (2002).
12. Costill DL, Dalasky GP, Fink WJ. *Medicine and Science in Sports and Exercise*, **10**: 155-158 (1978).
13. Erickson MA, Schwarzkopf RJ, Mckenzie RD. *Medicine and science in sports and exercise*. **19**(6):579-582 (1987).
14. Anselme, F, Collomp, K., Mercier, B., ahmaidi, S., Prefaut, C. *Eur. J. Appl. Physiol. Occup. Physiol.* **65**: 188-191 (1992).
15. Collomp, K., S. Ahmaidi, M. Audran, J.-L. Chanal, C. Prefaut. *Int. J. Sports Med*, **12**: 439-443 (1991).
16. Novum. Amurof Confections Company: USA, (1998).
17. Syed SA, Kamimori GH, Kelly W, Eddington ND. *Biopharmaceutics and Drug Disposition*, **26**: 403-409 (2005).
18. VanSoeren MH, Graham TE. *Journal of Applied Physiology*, **85**(4): 1493-1501 (1998).
19. Battram, D. S., Graham, T. E., Richter, E. A., & Dela, F. *Journal of Physiology*, **569**.1, 347-355 (2005).
20. Bangsbo J, Jacobsen, K, Nordberg N, Christensen NJ, Graham TE. *Journal of Applied Physiology*, **72**(4): 1297-1303 (1992).
21. Raguso, C. A, Coggan, A. R., Sidossis, L. S, et al. *Metabolism*, **45**(9): 1153-1160 (1996).
22. Trice I, Haymes EM. *International journal of sport nutrition* (Champaign, Ill.), **5**(1):37-44 (1995).
23. Graham TE, Spriet LL. *Journal of Applied Physiology*, **78** (3): 8 (1995).
24. Bell, D. & McMellan, T. *Journal of Applied Physiology*, **93**: 1127-1234 (2002).
25. Jackman M, Wendling P, Friars D, Graham TW. *Journal of Applied Physiology*, **81**(4): 1658-1663 (1996).
26. Spriet, L. L. *International Journal of Sport Nutrition*, **5**: 84-99 (1995).
27. Warren GL, Park ND, Maresca RD, McKibans KI, Millard-Stafford ML. *Med Sci Sports Exerc.* **42**:1375-1387 (2010).
28. Van Soeren MH, Mohr T, Kjaer M, Graham TE. *J Appl Physion*, **80**:999-1005 (1996).
29. Davis JK, Green JM. *Sports Med.* **39**:813-832 (2009).
30. Sokmen B, Armstrong LE, Kraemer WJ, Casa DJ, Dias JC, Judelson DA, Maresh CM. *J Strength Cond Res.* **22**:978-986 (2008).