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Article

Effects of caffeine on muscle signals measured with sEMG signals

Jisoo Park[1], Dennis Park[2]
[1] East Coweta High School, Sharpsburg, Georgia
[2] Sewon America, Inc., Lagrange, Georgia

SUMMARY

Caffeine is widely known for its ability to arouse the central nervous system. However, it is unclear whether caffeine can improve muscle performance. Previous studies have shown that caffeine has no significant effect on muscles during contraction. We conducted this experiment to determine if caffeine acts as a potential stimulant to the resting biceps, and unlike previous studies, ours was conducted on the resting human body after caffeine intake. The state of muscle was assessed with surface electromyography (sEMG), which non-invasively measures the muscle signals, or the level of electric activity of muscles. The sEMG results collected before caffeine intake and after 45 minutes of intake were compared to test the hypothesis that caffeine intake significantly changes the sEMG results. We also measured sEMG 10 hours after intake to investigate if there were any significant changes after caffeine is expected to be completely metabolized in the body and cleared from the bloodstream. We found that the average amplitudes were significantly increased in the sEMG 45 minutes after caffeine intake compared to before caffeine intake. Also, there were no significant changes in the sEMG 10 hours after caffeine intake, after most of the caffeine would have cleared from the bloodstream. We concluded that caffeine has significant potential to affect the resting biceps and suggest further research to study the contrasting outcomes from our experiments and previous research.

INTRODUCTION

The relationship between caffeine and neuromuscular activity has been widely known to have a positive correlation (1). Caffeine is popular for its ability to arouse the mind and elevate the mood as a stimulant to the central nervous system for a relatively low price. Aside from its effects on the mind, caffeine is also known to enhance voluntary activation which is the level of neural drive to the muscle during exercise (2). Studies suggest that caffeine exerts its effects on the central nervous system by blocking the adenosine receptors, especially A1 and A2 receptors, rather than directly stimulating the central nervous system (3). Adenosine receptors are known to inhibit dopamine release, and dopamine is known to have a positive relationship with locomotor and sensorimotor activities (4, 5). This phenomenon suggests that blocking adenosine receptors will disinhibit dopamine release and increase the central release and concentration of dopamine neurotransmitters.

Electromyography (EMG) systems measure the electrical response to the nerve stimulation in muscles by counting the number of muscle fibers activated. Therefore, EMG can be used to measure the level of activation in muscles (6). Research by Trevino et al. demonstrated that the intake of caffeine had no significant effect on the EMG of elbow flexors during peak torque in subjects who were active in recreation and had at least two years of extensive resistance training (7). Another study conducted by Williams et al. suggests that caffeine ingestion does not have a significant effect on EMG during the force production in actions requiring strength (8). These studies were restricted to testing the muscle signals during isometric contraction, which is contraction in the muscles without any visible movement (7, 8).

In a muscle, completely at rest, there would normally be almost no significant activities in the muscle signals (9). This is due to the lack of voluntary activities in the muscle fibers, leading to limited frequency in the signals; however, there are exceptions to this rule. In cases of the presence of abnormalities to the muscle that interferes with the motor neuron signals, such as nerve diseases like polymyositis, muscular dystrophy, myasthenia gravis, myotonic muscles, or with damage to the motor nerve, such as pinching nerves, there is higher frequency and amplitude to the muscle signals (6).

We conducted this research to test if caffeine significantly alters muscle signals during rest, unlike previous research that studied its effect on EMG signals during isometric contraction. We hypothesized that the “abnormalities” in the frequency of the motor neuron signals described neural activity by increasing neurotransmitter release (10). The control trials were conducted 1 hour before caffeine intake, as plasma caffeine concentration is at its peak 30–60 minutes post caffeine intake (11). Unlike the previous research, our research also measured surface EMG (sEMG) signals 10 hours after caffeine intake since the half-life of caffeine is around 5 hours, hypothesizing that there would be significant change in the muscle signals after an expected amount of 75% of caffeine has been metabolized due to possible muscle fatigue from aroused muscle activation at peak caffeine levels (12). Our results showed that there was a significant change in the peak amplitudes of the muscle signals 45 minutes

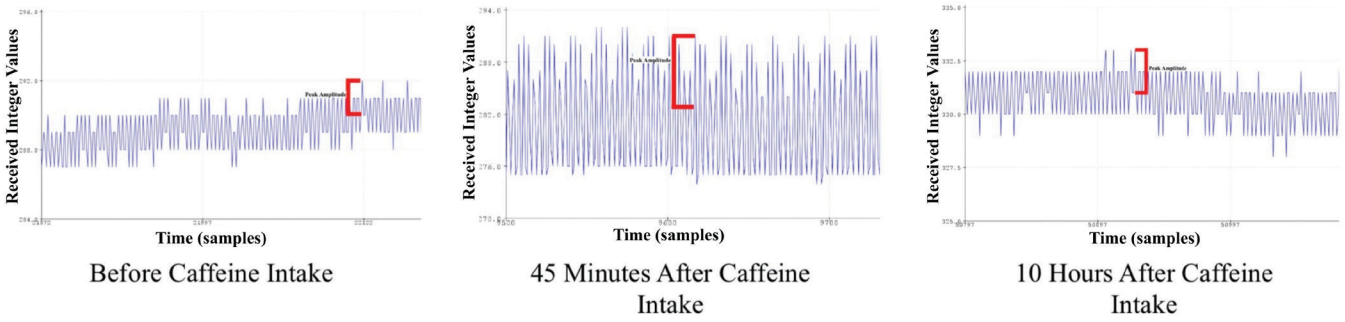

Figure 1: Representative sEMG signals graphed for trials before caffeine intake, 45 minutes after caffeine intake, and 10 hours after caffeine intake. The x-axis represents time in samples (five ms each) and the y-axis represents received voltage values. The peak amplitude is highlighted in each graph in red.

post caffeine intake and no significant change in the peak amplitudes of the muscle signals 10 hours post caffeine intake.

RESULTS

We measured sEMG signals prior to, 45 minutes after, and 10 hours after caffeine intake. This was repeated for four total trials. The amplitudes and the amplitude average of the signals were measured after conducting four trials each. In this case, the amplitude is considered the peak amplitude, or the signal values over the averaged median of the received voltage values in the graph. Peak amplitudes are highlighted **(Figure 1)**. Then, the average of each procedure was measured to perform a paired t-test to determine significance.

As expected, the sEMG value plot of the control trials conducted before caffeine intake had frequency values that were static and the amplitude values were relatively low, as compared to the values 45 minutes after caffeine intake, and the peak amplitude values were limited to an average of 9.185 mV (note: the range of mV output of the Arduino system can be different from that of EMG systems as EMG values range from 0.1–0.5 mV while Arduino units are 4.9 mV each, resulting in higher results in terms of mV). The sEMG value plot of the experimental trials 45 minutes after caffeine intake had distinct frequency values and the peak amplitude values had an average of 30.625 mV, showing signs of abnormality in activity. The graph of the experimental trials 10 hours after drinking coffee were similar in shape to that of the control trials and had peak amplitude values with an average of 7.35 mV. The sEMG data was greatest 45 minutes after caffeine intake **(Figure 1, Table 1)**.

The trials before intake and 45 minutes after intake had a significant difference (n = 4, p = 0.01425). This suggests that there may be a possibility that caffeine influences muscle activity, causing a phenomenon like the “abnormalities” that interferes with motor neuron signals. However, the trials before intake and 10 hours after intake did not have a significant difference (p = 0.2152, **Figure 2)**. This demonstrates that the muscle activation after the intake of caffeine, if any, did not cause any kind of muscle fatigue or significant difference after a majority of the caffeine had been metabolized.

Experiment/Trial	Trial 1	Trial 2	Trial 3	Trial 4	Average
Before intake	7.35 mV	14.7 mV	7.35 mV	7.35 mV	9.1875 mV
45 minutes	29.4 mV	39.2 mV	36.75 mV	17.15 mV	30.625 mV
10 hours	4.9 mV	9.8 mV	7.35 mV	7.35 mV	7.35 mV

Table 1. Peak ampllitudes of sEMG activity before and after caffeine intake.

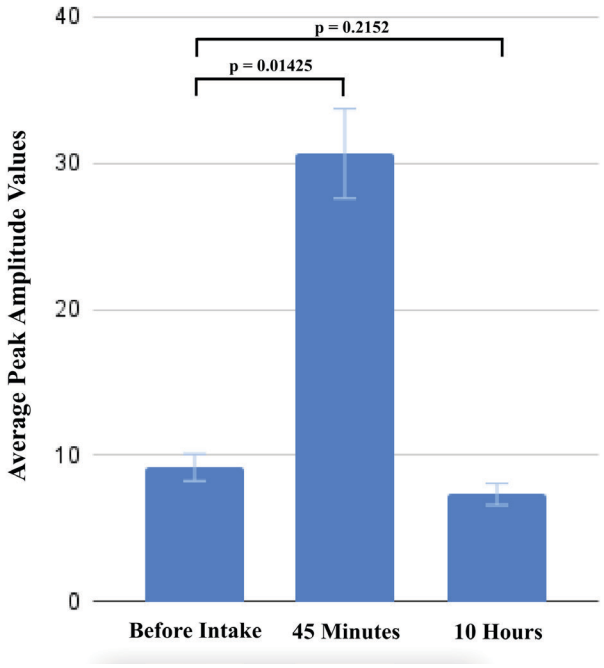

Figure 2: Average amplitudes and statistical analysis of each test. Data of each trial were compared with paired t-test and the significance level was 0.025. Data shown as mean ± standard error.

DISCUSSION

The significant increase in amplitude demonstrated an increase in the number of activated muscle fibers during rest, supporting our hypothesis that caffeine does have a potential on resting bicep muscle signals. This data shows that caffeine potentially affects voluntary muscle activation during rest. This could be beneficial because caffeine- induced activity in muscle during rest can burn fat. There was no significant change in amplitude of muscle signals for the trials conducted 10 hours after caffeine intake, indicating that the muscles return to normal after the caffeine clears out of the body. However, 10 hours after caffeine intake may have been enough time to recover from additional muscle stimulation. A measurement of amplitude of muscle signal five hours after caffeine intake, which is within the half-life of caffeine, might better measure the after-effects of muscle stimulation from caffeine.

There were several limitations in this study. First, the myoware muscle sensor used to measure the sEMG signals could be inadequate for thorough data collection needed for this research, as it is not a professional EMG device used for medical or research purposes. We tried to mitigate this weakness by conducting the trials in a silent environment and braiding the cords to minimize the noise it might pick up, since the signals are susceptible to noise. Second, since only one subject was involved in the study, the results of this research might not apply to those with other conditions. Factors such as age, gender, hormones, diet, smoking, drug use, and genetic background may affect the results of the study (13). Third, a limitation of the study is the absence of a fixed-dose procedure for the acute amount of caffeine and rather using coffee with an average 250 mg caffeine. The lack of control for the amount of caffeine might affect the results.

The results of this research are not in concordance with the previous research. Of course, previous research compared contraction signals of muscles and our research compared signals in muscles at rest. However, it is not clear why there was an abnormal activity in the muscle signals during rest, whereas the muscle signals during contraction were not significantly different. This difference in findings merits further research.

MATERIALS AND METHODS

The subject and the experimenter were the same person. The subject was a female, 18 years of age, and did not take any extensive resistance training three months prior to the experiment and during the experiment. The subject did not consume caffeine three months before the experiment to prevent any tolerance to caffeine.

The sEMG signals were collected with the myoware sensor provided by Adafruit with the Arduino Uno machine, which was connected to a laptop. The electrodes were connected to the biceps brachii portion of the subject. Three electrodes were connected for accurate measurement; the electrodes consisted of one middle electrode placed on the middle section of the bicep, one end electrode placed towards the distal end of the muscle, and one reference electrode placed on the tendon of triceps brachii area **(Figure 3)**.

The AnalogReadSignal function of the Arduino system and the graph on the Serial Plotter was used to measure the amplitude and frequency of the signal in each trial. The AnalogRead function inputs voltages into integer values which can be plotted into the y-axis of the Serial Plotter program as a graph. In the system, the unit for the x-axis was in arduino sampling rate, or samples, and one sample is five milliseconds. The subject's sEMG signals of the biceps were measured one hour before drinking coffee as negative control data. The subject's sEMG signals were measured 45 minutes after drinking coffee with caffeine content of 5 mg/kg of body mass as experimental data, or an average of 250 mg, using the same sensor. The subject's signals were then measured 10 hours after drinking coffee, using the same sensor. Each unit of inputted voltages is equal to 4.9 millivolts (mV) and all data was converted to mV when analyzing data.

Four trials were conducted for each procedure, and the data was statistically analyzed with paired *t*-test with GraphPad's *t* Test Calculator. The peak amplitudes of each trial were compared with a paired *t*-test with the significance level being 0.025.

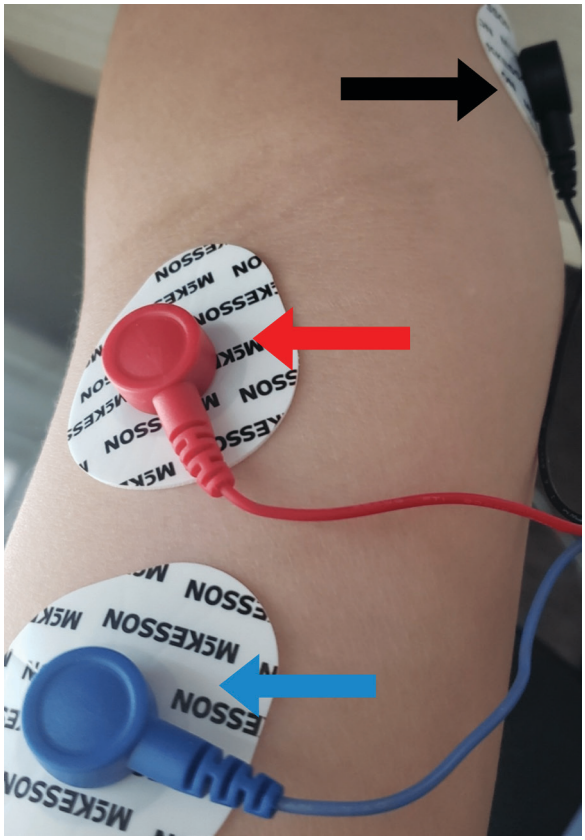

Figure 3: Setup of electrodes on the bicep. Electrode with red cable is the end electrode, electrode with blue cable is the middle electrode, and the electrode with the black cable is the reference electrode. Arrows indicate electrodes with corresponding colors.

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