

Analysis of the Impact of Normal Doses of Caffeine on Students' Reaction Time

Lotfali Massoumi-Moghadam¹, Masoud Ganji², Yoosef Abdollahi³

¹Department of Biology, Mohaghegh Ardabili University, Ardabil, Iran

²Department of Statistics, Mohaghegh Ardabili University, Ardabil, Iran

³Department of Animal Science, Mohaghegh Ardabili University, Ardabil, Iran

ABSTRACT

Caffeine is a methylxanthine that exists in food ingredients such as tea, coffee, cola, cocoa, etc. It is widely used in many materials for pharmaceutical applications. In this respect, it is probably considered the most widely used material with diverse effects compared to other consumable materials. For this study, a computer application was designed that allowed the measurement of the reaction time of 75 test subject (in milliseconds) and then, the effects of various doses of oral caffeine on their reaction times were determined. All the volunteers - who were students from universities in Ardabil city - were tested in five groups. Each group consisted of fifteen normal and non-smoker individuals with mean age of 25.2 ± 0.6 and mean weight of 65.62 ± 1.8 . The tests were conducted in two stages with Double Anonymous method; first, before consumption of caffeine (as control) and then after caffeine consumption (with an interval of 40 ± 5 minutes) with one of 5mg/kg doses. Reaction times were recorded in milliseconds. Statistical analysis using paired t method showed that among the doses used, amount of 1 and 2 mg/kg doses no significant effects ($P \leq 0.05$) on reaction time while it declined significantly by the doses of 3, 4 and 5 mg/kg ($P \leq 0.01$, $P \leq 0.0001$, and $P \leq 0.03$, respectively). The maximum reduction of reaction time and the best impact was associated with dose of 4 mg/kg ($P \leq 0.001$). From result of the current study, it can be concluded that low doses of caffeine have little impact on reaction time so that the minimum required dose of caffeine for reducing reaction time in young people is "3 mg/kg".

Keywords: Oral Caffeine, Methylxanthine, Reaction Time.

1. INTRODUCTION

Caffeine is a white crystalline xanthine alkaloid that acts as a stimulant drug and an acetylcholinesterase inhibitor [1]. Caffeine is found in varying quantities in the seeds, leaves, and fruit of some plants, where it acts as a natural pesticide that paralyzes and kills certain insects feeding on the plants. It is most commonly consumed by humans in infusions extracted from the seed of the coffee plant and the leaves of the tea bush, as well as from various foods and drinks containing products derived from the kola nut. Other sources include yerba maté, guarana berries, guayusa, and the yaupon holly.

In humans, caffeine acts as a central nervous system stimulant, temporarily warding off drowsiness and restoring alertness. It is the world's most widely consumed psychoactive drug, but, unlike many other psychoactive substances, it is both legal and unregulated in nearly all parts of the world. Beverages containing caffeine, such as coffee, tea, soft drinks, and energy drinks, enjoy great popularity; in North America, 90% of adults consume caffeine daily [2].

Caffeine is toxic at sufficiently high doses. Ordinary consumption can have low health risks, even when carried on for years – there may be a modest protective effect against some diseases, including certain types of cancer. Caffeine can have both positive and negative effects on anxiety disorders. Some people experience sleep disruption if they consume caffeine, especially during the evening hours, but others show little disturbance and the effect of caffeine on sleep is highly variable.

Evidence of a risk to pregnancy is equivocal, but some authorities have concluded that prudent advice is for pregnant women to limit consumption to the equivalent of two cups of coffee per day or less [3] Caffeine has diuretic properties when administered to people who are not used to it, but regular users develop a tolerance to this effect, and studies have generally failed to support the common notion that ordinary consumption contributes significantly to dehydration [4]. With heavy use, strong tolerance develops rapidly and caffeine can produce clinically significant physical and mental dependence.

Caffeine is a trimethylxanthine which exists in many foods such as tea, coffee, cola and some medications. Caffeine is absorbed through the digestive tube after about 40 ± 5 minutes and reaches its highest level in plasma. Various mechanisms have been proposed for the way caffeine acts (operates) such as phosphodiesterase inhibitors and an increase of intracellular calcium [5]. Phosphodiesterase inhibitor is a drug

*Corresponding Author: Lotfali Massoumi Moghadam (PhD), Department of Biology, Mohaghegh Ardabili University, Ardabil, Iran. Email: lotfi.massoumi@gmail.com

that blocks one or more of the five subtypes of the enzyme phosphodiesterase (PDE), therefore preventing the inactivation of the intracellular second messenger's cyclic adenosine monophosphate (cAMP) and cyclic guanosine monophosphate (cGMP) by the respective PDE subtype(s) [6]. Also, it is believed that the main mechanism of caffeine is blocking adenosine receptors [7]. Different researchers have conducted many studies to assess the effects of caffeine on the nervous system using electroencephalography, psychological tests, reaction time, and etc. [9]. By studying the effect of caffeine before consumption and after consumption for about thirty minutes, Kawamura *et al.* [10] did not observe significant changes in reaction time. Hogervorst *et al.* [11]-realized that 250 mg of caffeine reduces the speed of short-term memory, while it improves short-term memory in adults.

Most studies conducted on the effects of caffeine on the central nervous system have been performed using pure caffeine which is not the normal form of caffeine consumption. In addition, most tests have been qualitative. In the present study, oral caffeine was used. Also, a computer application was specifically designed to record reaction time accurately which provides the possibility to record reaction time with millisecond accuracy.

2. MATERIALS AND METHODS

This study was conducted on students of Science Faculty of Mohaghegh Ardabili University. The study group was composed of 75 young, healthy and non-smoker people with mean age of 25.2 ± 0.6 and mean weight of 65.62 ± 1.8 which were classified into five groups according to caffeine doses. Each group consisted of fifteen people who completed consent forms before the test. In the forms, some recommendations were also mentioned on the necessary preparations before the test (including not to use caffeine compounds up to twelve hours before the test and to eat a simple breakfast). The test was conducted after a uniform breakfast in a quiet room. One day before the test, the volunteers practiced with the relevant software to be familiar with its procedure and also to gain the skills necessary to perform the test. The control test was conducted before oral caffeine consumption based on the protocol.

The second part of the test was conducted using anonymous method 40 ± 5 minutes after oral caffeine consumption with one of doses. The procedure of the software is fully described in the protocol. At the central part of the monitor, three two-digit numbers would randomly appear with an interval of 200 milliseconds, each one with duration of 100 milliseconds. Then, the fourth double-digit number would also randomly appear with duration of 100 milliseconds in one of the places of the previous three numbers. This number (the fourth number) may be one of the numbers previously appeared, in which case the test subject would press a key and the application would consider it the correct answer and would write down the reaction time. The maximum response time was considered 750 milliseconds. The response times greater than 750 milliseconds and less than 300 milliseconds were considered incorrect. The fourth number appeared may not be among the two-digit numbers, in which case pressing the corresponding key by volunteers would be considered an incorrect answer. In each of the shifts before or after oral caffeine consumption, sixty-series would be displayed and in each series, four double-digit numbers would be displayed. Thus, in total, each volunteer randomly faced with 120 double-digit numbers and 120 double-digit numbers as the fourth number.

Reaction time in each test was recorded (in milliseconds) by the computer software and the data obtained were analyzed by SPSS software and paired t method.

3. RESULTS

In this research, crud data obtained from 75 persons among subjects were analyzed; the subjects were healthy without any acute or chronic diseases. The results from five groups were statically evaluated in two parts. The first part is associated with reaction times of test subjects before oral caffeine consumption in which the required data was collected for the 9 states of fast binocular vision based on the principles of babsky at one meter distance from the monitor and the angle of four degrees. Then, the total average reaction time of volunteers was calculated and also, precious statistical calculations were done for required cases. The second part is associated with reaction times of test subjects after oral caffeine consumption in which the same required data was collected for the 9 states of fast binocular vision and precious statistical calculations were done for required cases. The overall results of the tests for five groups were obtained by computer analysis, and with the help of statistical calculations which will be presented as the conclusion.

Table 1 shows the effects of caffeine on reaction time, with significant levels for their changes before and after caffeine consumption related to each of the five groups of volunteers. Also, the following figure shows the implications of this data as a graph.

Table 1. The effects of caffeine on reaction time

Test Groups	Consumption Dose of Oral Caffeine	Average Reaction Time		Significance
		Before Consumption	After Consumption	
First Group	1 mg/kg	522.45 ± 7.4	514.52 ± 8.80	No significant
Second Group	2 mg/kg	474.57 ± 14	469.77 ± 12.60	No significant
Third Group	3 mg/kg	528.05 ± 10.80	510.74 ± 13.80	Significant
Fourth Group	4 mg/kg	497.22 ± 14.30	441.52 ± 14.83	Significant
Fifth Group	5 mg/kg	492.12 ± 7.40	473.80 ± 9.84	Significant

Based on the table's data, reaction time after caffeine consumption was decreased in all groups but analysis of this data using paired t method showed that this time reduction is not significant for doses of 1 and 2mg in the first and the second group ($P > 0.05$) but in the third group ($P \leq 0.01$), the fourth group ($P \leq 0.0001$) and the fifth group ($P \leq 0.03$), reaction time is significantly decreased.

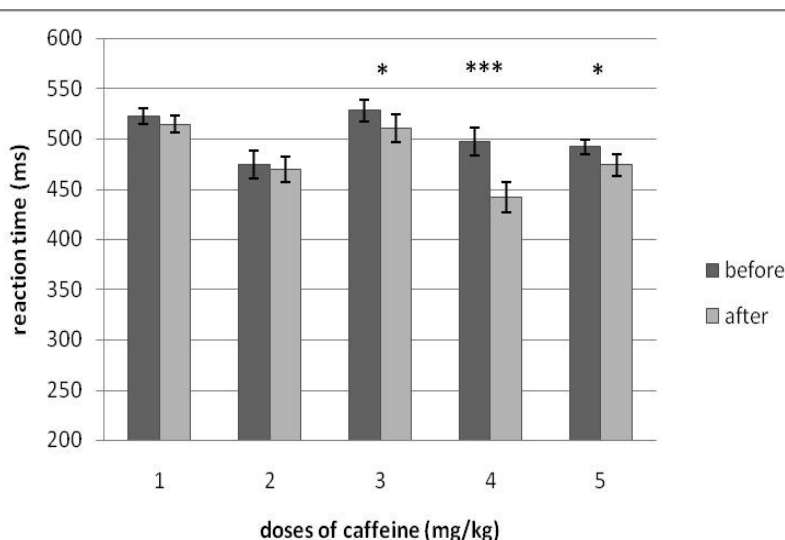


Figure 1. Average reaction times before and after caffeine consumption; *= $P \leq .05$, ***= $P \leq .000$.

4. DISCUSSION AND CONCLUSION

The findings show that significant reductions are seen in reaction times for test subjects after consumption of 3 mg/kg, 4 mg/kg and 5 mg/kg of caffeine. While in other doses used (1 mg/kg and 2 mg/kg), no significant changes were observed. These results are consistent with the results of the study by Seidl et al. [12]. Kawamura et al. [10] examined a dose of 500 mg caffeine on 10 people and observed that no significant changes were observed 30 minutes after caffeine consumption. Ruitjer et al. [13] also concluded that reaction time after caffeine consumption is reduced further in comparison to its reduction by placebo. They argued that caffeine expedites information processing [12, 14, 15]. Such a reduction in reaction time caused by caffeine has also been reported by Azcona et al. [7], Clublely et al. [8].

Caffeine acts by blocking adenosine receptors and thus changes the level of neurotransmitters such as dopamine, adrenaline and glutamate. A1 Adenosine receptors are associated with D1 dopamine receptors and A2a adenosine receptors are associated with D2 dopamine receptor [16]. Of dopamine receptors D1, D2, D4 and D5 that exist in hippocampus, stimulation of D1 and D2 hippocampus receptors in hippocampus improve retention of working memory and thus facilitates the motor activities of nigrostriatal dopaminergic system which are highly sensitive to caffeine compared with other parts of the brain [17, 18]. Low doses of caffeine (1 mg/kg) also increase the functional activity of striatum in rats [19]. Reduced motor activity is also considered one of the effects of caffeine on A2a agonist receptors [20].

In this study, some changes were made in cases of different consumption doses of caffeine because of dopamine release following inhibition of adenosine receptors by caffeine. These findings totally state that caffeine facilitates motor processes while it may have effects on cortical sections involved in functions such as alertness and motor processes. This material provides a review of evidence for improved motor activities which can be studied in the treatment of diseases associated with motor processes (such as Parkinson).

ACKNOWLEDGMENTS

The presented study was done in physiological research lab of Science Faculty with the financial support of Mohaghegh Ardabili University. I express my gratitude to my colleagues: Dr, Masood Ganji, and Mr. Abdollahi (MSc. Student). I also thank all the students who spent most of their time despite having little time and being busy with the studies in our physiological research lab.

REFERENCES

1. Lopes C, Monteiro E, Bentes de Paula DM, Erritto Barbo F, et al. 2009. Chemical composition, acetylcholinesterase inhibitory and antifungal activities of *Pera glabrata* (Schott) Baill. (Euphorbiaceae). *Revista Brasileira de Botânica* (São Paulo) 32 (4).
2. Lovett, R. 2005. Coffee: The demon drink? *New Scientist*, 2518.
3. Mayo Clinic staff. 2012. Pregnancy Nutrition: Foods to avoid during pregnancy. Mayo Clinic.
4. Nehlig A, Daval, JL, Debry G. 1992. Caffeine and the central nervous system: mechanisms of action, biochemical, metabolic and psychostimulant effects. *Brain Research Reviews* 17 (2): 139–70.
5. Daly JW, Padgett WL, Shamim MT, 1986. Analogues of caffeine and theophylline: effect of structural alterations on affinity at adenosine receptors. *Journal of Medicinal Chemistry* 29 (7): 1305–8.
6. Essayan DM. 2001. Cyclic nucleotide phosphodiesterases. *J Allergy Clin Immunol*. 108 (5): 671–80.
7. Azcona O, Barbanoj MJ, Torrent J, Jane F. 1995. Evaluation of the central effects of alcohol and caffeine interaction. *Br J Clin Pharmacol* 40: 393–400
8. Clubley M, Bye CE, Henson TA, Peck AW, Riddington CJ. 1979. Effects of caffeine and cyclizine alone and in combination on human performance, subjective effects and EEC activity. *Br J Clin Pharmacol*; 7: 157–163.
9. Fredholm BB. Astra Award Lecture. 1995. Adenosine, adenosine receptors and actions of caffeine. *Pharmacol Toxicol*, 76(2): 93–101.
10. Kawamura N, Maeda H, Nakamura J, Morita K, Nakazawa Y. 1996. Effects of caffeine on event related potentials: comparison of oddball with single tone paradigms. *Psychiatry Clin Neurosci*, 50(4): 217–221.
11. Hogervorst, E., Riedel, W.J., van Boxtel, M.P.J. and Jolles, J. 1995. Smoking and cognitive complaints. In *The Maastricht Aging Study*, Jolles, J., Ponds, R., van Boxtel, B.P.J. and Houx, P. (Eds), Neuropsych, Maastricht, pp. 105±111.
12. Seidl R, Peryl A, Nicham R, Mauser E. 2000. A taurine and caffeine containing drink stimulates cognitive performance and well being. *Amino Acids*; 19(3–4): 635–642.
13. Ruijter J, DéRuiter MB, Snel J. The effects of caffeine on visual selective attention to color: An ERP study. *Psychophysiology* 2000; 37: 427–439.
14. Loke WH, Meliska CJ. 1984. Effects of caffeine use and ingestion on a protracted visual vigilance task. *Psychopharmacology*; 84: 54–57.
15. Lorist MM, Tops M. 2003. Caffeine, fatigue and cognition. *Brain Cogn*; 53(1): 82–94.
16. Nikojevic O, Sarges R, Daly JW and Jacobson KA. 1991. Behavioral effects of A1- and A2-selective adenosine agonists and antagonists: evidence for synergism and antagonism. *Journal of Pharmacology and Experimental Therapeutics*, 259: 286–294.
17. Packard MG, White NM. 1991. Dissociation of hippocampus and caudate nucleus memory systems by post-training intracerebral injection of dopamine agonists. *Behav Neurosci*; 105: 295–306.
18. White NM, Packard MG, Seamans J. 1993. Memory enhancement by post-training peripheral administration of low doses of dopamine agonists: possible autoreceptor effect. *Behav Neural Biol*; 59: 230–241.
19. Nehlig A and Boyet S. 2000. Dose–response study of caffeine effects on cerebral functional activity with a specific focus on dependence. *Brain Research*, 858: 71–77.
20. Hollingsworth H. 1912. The influence of caffeine on mental and motor efficiency. *Arch Psych*; 3: 1–16.