ORIGINAL RESEARCH

Caffeine's acute impact on blood pressure, heart rate, stroke volume, and cardiac output before and after peak exercise

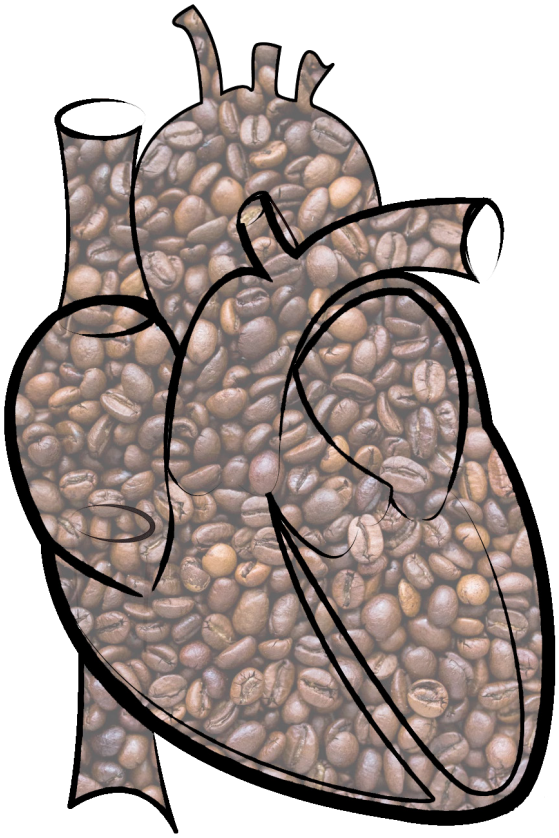
Joe Mehrabi
Sackler School of Medicine, Tel Aviv University, Tel Aviv

Abstract

Caffeine is a common substance in the sports energy drinks utilized by many athletes, permitting them to train more strenuously for longer periods of time. During exercise, several cardiovascular variables are amplified to accommodate the body's oxygen demand. This experiment aims to shed some light on caffeine's impact on blood pressure, heart rate, stroke volume, and cardiac output before and after exercise. Out of a pool of 93 students, 42 men and women (21 caffeinated; 21 non-caffeinated) were chosen at random and asked to pedal on a stationary bicycle until exhaustion. Their blood pressures and heart rates were recorded at rest and peak performance, and their stroke volumes and cardiac outputs were derived. It was hypothesized that all variables at rest and peak performance would be higher among those who consumed caffeine prior to the experiment as compared to those who did not consume caffeine. At peak exercise, caffeinated individuals exhibited higher heart rates, stroke volumes, and cardiac outputs than those who were non-caffeinated. At rest, caffeinated individuals only showed higher stroke volumes, while all other variables remained relatively similar to those of non-caffeinated individuals. Because of conflicting results from other studies of caffeine on blood pressure, heart rate, and cardiac output, it can only be definitively concluded that stroke volume is impacted by caffeine at rest and immediately after peak exercise.

Introduction

Coffee is consumed daily by millions of people across the United States for its main ingredient: caffeine.



Liat Litwin: *Caffeine Heart*

Caffeine can be consumed in multiple forms to increase short-term energy and metabolism. Athletes are notorious consumers who frequently ingest caffeine through energy drinks. It has been established that caffeine has an effect on cardiovascular variables, such as blood pressure and heart rate, at rest (Hartley et al., 2004).

While exercising, other metabolic functions like an increase in plasma epinephrine and norepinephrine may amplify the effects of caffeine (Van Soren et al., 1998; Robertson et al., 1978). Epinephrine is commonly known for its ability to increase metabolic

rate and maintain its elevation (Ratheiser et al., 1998). In addition to promoting epinephrine levels during exercise, caffeine also exerts its effect when the consumer is at rest. Adenosine decreases neuronal activity, lulling the brain to rest (Dunwiddie et al., 2001). Caffeine inhibits adenosine by binding its receptors in the brain, thus prolonging the consumer's alertness and attention, making him/her feel more awake for a longer period of time (Ribeiro et al., 2010).

While the effects of caffeine on the human brain and metabolism at rest and during exercise are clearly known, this study aims to shed light on the effects of caffeine on cardiovascular variables at rest and after exercise. During exercise, cardiovascular variables such as blood pressure (BP), heart rate (HR), stroke volume (SV), and cardiac output (CO) rise to accommodate the body's increased demand for oxygen (Burton et al., 2004). Due to caffeine's excitatory effect on neuronal and metabolic variables in the body, it is hypothesized that those who did consume caffeine (caffeinated) prior to exercising will exhibit (1) higher resting and peak HR, (2) higher resting and peak MAP, (3) higher resting and peak SV, and thus (4) higher resting and peak CO than individuals who did not consume any caffeine (non-caffeinated) prior to exercising.

In this experiment, 93 individuals between ages of 20 and 51 were asked to exercise until exhaustion. From this pool of individuals, 42 were randomly chosen for further statistical analysis based on their caffeine consumption prior to the experiment. This subset was divided into two groups of 21 based on whether or not caffeine was consumed prior to the experiment. Their BP and HR were measured before and after exercising to derive other cardiovascular variables.

Materials and Methods

Before exercise commenced, basic information was collected about the participants such as age, height, weight, and if they consumed caffeine prior to the experiment. The sources of caffeine included soda, a cup of coffee, and Redbull™ energy drinks, and there was no time-specified latent time between consumption of caffeine and the participation in the experiment. Subjects ranged from ages 19-44 (mean = 23.33). A total of 21 subjects consumed caffeine prior to the exercise trial (M=13, F=8) while the other 21 did not (M=7, F=14). The average body mass index (BMI) for the 42 subjects was 23.12.

Key Points: Adenosine Receptors

- Adenosine receptors are a G-protein coupled receptors, with adenosine acting as the ligand
- Caffeine also binds to the adenosine receptor, acting as a potent antagonist, and stimulates release of excitatory neurotransmitters1

Reference

Fredholm BB, Bättig K, Holmén J, et al. Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. Pharmacol Rev 1999; 51:83.

Each participant got on the stationary bicycle ergometer and his/her resting HR and BP was obtained. A facemask was placed on the participant to measure respiratory variables. It contained three ports with one-way valves. The subject inhaled air through the two side ports and air was exhaled through the third port. The third port was connected to a tube leading to a gas analyzer and a spirometer. The data from these inputs was recorded with an iWorx box and Labscribe2 setup.

Data was recorded at rest for a one-minute interval to gauge resting respiratory variables. After the resting period, the subjects were asked to pedal and maintain a speed of 80 rpm throughout the experiment and not stop until the end of the experiment. Once the subjects felt exhausted, they were allowed to stop pedaling.

The subjects pedaled at level 0 for two minutes for warm-up data to be recorded and to allow them to get used to pedaling on the stationary bicycle. After the warm-up, the experiment commenced at level 0. The subjects pedaled at increasing work intensity levels for one-minute intervals. At the end of each interval, HR was recorded from the bicycle ergometer. When the subjects could no longer maintain a speed of 80 rpm, the experiment ended, and the BPs of the subjects

were immediately recorded. The subjects were then permitted to cool down by pedaling leisurely for at least one minute.

The cardiovascular variables from the exercise were calculated via a series of equations. Below is the cascade of equations and variables used to calculate stroke volume (SV) in Equation 1.

Equation 1. $SV = P_{pulse} - AD$

Equation 2. $P_{pulse} = P_{sys} - P_{dia}$

Equation 3. $AD = 1.5$ ml/mmHg

Arterial distensibility (AD) is the measure of how well the arteries stretch. According to Equation 3, the default value of 1.5 ml/mmHg was applied to this variable. In Equation 2, BP measurements from before and after the exercise experiment were used to calculate $P_{pulse.}$

For the purposes of this study, mean arterial pressure (MAP) was used as a proxy to gauge BP from the participants before and after the exercise experiment. It was calculated using Equation 4.

Equation 4. $MAP = P_{dia} + \frac{P_{pulse}}{3}$

The final cardiovascular variable that was derived was cardiac output (CO). CO was derived from using Equation 5.

Equation 5. $CO = HR \times SV$

These variables were calculated and recorded for each individual. SV, MAP, and CO were recorded for rest and peak performance. For the purposes of this experiment, data from 42 individuals were chosen from a pool of 93 participants to examine the acute effects of caffeine on the cardiovascular system in response to exercise. 21 random individuals who reported consuming caffeine prior to the experiment were chosen to match 21 other random individuals who reported not consuming caffeine prior to the experiment. The data were represented with unpaired t-tests and p-values to convey statistical significance.

Results

The purpose of this experiment was to examine how the consumption of caffeine immediately impacts certain cardiovascular variables in response to exercise. The cardiovascular variables HR, SV, CO, and MAP were compared among 21 caffeinated and 21 non-caffeinated individuals at rest and peak exercise.

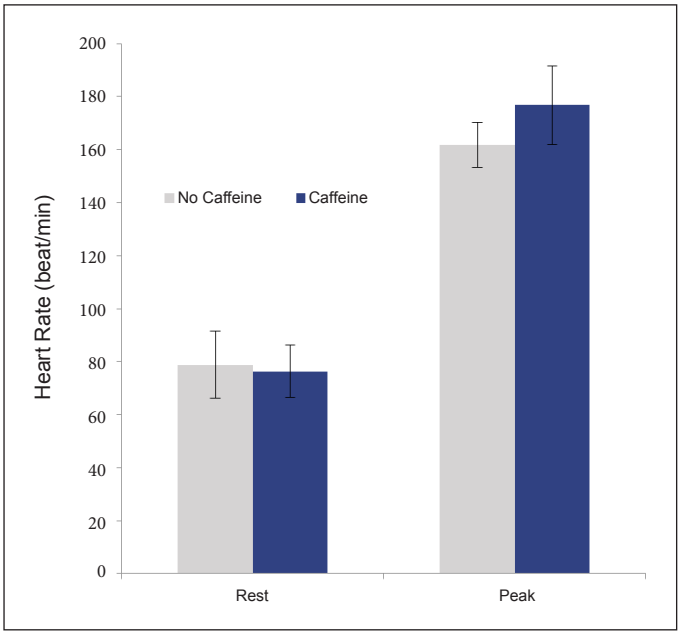


Figure 1: Resting and peak heart rates (HR) for subjects who did and did not consume caffeine prior to exercising.

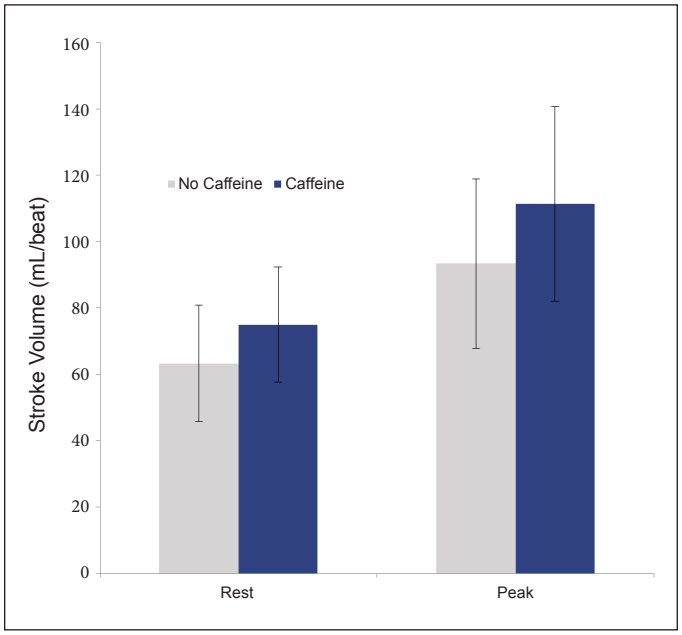


Figure 2: Resting and peak stroke volumes (SV) for subjects who did and did not consume caffeine prior to exercising.

At peak performance, the hypothesis was supported because the average HRs of individuals who consumed caffeine was observed to be ~8.5% ($p = 0.0002$) higher than those who did not consume caffeine prior to the experiment. These data are plotted and depicted in Figure 1. However, the resting heart rates of individuals who consumed caffeine did not differ from those who did not consume caffeine ($p = 0.481$). In fact, according to Figure 1, there appeared to be a slight, yet insignificant, decrease in resting HR among caffeinated participants, undermining the hypothesis. Yet, caffeine still seemed to be a factor in raising peak HR in response to exercise.

Unlike the results for HR, SV seemed to be greater for caffeinated individuals at rest and peak performance. In Figure 2, the caffeinated individuals showed an average SV that was ~19% higher at rest than those of the non-caffeinated individuals at rest ($p = 0.036$). And at peak exercise, those who consumed caffeine showed significantly higher SVs than those who did not consume caffeine ($p = 0.040$) by ~19.3%. Both results supported the hypothesis that the caffeinated individuals would express higher SVs than those who were non-caffeinated.

Figure 3 conveys results similar to those recorded for HR in Figure 1. At peak performance, the COs of the caffeinated group of participants were significantly higher than those of the non-caffeinated group ($p = 0.007$) by 30%, supporting the hypothesis. At rest, there was a slight increase in CO among those who were caffeinated, but these data did not convey any statistical significance ($p = 0.098$). Therefore, because the resting CO of caffeinated individuals was not significantly greater than those of non-caffeinated individuals, these data did not support the hypothesis. In addition to influencing peak heart rate, caffeine still appears to be a factor in increasing the CO at peak exercise.

The BPs of participants were measured using MAP as a proxy. The MAPs of caffeinated individuals did not seem to differ from those who were non-caffeinated at rest ($p = 0.827$) or peak exercise ($p = 0.263$). While Figure 4 suggests very slight differences in MAP for both variables, these differences were not statistically significant. The results for MAP between caffeinated and non-caffeinated individuals do not support the hypothesis and suggested that caffeine has little influence on BP during exercise.

Discussion

Caffeine is a staple resource in the lives of many university students for established reasons. It is used for a simple energy boost while studying, exercising, and waking up in the morning. The purpose of this

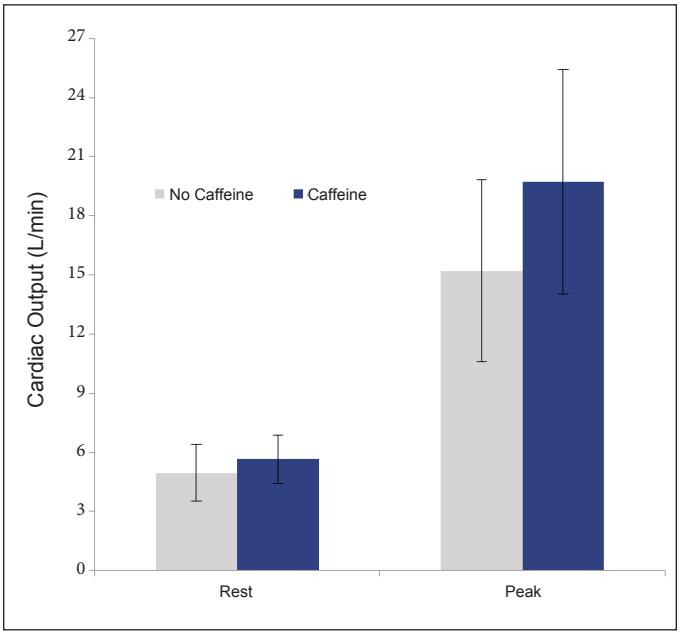


Figure 3: Resting and peak cardiac outputs (CO) for subjects who did and did not consume caffeine prior to exercising.

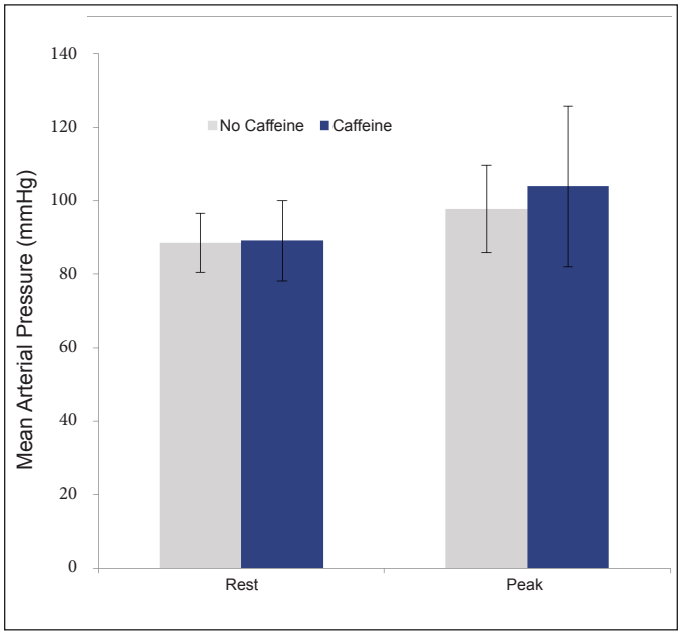


Figure 4: Resting and peak mean arterial pressures (MAP) for subjects who did and did not consume caffeine prior to exercising

study was to examine the effects of caffeine on the cardiovascular variables HR, SV, CO, and MAP at rest and immediately after peak exercise. It was hypothesized that all four variables would be higher at rest and after peak exercise in individuals who consumed caffeine prior to the experiment. According to the results, it appears that caffeine does have an impact on peak HR, SV, and therefore CO as well as resting SV, showing increases in each by 8.5%, 19%, 30%, and 19.3%, respectively. However, resting HR and CO along with both MAP measurements seemed to not differ significantly between non-caffeinated and caffeinated individuals, undermining the hypothesis. These data provoke a reason to believe that caffeine increases blood flow throughout the body by affecting the heart.

Stroke volume was expected to increase due to caffeine's ability to increase arterial stiffness (Mahmud et al., 2001). Equation 1 demonstrates that arterial distensibility affects SV. The stiffening of the arteries by caffeine decreases arterial distensibility and, therefore, increases SV. This relationship explains the significant increase ($p < 0.05$) observed at both rest and peak exercise.

While caffeine is known to raise the HR of individuals, it has been reported that HR does not significantly differ between caffeinated and non-caffeinated individuals in response to exercise (Daniels et al., 1998). Yet, caffeine has been shown to increase ambulatory heart rate (Green et al., 1995). While there are conflicting studies, this study measured that caffeinated individuals exhibited an ~8.5% increase in average HR compared to non-caffeinated individuals. The acute effects of HR may be variable when considering the heart's response to exercise.

According to Equation 5, CO is directly proportional to both HR and SV. Thus, the pattern for CO mirrored that of HR because of their direct relationship. At rest, average CO and HR did not exhibit a significant increase in caffeinated individuals; however, peak exercise yielded a significant increase in average CO and HR. This relationship can be understood using the general values of the two variables. While there are no coefficients in Equation 5 that would affect the value of CO, HR tends to be a greater number than SV, thereby carrying more weight in the equation. The variability of average HR between the two populations is too low for the increase in average SV of caffeinated individuals to significantly impact the average CO. If HR, rather than SV, were increased by 19% at rest, the difference in average CO between the two populations would have been significant. Because HR has a greater impact on CO, the CO-HR mirror is a valid pattern.

This study is not without its limitations. In a previous study of a similar experimental protocol, caffeinated individuals demonstrated a higher average MAP at rest and after peak exercise by 11% and 5%, respectively (Daniels et al., 1998). Daniels et al. have also shown that caffeine increases BP among resting individuals

Caffeine Content of Various Beverages and Substances

Brewed coffee
- Serving size: 235mL
- Content: 133mg (range: 102-200)

Brewed tea
- Serving size: 235mL
- Content: 53mg (range: 40-120)

Espresso
- Serving size: 30mL
- Content: 40mg (range: 30-90)

Red Bull
- Serving size: 245mL
- Content: 80mg

5-hour ENERGY
- Serving size: 60mL
- Content: 215mg*

* Reported by Consumer Reports

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Harland BF. Caffeine and nutrition. Nutrition 2000; 16:522.

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Center for Science in the Public Interest. Caffeine Content of Food and Drugs.

as compared to individuals who consumed a placebo. However, in the present study, the lack of a difference in MAP between caffeinated and non-caffeinated individuals is anomalous. No significant changes in MAP were seen between the two populations at rest and peak exercise. One interpretation of this result is this study did not control how much caffeine each individual consumed. Also, the latent BP conditions, regular caffeine consumption habits, and the period between caffeine consumption and the trial were not controlled. While the effects of caffeine were present in other variables regardless of the amount consumed, latent BP conditions, or latent caffeine consumption habits, it remains anomalous as to why MAP, and therefore BP, were not affected.

Another limitation of this study is the imbalance between male and female participants in each studied group. Additionally, the design of the study is an unpaired t-test whereas some may view a paired t-test to be more appropriate.

It can be concluded that caffeine has an impact on SV at rest and immediately after peak exercise. Additionally, caffeine's effect on HR and, therefore, CO may be varied when comparing results to other literature. We cannot conclude whether caffeine has an effect on BP and, therefore, MAP because this study's results conflict with those in other literature. Future experiments may aim to explore how certain respiratory variables are impacted by the consumption of caffeine before and after peak exercise in order to further establish caffeine's benefits and detriments in relation to physical training.

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