AUCTORES
Globalize your Research

Clinical Medical Reviews and Reports

Gary Merrill *

Open Access

Research Article

Caffeine and the Cardiovascular System of University Students

Patel S, Farooq, S, Oyedokun T, Mali K, Alatrash R, Harmon P, Hudgins R, Bernard L, Merrill GF*

Cell Biology and Neuroscience, Nelson Biological Laboratories, 604 Allison Road, Piscataway, NJ 08855

***Corresponding Author:** Merrill, G.F., Department of Cell Biology and Neuroscience, Division of Life Sciences, School of Arts and Sciences, Rutgers University, New Brunswick/Piscataway, New Jersey 08855.

Received Date: 19 November 2025; **Accepted Date:** 22 December 2025; **Published Date:** 12 January 2026

Citation: Patel S, Farooq, S., Oyedokun T, Mali K, Alatrash R, et al., (2026), Caffeine and the Cardiovascular System of University Students, *Clinical Medical Reviews and Reports*, 8(1); **DOI:10.31579/2690-8794/294**

Abstract

In this experiment we aimed to determine whether caffeine has any effects on the cardiovascular systems of university students who consume more than 200 mg of caffeine per day, and whether these effects differ from students who consume less than 200 mg per day. Twenty-one caffeine-consuming (e.g., coffee, energy drinks, soda, tea) juniors and seniors were investigated. All data were pooled, but most participants were female (n=17). While many influences of caffeine on the human body can be investigated, we chose to limit our observations to selected cardiovascular variables. After resting, baseline data were collected, each student consumed 200 mg of caffeine (oral tablet). Post-caffeine data were collected 60 and 120 minutes later. Blood flow to a finger was measured plethysmographically, and systemic arterial blood pressure was measured with a cuff and stethoscope. Heart rate was measured from pulsatile pressure waveforms, and peripheral vascular resistance was calculated as mean arterial pressure divided by blood flow. All data were continuously recorded on an automated data acquisition system and displayed on a desktop computer monitor. Finger volume and blood flow were markedly and consistently reduced at 60- and 120-minutes post-caffeine. Peripheral vascular resistance to blood flow was simultaneously and significantly increased. We conclude that caffeine, at 200 mg, significantly disrupts homeostasis of the young adult cardiovascular system, and consequently, is not good for the heart and circulation.

Keywords: caffeine-mediated vasoconstriction; reduced blood flow; ischemia

Introduction

Besides water (consumed directly from the tap/cooler, etc.) coffee and other caffeine-containing drinks are the adults' beverages of choice. Globally, most adults consume around three espressos worth of caffeine a day (200 mg x 3 = 600 mg/day) with this rate increasing in industrialized nations (1). On average, university students begin caffeine consumption at ages 14-15, but some students might be exposed to caffeine as early as 5 years (current study; cultural norms). As a result of early exposure, young adults are conditioned consumers of the drug by ages 20-21, which typically overlap with junior and senior years of college. The ever-growing popularity of caffeinated drinks is perpetuated through social media and the day-to-day lives of college students. One would not be hard pressed to find many college students with a caffeinated beverage in hand. This should raise concern for the health impacts of caffeine on youth and young adults.

On the Rutgers University Busch Campus (sciences and medicine), and between classes, it is common to see 30-45 students standing on-line at a Starbuck's Truck waiting for their next hit of caffeine. This occurs during the heat and humidity of spring/summer and the cold of fall/winter. Not only do many such students lose 45-60 minutes of valuable time waiting, but prolonged exposure to cold and heat are known to have profound effects on the bodies' thermoregulation (2). This exposure is outside of the homeostatic disruption caused by hot caffeinated beverages (3). Studies conducted by this lab have shown that caffeine significantly increases peripheral vascular resistance, elevates arterial blood pressure, and markedly reduces peripheral blood flow and reactive hyperemia in young adults (4-7). Through competitive inhibition of the adenosine receptor, caffeine can impair cardiovascular autoregulation and can predispose the heart to arrhythmias (8-13). It seems reasonable to speculate that, without caffeine addictions, the general health of such students would be improved.

Methods

Twenty-one young adult university students were investigated (17 women/4 men). All were enrolled in Systems Physiology Laboratory at Rutgers University during Fall Semester, 2025. This is an experimental physiology laboratory where experimentation is the focus of pedagogy and where students voluntarily register for the course. The experiments are safe and innocuous. Participants and their data can never be identified because we do not retain/store information permanently. For these and related reasons, it has been determined that the course is exempt from IRB review and approval.

After arriving at the Dr. Norman and Syril Reitman Systems Physiology Teaching Laboratory, Rutgers University, students were seated comfortably at an experimental bench. A finger pulse plethysmograph was attached to either the index or middle finger (model TN1012ST, AD Instruments, Colorado Springs), and a pressure cuff with mercury manometer (model MLT0699, AD Instruments, Colorado Springs) was placed on the upper arm of the same limb to which the plethysmograph was attached. An electronic stethoscope was also used to confirm the MLT0699 data (model ANR2, Think Labs, Centennial, CO).

Subsequently, laboratory lights were dimmed and students were instructed to rest quietly for 15-20 minutes as their cardiovascular systems achieved baseline, steady-state conditions. A baseline set of data were then collected and students were divided into two groups (by prior survey results): those who regularly consume more than 200 mg/day of caffeine and those who consume less than that. The data included measurements of: digital blood volume, digital blood flow, heart rate, systolic, diastolic, and systemic mean arterial blood pressure. Peripheral vascular resistance was calculated as mean arterial pressure divided by blood flow. Beyond this collection of data, each student consumed a 200 mg caffeine pill. They rested quietly and two additional sets of data were collected at 60- and 120-minutes post-caffeine.

All data were continuously captured by an electronic data acquisition system (Model 26T, ADInstruments, Colorado Springs) coupled with a desktop computer running Lab Chart software (v. 8.1.30, July, 2024). Our null hypothesis (H_0) stated that cardiovascular function would not differ between individuals consuming >200 mg/day of caffeine and those consuming <200 mg/day. The alternative hypothesis (H_a) stated that cardiovascular function would differ between these two groups. These data were then exposed to statistical evaluation. They are reported here as means plus or minus one standard error of the mean ($\bar{x} \pm$ s.e.m.). For each variable we compared data from the two groups (> 200 mg/day vs < 200 mg/day). Statistically significant differences were identified at the physiological standard, $P<0.05$.

Results

Selected data from a pre-experiment survey are presented in Table 1.

	>200 mg/day	<200 mg/day
m/f (%)	25/75	15/85
% (n=21)	38	62
time caffeine consumed (%n)		
6-12 a.m.	45	43
12-6 p.m.	30	32
6-12 p.m.	25	25
Culture/ethnicity	C>A>B>H>I>L>O	C>A>B>H>I>L>O

m/f, male to female ratio of subjects; C, Caucasian; A, Asian, B, Black; H, Hispanic; I, Indian; L, Latino, O, Other (e.g., Middle Eastern)

Table 1: Selected characteristics/data from subjects who completed a pre-experimental caffeine survey.

From this survey we were able to determine that the students' primary, caffeine-containing, beverage of choice was coffee (vs tea, energy drinks, soda). Generally, college students preferred consuming such beverages between 6-12 a.m., and noon-6 p.m. Regardless of this temporal preference, a large portion of this sample size of twenty-one students, also consumed these drinks as late as 6-12 p.m. We also learned from the survey that both male and female students began their caffeine habits between 14-15 years of age, although some started as early as 5-6 years (cultural norms, personal communications).

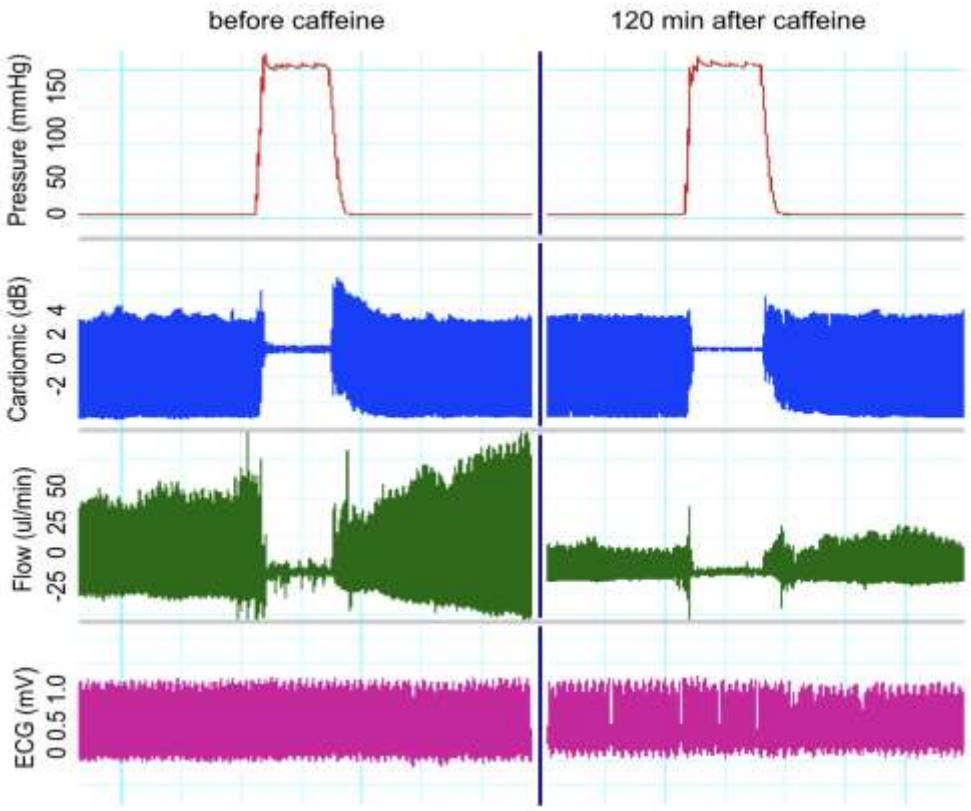

Figure 1: A horizontally-compressed sample tracing from one subject. Note the marked reduction in baseline and reactive hyperemic blood flow 120 min post-caffeine.

A sample tracing of a representative experiment is illustrated in Figure 1. This tracing was compressed horizontally so the entire 120-minute period could be displayed. This prevents examination of the record in detail, but it allows the crucial comparison, i.e., pre-caffeine data vs those obtained 120 minutes post-caffeine. Most notably, one can see a marked caffeine-mediated decrease in both finger volume and blood flow. Data for heart rate (HR, cycles per minute, cpm) are presented in Figure 2.

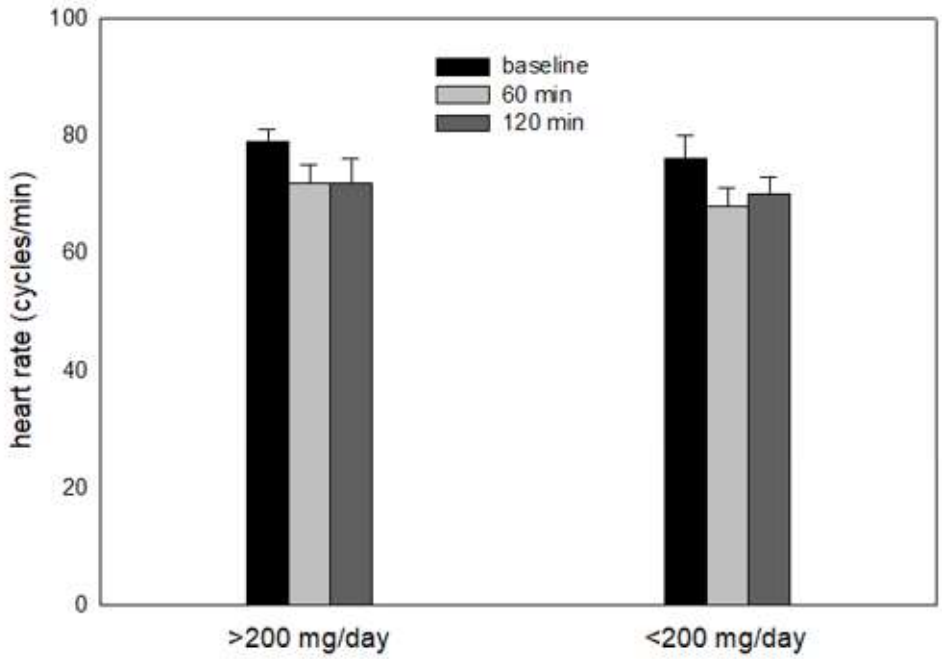

Figure 2: Caffeine's absence of effect on heart rate in young adult university students 120 minutes after exposure to 200 mg caffeine (oral).

Despite some variability, there were no statistically significant differences over time between the two groups. For example, under baseline conditions HR was 81 ± 4 vs 75 ± 3 in the >200 mg/day vs < 200 mg/day groups, respectively. After 120 minutes post-caffeine, corresponding numbers were 70 ± 4 for both groups. Finger blood volume (Figure 3) and blood flow (Figure 4)

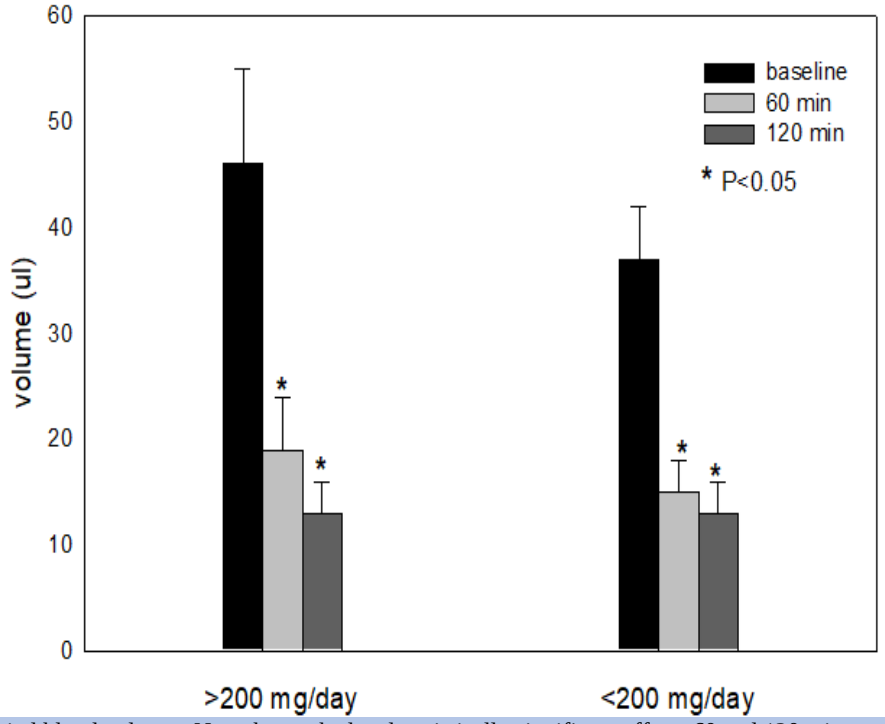

Figure 3: Caffeine and digital blood volume. Note the marked and statistically significant effects 60 and 120 minutes after exposure. Such effects are often seen as early as 15-30 minutes post-caffeine.

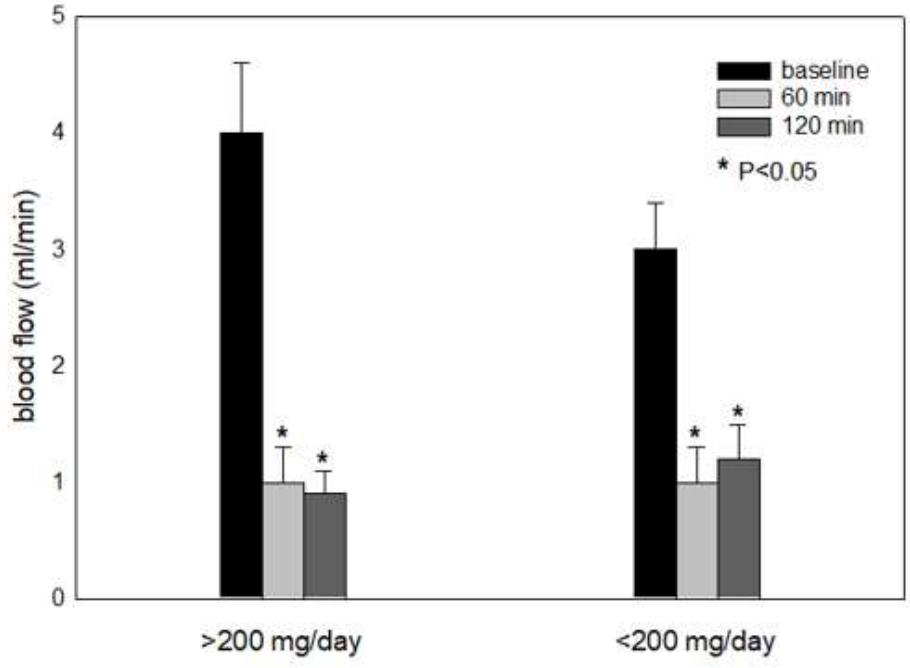

Figure 4: Caffeine and digital blood flow. Note the marked and statistically significant effects 60 and 120 minutes after exposure. Such effects are often seen as early as 15-30 minutes post-caffeine.

were markedly and significantly (P<0.05) reduced at both 60- and 120-minutes post-caffeine. For example, in the baseline, pre-caffeine state finger volumes were 46 ± 9 and 43 ± 4, respectively for the >200 mg/day and <200 mg/day groups. By 60 minutes, volume decreased to 18 ± 4 in both groups, and by 120 minutes, volume for >200 mg/day vs < 200 mg/day groups were 13 ± 3 and 17 ± 6 respectively (P<0.05, vs corresponding baseline values). For finger blood flow (ml/min) numbers were: 4.0 ± 0.6, 3.0 ± 0.4 (baseline), 1.0 ± 0.3 for both groups (60 minutes, P<0.05 vs corresponding baselines),

and 0.9 ± 0.2, 1.2 ± 0.4 (120 minutes, P<0.05 vs corresponding baselines) (Table 2).

	Heart rate (pulse/min)		Volume (uL)		Flow (mL/min)		PRU (mmHg/mL/min)	
	High	Low	High	Low	High	Low	High	Low
0 Minutes	Mean: 80.75 SEM: 76.6 - 84.9	Mean:75.62 SEM: 71.94 - 79.3	Mean:46.22 SEM 37.37 -55.07	Mean:42.98 SEM 38.55 - 47.41	Mean:3.66 SEM 3.03 - 4.29	Mean:3.26 SEM 2.89 - 3.63	Mean:28.21 SEM 23.12 - 33.3	Mean:31.12 SEM 27.31 - 34.93
60 Minutes	Mean:70.25 SEM 66.1 - 73.2	Mean:67.54 SEM 63.86 - 70.73	Mean:18.81 SEM 9.96 - 23.64	Mean:18.27 SEM 13.84 - 22.6	Mean:1.25 SEM 0.62 - 1.53	Mean:1.28 SEM 0.91 - 1.59	Mean:93.64 SEM 73.6 - 113.68	Mean:133.90 SEM 103.66 - 164.14

Table 2: Select cardiovascular variables of significance with noted means and SEM ranges during the 0 minute and 60-minute intervals

Figures 5, 6, and 7 present data for systemic arterial blood pressures: $P_{\bar{a}}$ (mean arterial pressure, mmHg), P_s (systolic pressure, mmHg), and P_d (diastolic pressure, mmHg). In all cases post-caffeine, there were numeric trends towards an increase. However, at the physiological standard, P<0.05, none of these achieved significance. For example, P_s rose from a baseline value of 105 ± 4 to 113 ± 3 mmHg (>200 mg/day), and from 112 ± 4 to 116 ± 6 mmHg (<200 mg/day) 120 minutes post-caffeine. Similar numbers were obtained at 60 minutes post-caffeine.

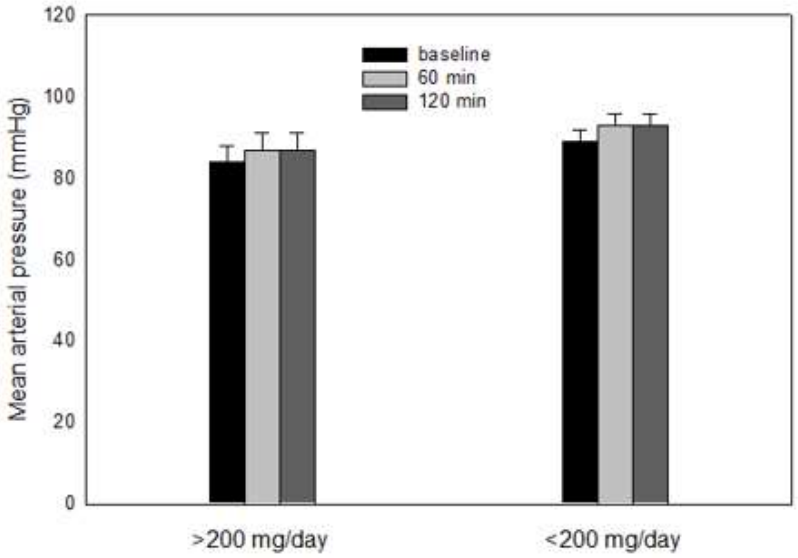

Figure 5: Caffeine and systemic mean arterial blood pressure. Although there are numeric tendencie towards an increase, these are not statistically significant at P<0.05.

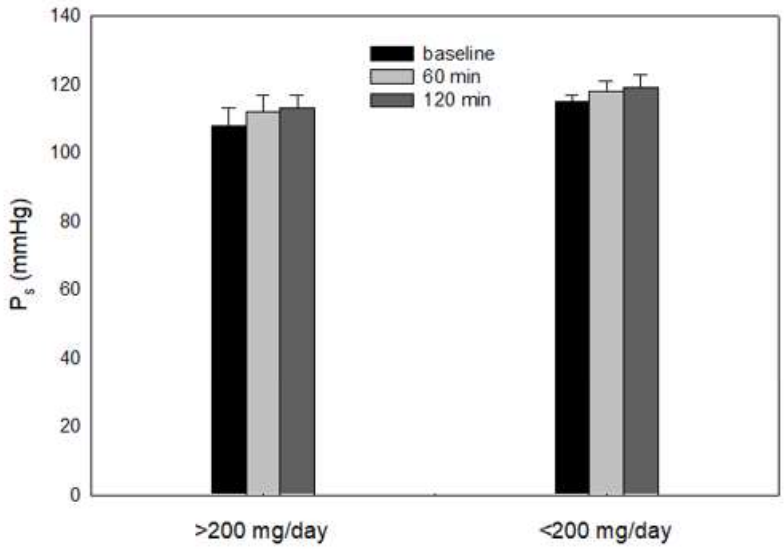

Figure 6: Caffeine and systolic blood pressure. Although there are numeric tendencies toward an increase, these are not statistically significant at P<0.05.

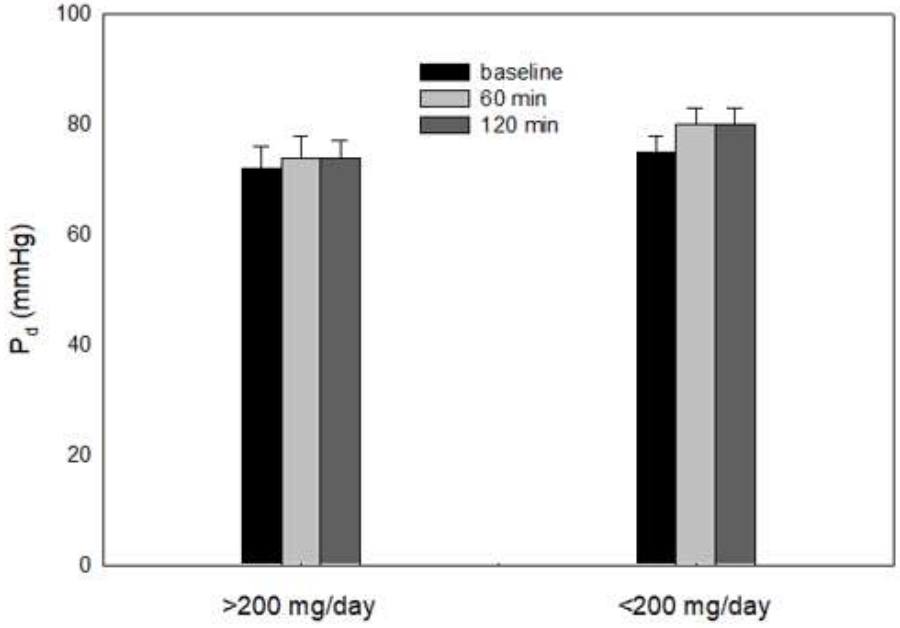

Figure 7: Caffeine and diastolic blood pressure. Although there are numeric tendencies toward an increase these are not statistically significant at P<0.05.

Peripheral vascular resistance (Figure 8) was calculated as: $P_{\bar{a}}$ (mmHg) ÷ ml/min (blood flow), and was expressed in units of mmHg/ml/minute as is standard. By inspecting Figure 8 one can see that at 60 and 120 minutes, caffeine caused potent and statistically significant increments in calculated peripheral vascular resistance. These were not modest changes. For example, in the <200 mg/day group, resistance rose from 31 ± 4 (baseline) to 133 ± 30 (60 minutes, P<0.05, a mean increase of 329%) and 149 ± 40 (120 minutes, P<0.05, a mean increase of 381% from baseline). A similar trend is observed in the >200 mg/day group who had a mean increase of 232% from baseline at 60 minutes and a 485% increase from baseline at 120 minutes (Table 2).

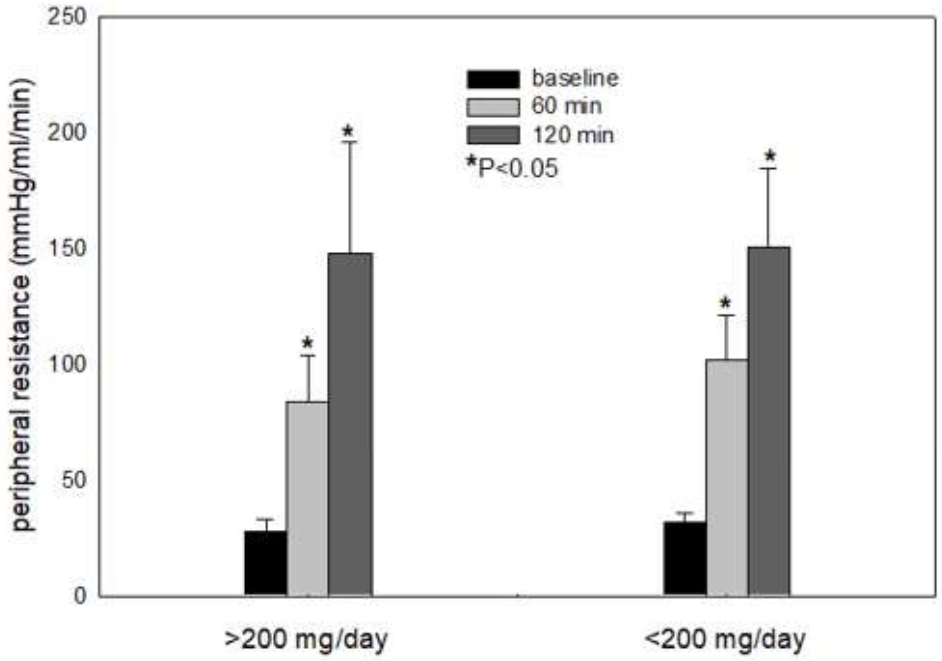

Figure 8: Caffeine and calculated peripheral vascular resistance. Note the marked and statistically significant increments at both 60- and 120-minutes post-caffeine.

Discussion

Recent studies have illuminated the increasing link between the use of caffeine and disorders in physicians, particularly females (14). This emphasizes the importance of educating our youth and young adults on the harmful side effects of consuming caffeine (14). We are interested in the physical health of our students, and try to encourage them to improve/sustain it. Over the years we have interacted with students on the Rutgers University Campus, as well as in courses such as Systems Physiology Lecture, Systems Physiology Laboratory, and Advanced Physiology. All students in these formal courses are bright, well-educated juniors and seniors. Many of them are planning careers in medicine and allied health professions (e.g., Physicians, Physician's Assistant, Nurses, Physical Therapy, Doctor of Physical Therapy, et al).

Unfortunately, like many adults in the world, university students believe they need to consume caffeine-containing beverages to wake up, to be energized, and to do well in their careers and on midterm and final exams, etc. (personal communications). Thus, we have become interested in their caffeine-consuming habits, and have wondered if caffeine has important effects on the healthy, young adult cardiovascular system. The current experiment was part of our continuing interests.

We have been unable to reconcile what others have published about caffeine and heart rate in adults, young and old, student and non-student. For example, if one does a literature search (PubMed, Google Scholar) using key words/phrases such as: 'caffeine decreases heart rate', 'caffeine increases heart rate', 'caffeine has no effect on heart rate', they will probably be confused also. The confusion will come because there is a large literature supporting all three conclusions, i.e., caffeine increases, decreases, and has no effect on heart rate (15-17). As physiologists having access to experimental and teaching laboratories, and a reasonable understanding of experimentation and function of the human body, we have tried to answer the question ourselves.

In the current experiment we have found that whether the university student consumes less than 200 mg/day or more than 200 mg/day, caffeine does not affect heart rate, at least in the first 120 minutes post-consumption. These results are consistent with other results we have published in the past few years (4-8). This appears to be the case whether caffeine is consumed as a pill, or in coffee, energy drinks, soda, tea, etc. We have also found our conclusion to be true over the range of doses of 100-400 mg caffeine (we have not examined lower or higher doses) (3-7). We have opinions why others get conflicting results, but our opinions are probably best reserved for a philosophical/psychological debate on the subject.

Equally consistent in our hands, caffeine is a potent peripheral vasoconstrictor. We see these effects in the vascular beds of both fingers and toes of university students, and at all doses studied. The vasoconstriction occurs as early as 15 minutes post-consumption of caffeine, and is sustained through 60-120 minutes. These undesirable circulatory actions occur in young women and young men, across all cultures, and throughout the day. They are as evident in the early morning as later in the afternoon, and during the evening hours (5-8). If this is true of the drug's actions on blood supply in the digits, we wonder what effects caffeine is concurrently having on the coronary, cerebral, retinal, carotid/aortic bodies, and other important blood supplies in the university student and others.

In the current experiment, caffeine did not significantly alter blood pressure, whether expressed as systolic, diastolic, or mean arterial pressure. There was, however, a clear tendency towards elevated blood pressure, but the numbers did not achieve statistical significance. These pressure-related results are also consistent with our earlier observations (18-20). Among the variables that are directly correlated with elevated blood pressure in university students are: male gender, body weight, and time of day (our work, others). Thus, one might anticipate that an overweight, young adult male, consuming caffeinated drinks in the late afternoon/evening hours, should expect a greater caffeine-mediated impact on his cardiovascular system (e.g., elevated peripheral vascular resistance, reduced tissue blood flow), than

would a young woman of a healthy weight consuming caffeine in the morning hours. Of course, more work is needed on such demographics/speculations to draw any definitive conclusions.

Consistently, we have also observed caffeine-mediated reductions in reactive hyperemic blood flow following brief periods of occlusion/obstruction of flow. That is, if an organ/tissue is made ischemic in the presence of caffeine, reperfusion of that tissue post-obstruction of flow will be reduced when compared with the caffeine-free state (6, 21-22). This is one reason why physicians/medicine currently recommend patients abstain from caffeine 4-24 hours before undergoing a treadmill stress test, and/or cardiac catheterization tests/procedures such as placing stents in coronary arteries (23). There is mounting evidence that caffeine constricts both coronary and cerebral vasculature (24-28).

Summary and conclusions

In junior and senior university students, daily consumption of caffeine harmfully affects their cardiovascular systems. The effects are potentially dangerous. For example, whether they consume less or greater than 200 mg/day, caffeine profoundly and significantly decreases digital blood flow. It simultaneously increases calculated peripheral vascular resistance. These are undesirable effects in the young adult cardiovascular system and should be avoided.

References

1. Samoggia A, Rezzaghi T. (2021). The consumption of caffeine-containing products to enhance sports performance: An application of an extended model of the theory of planned behavior. Nutrients.13:344.
2. Beker BM, Cervellera C, De Vito A, Musso CG. (2018). Human physiology in extreme heat and cold. Int Arch Clin Physiol. 1(1):1-8.
3. Merrill G. (2021). Caffeine and blood flow. Clin Med Rev Rep. 3:1-7.
4. Merrill G. (2020). Caffeine and peripheral blood flow. Clin Med Rev Rep. 2:1-4.
5. Merrill GF, Costea DM, Sharp VA. (2019). Caffeine and pressure flow autoregulation. World J Cardiovasc Dis. 9:253-266.
6. Merrill GF, Costea DM, Sharp VA. (2019). Caffeine and reactive hyperemia. World J Cardiovasc Dis. 9:437-448.
7. Merrill GF. (2023). Ischemia and reperfusion with and without caffeine. Clin Case Rep. 6:267-274.
8. Merrill GF. (2017). Experimentally-induced ventricular arrhythmias. Int J Physiol Pathophysiol Pharmacol.9:202-209.
9. Young MA, Merrill GF. (1982). Comparative effects of adenosine and nifedipine in rabbit vascular smooth muscle. Can J Physiol Pharmacol. 61:1057-1062.
10. Tozzi CA, Merrill GF. (1986). Differential effects of adenosine and verapamil on histamine-induced vascular contractions. Can J Physiol Pharmacol. 64:679-682.
11. Merrill GF, Haddy FJ, Dabney JM. (1978). Adenosine, theophylline and perfusate pH in the isolated, perfused guinea pig heart. Circ Res. 42:225-229.
12. Merrill GF, Young MA, Tozzi CA, Grosso PC, Marcus KM. (1982). Adenosine dilation and adrenergic constriction of coronary blood vessels: PCO_2 effects. Artery.10:395-411.
13. Young MA, Merrill GF. (1982). Differential effects of adenosine and hypoxia on potassium-induced coronary vasodilation in isolated, perfused guinea pig hearts. Blood Vessels.19:292-301.
14. Sağlam B, Tural E, Dayan A. (2024). Exploring the link between physicians' caffeine use disorders with sleep quality and professional burnout: A cross-sectional study. BMC Health Serv Res. 24:909.
15. Green PJ, Kirby R, Suls J. (1996). The effects of caffeine on blood pressure and heart rate: A review. Ann Behav Med. 18:201-216.
16. McClaran SR, Wetter TJ. (2007). Low doses of caffeine reduce heart rate during submaximal cycle ergometry. J Int Soc Sports Nutr. 4:11.
17. Newcombe PF, Renton KW, Rautaharju PM, Spencer CA, Montague TJ. (1988). High-dose caffeine and cardiac rate and rhythm in normal subjects. Chest. 94:90-94.
18. Teng CL, Lim WY, Chua CZ, Teo RS, Lin KT, Yeo JC. (2016). Does a single cup of caffeinated drink significantly increase blood pressure in young adults? A randomised controlled trial. Aust Fam Physician. 45:65-68.
19. Waring WS, Goudsmit J, Marwick J, Webb DJ, Maxwell SR. (2003). Acute caffeine intake influences central more than peripheral blood pressure in young adults. Am J Hypertens.;16:919-924.
20. Ammar R, Song JC, Kluger J, White CM. (2021). Evaluation of electrocardiographic and hemodynamic effects of caffeine with acute dosing in healthy volunteers. Pharmacotherapy.21:437-442.
21. Melik Z, Princi T, Grill V, Cankar K. (2019). The effect of caffeine on cutaneous post-occlusive reactive hyperaemia. PLoS One. 14: e0214919.
22. Matsumoto H, Nakatsuma K, Shimada T, Ushimaru S, Mikuri M, Yamazaki T, Matsuda T. (2014). Effect of caffeine on intravenous adenosine-induced hyperemia in fractional flow reserve measurement. J Invasive Cardiol. 26:580-585.
23. Kovacs D, Pivonka R, Khosla PG, Khosla S. (2008). Effect of caffeine on myocardial perfusion imaging using single photon emission computed tomography during adenosine pharmacologic stress. Am J Ther. 15:431-434.
24. Cameron OG, Modell JG, Hariharan M. (1990). Caffeine and human cerebral blood flow: A positron emission tomography study. Life Sci, 47:1141-1146.
25. Mathew RJ, Wilson WH. (1985). Caffeine-induced changes in cerebral circulation. Stroke. 16:814-816.
26. van Dijk R, Ties D, Kuijpers D, van der Harst P, Oudkerk M. (2018). Effects of caffeine on myocardial blood flow: A systematic review. Nutrients. 10:1083.
27. Namdar M, Schepis T, Koepfli P, Gaemperli O, Siegrist PT, Grathwohl R, Valenta I, Delaloye R, Klainguti M, Wyss CA, Lüscher TF, Kaufmann PA. (2009). Caffeine impairs myocardial blood flow response to physical exercise in patients with coronary artery disease and in age-matched controls. PLoS One. 4: e5665.
28. Addicott MA, Yang LL, Peiffer AM, Burnett LR, Burdette JH, Chen MY, Hayasaka S, Kraft RA, Maldjian JA, Laurienti PJ. (2009). The effect of daily caffeine use on cerebral blood flow: How much caffeine can we tolerate? Hum Brain

DOI:10.31579/2690-8794/294