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Acute Effect of Caffeine Intake on Hemodynamics after Resistance Exercise in Young Non-hypertensive Subjects

DIEGO SOUZA, JULIANO CASONATTO, ROBERTO POTON, JEFFREY WILLARDSON, MARCOS POLITO

This study aimed to examine the effect of caffeine on hemodynamics after a resistance exercise session. Fifteen subjects completed two randomly ordered experimental resistance exercise sessions 45 min after the ingestion of either caffeine (4 mg.kg⁻¹) or placebo. Systolic (SBP), diastolic (DBP) and mean (MAP) blood pressures were measured before consuming caffeine; SBP, DBP, MAP, heart rate, stroke volume, cardiac output and peripheral vascular resistance (PVR) were measured immediately before and after each of the sessions; SBP, DBP and MAP were measured for 9 hours after sessions. Caffeine increased ($p < 0.05$) pre-exercise DBP and MAP. In caffeine and placebo conditions significant decreases ($p < 0.05$) were noted in SBP, MAP, and PVR between the pre- and post-exercise time points. Notwithstanding, the mean values for SBP, DBP and MAP during the 9 h of post-exercise monitoring were increased ($p < 0.05$) for the caffeine. In conclusion, the cardiovascular effects of caffeine are different over the post-exercise period after resistance exercise in normotensive young adults.

KEYWORDS blood pressure, strength exercise, caffeine

INTRODUCTION

Caffeine is a commonly consumed substance that is present in many foods and medicinal products (Heckman, Weil, & Gonzalez de Mejia, 2010; Mandel, 2002). Caffeine has also been used as an ergogenic aid (Woolf, Bidwell, & Carlson, 2008) to enhance physical performance (Nehlig & Debry, 1994). Physiological alterations caused by caffeine that may improve physical performance are related to enhanced propagation of neural signals from the brain across the neuromuscular junction (Doherty & Smith, 2005) facilitating the process of contraction (Tarnopolsky, 2008, 2010).

Other mechanisms by which caffeine can improve physical performance include an antagonistic effect on adenosine receptors A1 and A2, inhibition of phosphodiesterase, and greater release of catecholamines and corticosteroids (Davis & Green, 2009). These mechanisms result in an increased heart rate (HR), blood pressure (BP) and peripheral vascular resistance (PVR) (Nurminen, Niitynen, Korpela, & Vapaatalo, 1999). In this context, the intake of caffeine in conjunction with exercise may induce higher cardiovascular responses than exercise with no caffeine (due to additive or synergistic effects). Thus, these cardiovascular responses related to exercise and caffeine intake can provide several implications for health, as a reduction of myocardial blood flow during exercise (Higgins & Babu, 2013). Several studies have examined the acute cardiovascular responses of caffeine

intake in conjunction with aerobic modes of exercise (Ganio, Klau, Casa, Armstrong, & Maresh, 2009; Magkos & Kavouras, 2004).

However, fewer studies have examined the acute cardiovascular responses of caffeine intake in conjunction with resistance exercise. Some studies have found that caffeine intake increases cardiovascular responses both during (Astorino, Rohamnn, Firth, & Kelly, 2007) and after resistance exercise (Astorino, Martin, Schachtsiek, & Wong, 2013). However, the effect of caffeine intake on cardiovascular responses after resistance exercise should receive additional examination, considering that the methodological designs employed in previous studies were limited by a short post-exercise monitoring time (less than 2 hours) or limited variables, such as BP and HR only (Astorino et al. 2013; Notarius, Morris, & Floras, 2006). Beyond BP and HR, other key cardiovascular variables that have a relationship with BP during the postexercise period must be examined, such as PVR, cardiac output (CO) and stroke volume (SV). Furthermore, BP responses should be monitored for several hours following a resistance exercise session.

Some studies have shown that BP decreases after a resistance exercise session (Farinatti, Nakamura, & Polito, 2009; Polito & Farinatti 2009; Rezk, Marrache, Tinucci, Mion, & Forjaz, 2006), which can be associated with a decrease in CO and PVR (Rezk et al., 2006). It is also possible that BP remains depressed for several hours after a resistance exercise session (Anuniação & Polito, 2011). Therefore, the purpose of the current study was to examine the effect of caffeine intake on cardiovascular and hemodynamic variables after a resistance exercise session.

METHODS

Subjects

The sample consisted of 15 healthy, non-smoking volunteers (12 male, three female) who had recreational resistance training experience, were non-hypertensive (SBP and DBP lower than 140/90 mmHg, respectively), had no orthopedic problems, and used no substances or medications with cardiovascular effects. Post-resistance exercise hypotension (Queiroz et al., 2013) and cardiovascular response related to caffeine intake (MacDougall, Musante, Castillo, & Acevedo, 1988) are similar in men and women. All subjects were light caffeine habituated (<250 ml of black coffee by day) (Noordzij et al. 2005). Subjects were instructed not to drink any caffeinated beverages, perform strenuous physical exertion, or consume alcohol for 72 hours before the start of data collection and throughout the experiment. All volunteers signed an informed consent according to the Declaration of Helsinki. Human Ethics Committee of Londrina State University (PR, Brazil) approved the present study.

Maximum Strength Test

One-repetition maximum tests (1RM) were performed 72 hours before the

experiment. Individuals were instructed on the proper execution of each exercise and performed a specific warm-up of 10 repetitions with a subjectively light load. After the warm-up, subjects were tested on the following exercises: lat pull down, knee flexion, chest press, knee extension, biceps curl, leg press 45°, and triceps curl. A maximum of five attempts were allowed to determine the 1RM for each exercise, with 3 to 5 minute rest intervals between attempts and exercises (Fleck & Kraemer, 2004).

Experimental Design

The study was conducted on two non-consecutive days, a minimum of 72 hours apart, between 8:00 and 9:00 in the morning. Upon arrival at the testing site, subjects remained comfortably seated and quiet for 10 minutes. Immediately thereafter, baseline BP was measured using an automatic device (Omron HEM 742 INT). The baseline BP value was the calculated mean of three consecutive measurements. After the baseline BP measurements were complete, subjects ingested either a capsule containing 4 mg caffeine per kg of body weight or a placebo capsule (talc). The dose of 4 mg per kg seems to be sufficient to antagonize A1 and A2 adenosine receptors (Fredholm, 1995) and this dose was reported in a previous study with exercise (Notarius et al., 2006). The order of caffeine or placebo ingestion followed a randomized design, and all subjects completed both conditions. Subjects were told that both capsules contained caffeine.

Forty-five minutes following the ingestion of caffeine or placebo, systolic blood pressure (SBP), diastolic BP (DBP), mean arterial pressure (MAP), HR, stroke volume (SV), cardiac output (CO) and peripheral vascular resistance (PVR) were continuously and non-invasively monitored with beat-to-beat measurements (Finometer PRO, Finapres Medical Systems). Later, subjects underwent a resistance exercise session consisting of three sets of 10 repetitions each (70% 1RM; 90 seconds rest between sets) of the following exercises: lat pull down, knee flexion, chest press, knee extension, biceps curl, leg press 45°, and triceps curl. Subjects performed a 5-minute warm-up before each experimental session. Fifteen minutes after the end of the exercise session, the subjects' SBP, DBP, MAP, HR, SV, CO and PVR were again continuously and non-invasively measured with beat-to-beat measurements. Next, an ambulatory blood pressure monitoring device (SpaceLabs 90207) was placed on the right arm of each subject to monitor their BP at 30-minute intervals for the succeeding 9 hours. The MAP was calculated by the equation: $MAP = (2 \times DBP + SBP) \div 3$.

The procedures for data collection are illustrated in Figure 1.

Statistical Analysis

Mauchly's test was applied to confirm the sphericity of the data. For those cases in which sphericity was not preserved, the authors employed the Greenhouse-Geisser correction. The comparison of resting variables before and after the intake of

caffeine or placebo was performed using a two-way analysis of variance (ANOVA). The same statistical procedures were used for comparison of the caffeine/placebo conditions at the post-exercise time point (i.e. 15 minutes after exercise) as well as the mean values during the 9 hours of ambulatory monitoring. Tukey's post-hoc tests were utilized when necessary. To analyze the period of ambulatory monitoring, multiple comparisons were performed using a repeated measures ANOVA, followed by Bonferroni post-hoc tests, when necessary, to identify the differences. The significance was set at $p < 0.05$. All calculations were performed using SPSS 17.0 software.

RESULTS

The physical and functional characteristics of subjects are shown in Table 1. The results of the ambulatory BP monitoring are shown in Figures 2, 3 and 4. A significant difference ($p < 0.05$) was demonstrated in SBP measures (Figure 2) between the caffeine and placebo conditions at 2:30 hours and 3:00 hours post-exercise. Caffeine ingestion resulted in significantly higher SBP values ($p < 0.05$) than at rest between 30 minutes and 9:00 hours postexercise. Placebo ingestion resulted in inconsistent post-exercise SBP values, with some significantly higher ($p < 0.01$) than at rest (1:30 hours and 6:30 hours) and another significantly lower ($p < 0.05$) than at rest (3:00 hours). DBP values (Figure 3) were significantly different ($p < 0.01$) between the caffeine and placebo conditions at 30 minutes post-exercise as well as between 5:00 hours and 6:30 hours post-exercise. Caffeine ingestion resulted in an increase in DBP values ($p < 0.05$) compared to resting values from 30 minutes to 9:00 hours post-exercise. For the placebo condition, a significant increase ($p < 0.05$) in DBP was demonstrated post-exercise at all measurement time points, except for the measures at 2:00 hours and 4:00 hours post-exercise, which were not significantly different. For MAP (Figure 4), a significant difference ($p < 0.01$) between the caffeine and placebo conditions was shown between 1:30 hours and 3:00 hours post-exercise, as well as at 5:30 hours and 6:00 hours post-exercise. For the caffeine condition, MAP was significantly higher ($p < 0.05$) than at rest in all measures, with the exception of measures at 2:00 hours, 2:30 hours, and 4:00 hours post-exercise, which were not significantly different.

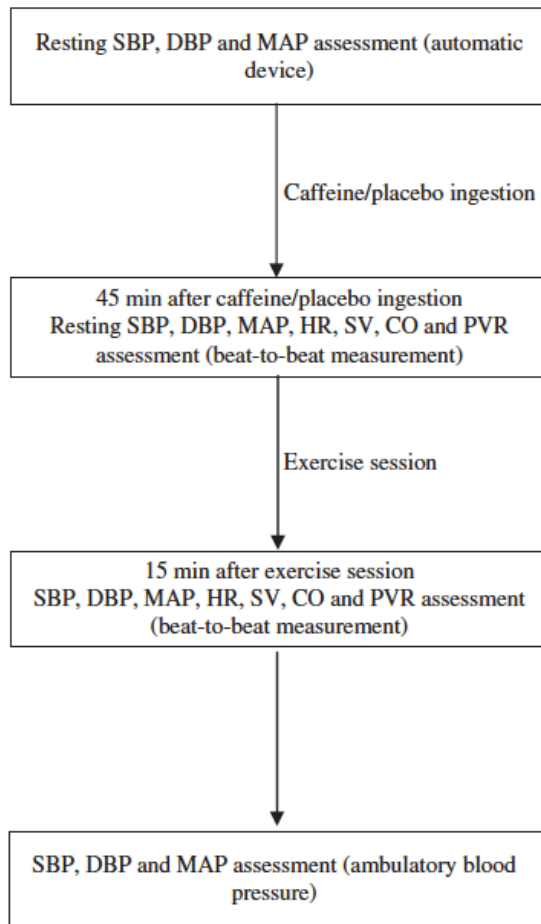


FIGURE 1 Procedures for data collection.

TABLE 1 General Characteristics of the Sample

<i>N</i>	15 (12 M; 3W)
Age (years)	21.3 ± 2.1
Body weight (kg)	70.3 ± 8.7
Height (cm)	172.8 ± 6.9
BMI (kg.m ⁻²)	23.5 ± 2.4
1RM Lat pull down (kg)	73.2 ± 20.3
1RM Knee flexion (kg)	47.2 ± 11.8
1RM Chest press (kg)	66.4 ± 24.5
1RM Knee extension (kg)	51.2 ± 12.6
1RM Biceps curl (kg)	37.8 ± 10.1
1RM Leg press 45° (kg)	226.2 ± 81.4
1RM Triceps curl (kg)	65.6 ± 16.9
Baseline systolic blood pressure (mmHg)*	126.1 ± 5.4
Baseline diastolic blood pressure (mmHg)*	67.2 ± 6.2
Baseline mean arterial pressure (mmHg)*	86.7 ± 3.0

*Values obtained by an automatic device; M = men; W = women; RM = repetition maximum.

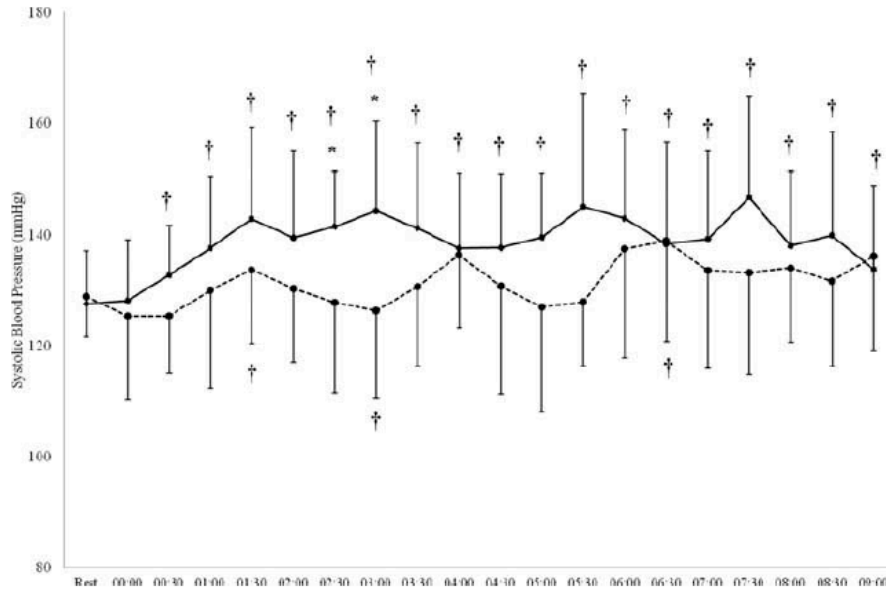


FIGURE 2 Ambulatory monitoring of systolic blood pressure after the exercise session. The resting value was obtained by an automatic device. Continuous line = caffeine condition; dashed line = placebo condition; *significant difference compared to the resting time point; †significant difference between the caffeine and placebo conditions.

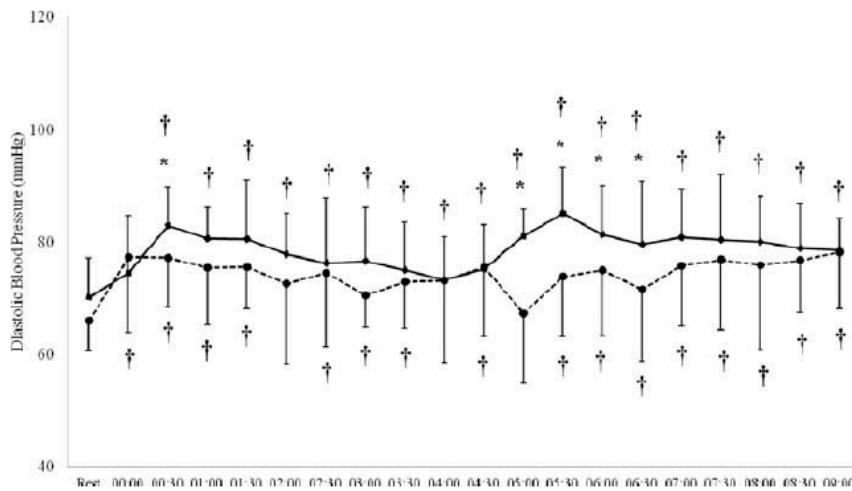


FIGURE 3 Ambulatory monitoring of diastolic blood pressure after the exercise session. The resting value was obtained by an automatic device. Continuous line = caffeine condition; dashed line = placebo condition; *significant difference compared to the resting time point; †significant difference between the caffeine and placebo conditions.

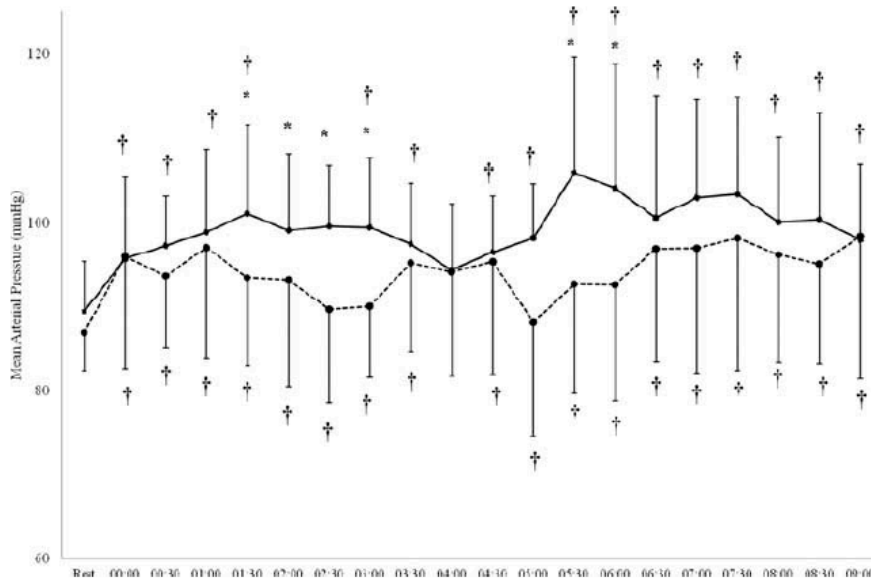


FIGURE 4 Ambulatory monitoring of mean arterial pressure after the exercise session. The resting value was obtained by an automatic device. Continuous line = caffeine condition; dashed line = placebo condition; *significant difference compared to the resting time point; †significant difference between the caffeine and placebo conditions.

Table 2 lists the mean values during the 9 hours post-exercise for the caffeine and placebo conditions. During this time period, significant differences were shown between the caffeine and placebo conditions for SBP ($p = 0.044$); DBP ($p = 0.021$) and MAP ($p = 0.049$). Further comparisons showed a significant increase for the caffeine condition in the resting SBP ($p = 0.004$), DBP ($p = 0.001$) and MAP ($p = 0.001$) and for placebo condition in the DBP ($p = 0.002$) and MAP ($p = 0.005$).

The values for variables measured 45 minutes after ingestion of caffeine or placebo (pre-exercise) and fifteen minutes after the end of the exercise (post-exercise) are shown in Table 3. The comparison between the caffeine and placebo conditions showed a significant difference in DBP ($p = 0.004$) and MAP ($p = 0.006$) for the caffeine condition pre-exercise, as well as a significant increase in PVR ($p = 0.03$) for the caffeine condition post-exercise. The comparison between the pre- and post-exercise values showed a significant decrease in SBP ($p = 0.0007$), MAP ($p = 0.04$) and PVR ($p = 0.01$) and an increase in HR ($p = 0.00001$) and CO ($p = 0.04$) for the placebo condition. For the caffeine condition, a significant decrease was shown in SBP ($p = 0.0003$) and MAP ($p = 0.004$) and an increase in HR ($p = 0.00007$).

TABLE 2 Values of Blood Pressure at Rest and in the Mean of 9 h after the End of Resistance Training

	Resting Placebo	Resting Caffeine	Placebo 9 h	Caffeine 9 h
Systolic blood pressure (mmHg)	129.2 ± 7.6	128.0 ± 9.4	131.3 ± 4.3	139.1 ± 4.9*†
Diastolic blood pressure (mmHg)	66.3 ± 5.5	70.2 ± 7.8	75.1 ± 3.4*	79.5 ± 3.8*†
Mean arterial pressure (mmHg)	87.4 ± 5.7	89.1 ± 6.3	94.2 ± 3.6*	100.6 ± 3.4*†

*Significantly different from resting value within the same condition; †significant difference between conditions.

TABLE 3 Hemodynamic Responses after the Intake of Caffeine/Placebo and after the End of Resistance Training (Obtained by Continuous Beat-to-Beat Measures)

	Pre-exercise (placebo)	Pre-exercise (caffeine)	Post-exercise (placebo)	Post-exercise (caffeine)
Systolic blood pressure (mmHg)	123.4 ± 8.2	127.3 ± 7.6	113.2 ± 7.1*	112.8 ± 12.2*
Diastolic blood pressure (mmHg)	72.1 ± 4.3	79.0 ± 7.1†	71.5 ± 6.7	74.9 ± 7.5
Mean arterial pressure (mmHg)	90.2 ± 4.7	97.4 ± 8.9†	86.5 ± 6.2*	88.3 ± 8.4*
Heart rate (bpm)	74.3 ± 7.5	73.2 ± 12.0	97.6 ± 8.8*	96.2 ± 14.5
Stroke volume (ml)	83.4 ± 18.6	77.3 ± 15.7	72.4 ± 12.6	64.3 ± 18.9
Cardiac output (L.min ⁻¹)	6.1 ± 1.2	5.5 ± 1.2	6.9 ± 1.1*	6.1 ± 1.4
Peripheral vascular resistance (mmHg.min.L ⁻¹)	15.6 ± 4	18.5 ± 5.1	13.2 ± 2.2*	15.7 ± 4.2 †*

*Significantly different from resting value within the same condition; †significant difference between conditions.

DISCUSSION

This study analyzed the effect of caffeine ingestion on numerous hemodynamic variables before, during and for 9 hours after resistance exercise. A key finding was that the ingestion of caffeine resulted in significantly greater preexercise DBP and MAP versus the placebo. Another key finding was that the ingestion of caffeine resulted in a significantly greater post-exercise PVR versus the placebo. Furthermore, the 9 hour post-exercise mean values for SBP, DBP and MAP were significantly greater for the caffeine versus placebo condition.

Contrary to our findings, Astorino et al. (2013) showed an increase only in the resting SBP 60 min after the ingestion of caffeine (6 mg.kg⁻¹). Blood pressure regulation depends on the response of different variables, including CO and PVR, which were not assessed in the Astorino et al. (2013) study. At 15 minutes after exercise, however, we noted a decrease in SBP, MAP and PVR in both the caffeine and placebo groups.

The reduction in PVR after the exercise for the caffeine versus placebo condition may indicate that the hypotensive effect of exercise may still exert a relative blockade of adenosine receptors. The reduction in SBP and MAP may be explained by the reduction in PVR. The reduction in PVR may have been triggered by reduced sympathetic stimuli via the central nervous system soon after the end of exercise (Chen & Bonham, 2010). In terms of hemodynamic compensation, we found a significant increase in HR and, consequently, in CO in the placebo condition. In the caffeine condition, no change was observed in HR or CO after exercise, possibly because the PVR was diminished less than in the placebo condition. It may be that the increase in HR for the placebo condition could be explained by a reduction (even non-significant) in the SV, thus suggesting a decreased preload due to the drop in plasma volume, which usually occurs after resistance exercise (Collins, Cureton, Hill, & Ray, 1989). Nevertheless, the reduction in plasma volume should trigger an increase in PVR. Thus, even assuming a possible reduction in plasma volume, we did not find that it was sufficient to increase the PVR.

Few studies have examined the effect of caffeine on cardiovascular performance post-exercise, and the available evidence is not completely convergent. For instance, regarding aerobic exercise, Notarius et al. (2006) found an increase in SBP and MAP 15 min after exercise for caffeine (intravenous infusion – 4 mg.kg⁻¹) versus a placebo condition. However, Costa, Anunciação, Ruiz, Casonatto, and Polito (2012) observed no changes in BP 60 minutes post-exercise for both a caffeine (oral administration – 4 mg.kg⁻¹) and placebo condition. The recent study of Astorino et al. (2013) identified no reduction in BP during the 75 minutes after a resistance exercise session, with or without caffeine administration (oral administration – 6 mg.kg⁻¹). The possible explanations for these divergent results may be associated with the different exercise modes, monitoring periods and methods of caffeine administration. It is also possible that post-exercise cardiovascular performance may be linked to different physiological pathways (Hamer, 2006).

To add to the previously observed hypotensive effect 15 minutes after exercise, the model used in the present study to include monitoring for several hours post-exercise expands on the body of literature in understanding the effects of caffeine and exercise on the cardiovascular system. In fact, by monitoring for 9 hours, we observed an inverse effect from that observed 15 minutes after exercise. In other words, a significant increase in SBP, DBP and MAP was observed for the caffeine condition both in relation to the resting and placebo conditions. For the placebo condition, a significant increase in DBP and MAP was observed in relation to the resting condition. The literature previously showed no increase in BP several hours after resistance exercise (Anunciação & Polito, 2011), contradicting our results for the placebo condition. A placebo effect may have been possible; as our subjects were all told they were consuming caffeine. However, the increases identified for the caffeine condition illustrate the effect of this substance on the cardiovascular system, given the significant differences in relation to the placebo condition. Our study confirmed that the hypotensive effect previously observed at 15 min after exertion was temporary and BP actually increased during the 9-hour post-exercise

monitoring. Therefore, we believe that different physiological pathways operate during the post-exercise period. One hypothesis is that the blockade of adenosine receptors continues to occur for some hours after caffeine intake, resulting in a late increase of BP post-exercise. However, it is not possible to establish the relationship of caffeine consumption to some physiological mechanisms that determine the hypotensive effects of exercise, such as the adjustment via the solitary tract nucleus (Chen & Bonham, 2010) and the hypothesis of histamine receptors (Halliwill, Buck, Lacewell, & Romero, 2013).

One limitation of the present study is the lack of a control group that did not perform exercise with or without caffeine/placebo intake. Consequently, we have no information about cardiovascular responses in the absence of exercise and/or in the absence of caffeine intake. However, as the increased mean SBP, DBP and MAP values for the caffeine condition persisted during 9 hours of monitoring compared WITH the placebo condition, our results suggest that caffeine does affect cardiovascular responses post-exercise.

CONCLUSION

The intake of caffeine increased the DBP and MAP at rest, but a reduction in the SBP, MAP and PVR was observed soon after the completion of resistance exercise. We also observed that mean SBP, DBP and MAP values were higher during 9 hours after the exercise with caffeine. These results suggest that the cardiovascular effects of caffeine are different over the post-exercise period after resistance exercise in normotensive young adults. To more completely understand the implications of these findings, further investigations need to be conducted with different study populations and other exercise models.

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