Effect of caffeine on metabolic and cardiovascular responses to submaximal exercise in lean and obese men

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Summary

Study aim: To compare the effects of caffeine on metabolic and cardiovascular responses to exercise in lean and obese subjects.

Material and methods: In a double blind random design, 6 lean (BMI<20) and 6 obese (BMI>28) sedentary young men performed treadmill running for 30 min at similar exercise intensities (60% VO2max) one hour after caffeine (5 mg/kg body mass) or placebo ingestion. Gas exchange was measured by indirect calorimetry/open-circuit spirometry. Heart rate (HR) was measured throughout the exercise. Blood pressure (BP) measurements were taken at baseline, 1 h after caffeine/placebo ingestion and immediately after exercise. Repeated measures ANOVA was used in data analysis.

Results: Caffeine significantly (p<0.05 – 0.01) increased the exercise-induced oxygen uptake, energy expenditure, systolic blood pressure and heart rate in both groups, the respiratory exchange ratio remaining unchanged. The effects of caffeine were in both groups alike.

Conclusions: Caffeine activates metabolism without inducing major changes in fat/carbohydrate oxidation. The differences in body fat content seem not to affect the caffeine-induced effects in a submaximal exercise.

Keywords: Caffeine – Body composition – Responses to exercise

Introduction

Light physical activity enhances energy expenditure and fat oxidation in skeletal muscles [14]. Under resting conditions, 70% of free fatty acids generated by lipolysis are utilised in triglyceride resynthesis. During exercises even of low intensity, the free fatty acid uptake from plasma by muscles increases five-fold [23,29,36] and that process is greatly influenced by their arterial concentration [2,18]. Also other factors are known to improve fatty acid utilisation; among them are exercise, ingestion of medium-chain triglycerides, heparin injections, supplements containing L-carnitine and caffeine, etc. [5].

It was demonstrated that caffeine enhances utilisation of fats as an energy source during aerobic activities [7]. It was also reported that metabolic effects of caffeine under resting conditions consist of a temporary increase of calorie consumption [19,22,29]. Although some authors claimed that caffeine might significantly augment fat oxidation and energy consumption during prolonged activities at low or moderate intensities (below 60% VO2max) [6,11,13], other studies did not confirm those findings [10,35]. The reasons of those inconsistencies are not clear. Caffeine may exert various cardio-vascular effects and the consequences are often controversial [8,12,25].

There are reports that caffeine-induced thermogenesis, fat oxidation, and lipolysis may enhance weight reduction to a greater extent in non-obese than in obese subjects. That thermogenic effect was confirmed in some [1,4] studies but controversies still exist [20]. Similarly, Acheson et al. [1] and Daubresse et al. [9] found that caffeine induced a more increased lipolysis in non-obese than in obese subjects, and Acheson et al. [1] found the same pattern for fat oxidation. In contrast, Bracco et al. [4] did not confirm a greater expression of caffeine-induced fat oxidation in non-obese vs. obese females. However, all those studies were conducted under resting conditions. The question remains, whether similar responses would be observed under exercise loads, considering that skeletal muscles of obese individuals contain greater amounts of triglycerides than the lean ones [15,27]. Thus, the aim of this study was to identify the caffeine-induced metabolic and cardio-vascular responses in lean and obese men performing a sub-maximal exercise.

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Material and Methods

Subjects: 6 lean (BMI<20) and 6 moderately obese (BMI>28) men volunteered to participate in the study. All subjects practiced leisure physical activities but not competitive sports. They were medically screened and completed a detailed questionnaire regarding their habitual caffeine consumption. All subjects were non-smokers, healthy at the time of the study without any history of cardiovascular or lung diseases. Individuals who declared high caffeine intake (>300 mg/day) were not recruited. All subjects submitted written informed consents to participate; the study was approved by the local Committee of Ethics.

Experimental protocol: The initial, pre-experimental measurements were conducted at least a week prior to the study and included body height and mass, body fat content, respiratory exchange ratio (RER) and VO₂max. All measurements were done in the preprandial state. Body fat percentage was assessed by bioimpedance technique (Inbody 3.0®, Biospace Co. Ltd, Seoul, Korea); VO₂max was determined in an incremental treadmill running test till exhaustion preceded by a 5-min warm-up, initial speed amounting to 3 km/h and was increased by 1 km/h every minute. The exercise test was terminated at volitional exhaustion or when RER reached 1.15. All subjects were familiarised with the experimental protocol and equipment.

The randomised cross-over design was conducted in double-blind mode. Exercise sessions were conducted in the mornings twice, 5 – 7 days apart, following an at least 12-h overnight fasting and caffeine abstinence. Subjects were instructed not to engage in any strenuous exercise on the preceding day. Upon arrival, the subjects were given gelatine capsules containing caffeine (5 mg/kg body mass) or empty (placebo). The capsules were placed in labelled envelopes (subjects’ initials and “Day 1” or “Day 2”) by a technician not involved in any way in the study. The subjects remained seated in the laboratory for 60 min on the preceding day. Upon arrival, the subjects were given placebo and were instructed not to engage in any strenuous exercise before and 1 h after caffeine/placebo ingestion, and immediately post-exercise.

Statistical analysis: Factorial ANOVA design with repeated measures was used. The factors were treatment (caffeine/placebo), body build (lean/obese) and sampling times. The level of p≤0.05 was considered significant.

Results

Values characterising lean and obese subjects are presented in Table 1. The results of exercise-related measurements of oxygen uptake, respiratory exchange ratio and arterial blood pressure are shown in Table 2 and in Figs. 1 and 2.

Table 1. Mean values (±SD) of somatic variables and VO₂max recorded in lean and obese subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Lean n = 6</th>
<th>Obese n = 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>21.8 ± 1.3</td>
<td>22.5 ± 0.8</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>56.1 ± 5.6</td>
<td>89.3 ± 8.9*</td>
</tr>
<tr>
<td>Body height (cm)</td>
<td>175.2 ± 4.8</td>
<td>174.0 ± 9.1</td>
</tr>
<tr>
<td>BMI</td>
<td>18.3 ± 1.4</td>
<td>29.6 ± 1.2*</td>
</tr>
<tr>
<td>Body fat content (%)</td>
<td>11.4 ± 3.9</td>
<td>26.4 ± 3.6*</td>
</tr>
<tr>
<td>VO₂max (ml/kg/min)</td>
<td>43.6 ± 8.1</td>
<td>38.8 ± 2.2</td>
</tr>
</tbody>
</table>

* Significantly (p<0.05) different from the respective value in lean subjects

Table 2. Mean values (±SD) of oxygen uptake, respiratory exchange ratio and arterial blood pressure

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gr. Tr.</th>
<th>Lean n = 6</th>
<th>Placebo</th>
<th>Caffeine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen uptake (ml/kg/min)</td>
<td>26.1 ± 3.0</td>
<td>28.0 ± 2.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RER</td>
<td>0.87 ± 0.05</td>
<td>0.91 ± 0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EE (kcal/min)</td>
<td>7.14 ± 2.77</td>
<td>7.72 ± 2.94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>100.6 ± 7.6</td>
<td>107.2 ± 11.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP – DBP (mm Hg) @</td>
<td>33.2 ± 1.5</td>
<td>36.9 ± 5.0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Gr. Tr.</th>
<th>Obese n = 6</th>
<th>Placebo</th>
<th>Caffeine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen uptake (ml/kg/min)</td>
<td>23.0 ± 2.7</td>
<td>24.8 ± 1.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RER</td>
<td>0.90 ± 0.03</td>
<td>0.88 ± 0.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EE (kcal/min)</td>
<td>10.16 ± 3.85</td>
<td>10.89 ± 2.72</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>116.9 ± 9.3</td>
<td>119.7 ± 7.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP – DBP (mm Hg) @</td>
<td>40.0 ± 2.4</td>
<td>37.6 ± 3.4</td>
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</tr>
</tbody>
</table>

Significance: * p<0.05; ** p<0.01; *** p<0.001; NA – Not applicable

Physiological measurements: Oxygen uptake (VO₂) was recorded continuously by indirect calorimetry/open-circuit spirometry using a gas analyser (Casmed Italy MED 150) after correcting for temperature, pressure and humidity. Energy expenditure rate was determined from respiratory gas exchange data by using the standard conversion for non-protein RER for steady-state exercise [24]. Gas analysers were carefully calibrated prior to each experimental session for each subject using medical gas mixture of known composition. Heart rate was recorded throughout the exercise using a telemetric device with monitor (Polar, Kempele, Finland). Systolic (SBP) and diastolic (DBP) blood pressures were measured sphygmomanometrically above the left brachial artery [8] before and 1 h after caffeine/placebo ingestion, and immediately post-exercise.
The exercise-induced oxygen uptake was significantly (p<0.05) increased by caffeine (by over 7%) in both lean and obese subjects but in the latter ones was by nearly 12% lower than in the lean subjects. No significant changes were found in case of RER while energy expenditure was significantly (p<0.001) higher (by about 42%) in obese than in the lean ones subjects and higher (p<0.05) following caffeine ingestion (by about 8%; see Table 2).

**Fig. 1.** Mean values (±SE) of systolic blood pressure (left) and of the systolic-diastolic difference (right) in lean (n = 6) and obese (n = 6) men before caffeine or placebo ingestion (0), 1 h following ingestion (1 h) and immediately post-exercise (P)

* Significant (p<0.05) difference between lean and obese subjects by t-test; for detailed significances see Table 2

Blood pressure was expressed by two measures: systolic pressure (SBP) and the systolic-diastolic difference (SBP – DBP). As to SBP, all three ANOVA factors were significant – lean vs. obese (p<0.01), caffeine vs. placebo (p<0.01) and the time points of measurements (p<0.001; see Fig. 1). When the measurements at 3 time points were averaged, the effects of group of subjects and of caffeine remained significant (p<0.01; see Table 2).

**Fig. 2.** Mean values (±SE) of heart rate recorded every 5 min throughout the treadmill exercise in lean (n = 6) and obese (n = 6) men following caffeine or placebo ingestion

Legend: O – Obese; L – Lean; C – Caffeine; P – Placebo; * Significant (p<0.05) overall difference between caffeine/placebo trials; *** Significant (p<0.001) overall difference between lean and obese subjects

Heart rate was, on average, significantly (p<0.05) higher in caffeine than in placebo trials and much higher (p<0.001) in obese than in lean subjects throughout the exercise (Fig. 2). However, the caffeine-induced effect expressed as average HR during the exercise related to the respective placebo value, was lower than in case of oxygen uptake and amounted to 3.4 and 4.6% in lean and obese subjects, respectively.

### Discussion

The results of this study demonstrated that lean and obese subjects similarly responded to caffeine in a sub-maximal treadmill test at 60% VO2max. The lack of differences in RER is in agreement with other studies in which similar dosage of caffeine was used [10,12,35] and that energy expenditure was stimulated by increasing both lipid and carbohydrate oxidation in both lean and obese men to a comparable degree. This might mean that the effects of caffeine were due to either increasing plasma FFA in lean and obese subjects alike or to enhance FFA oxidation to a similar degree despite differences between lean and obese subjects in the amounts of FFA in plasma. Schiffelers et al. [31] showed that heparin infusions increased plasma FFA concentrations and RER was decreased in lean and obese men identically. This result indicated that increases in plasma FFA have same effect on FFA oxidation in lean and obese men. Further studies would be needed to clarify this question.

Bracco et al. [4] showed that coffee had a higher thermogenic effect in lean than in obese women under resting conditions, the energy expenditure and RER
during a 30-min treadmill exercise at 50% VO₂max being alike. Inasmuch our results are not directly comparable to Bracco’s et al. study due to differences in protocols (gender, form of administering caffeine, exercise intensity), both studies demonstrated lack of differences in some caffeine effects between lean and obese groups.

Our results suggest that fat oxidation, reflected by RER data, does not differ between lean and obese subjects both in caffeine and placebo groups. This finding is in contrast with Goodpaster et al. [16] who showed that fat oxidation was more stimulated in obese than in lean subjects during submaximal exercise. That contrast may have been due to higher intensity (60 vs. 50% VO₂max) but shorter duration (30 vs. 60 min) of exercise used in our study.

Caffeine was shown to decrease heart rate in low-intensity exercises or at rest [10,25,35] but in heavy, submaximal exercises HR would remain unchanged [8,12,25,37]; it seems likely that caffeine increases the stroke volume only at a low work intensity. Heart rate in our study was significantly higher in obese than in lean subjects and the same was found by Bracco et al. [4] in women performing treadmill exercise (30 min at 50% VO₂max).

This may suggest that heart rate may serve as a useful indicator of exercise intensity when differentiating lean and obese subjects. However, more studies are needed to support this suggestion.

Caffeine appears to affect blood pressure via adenosine receptor inhibition and increased release of some neurotransmitters [26]. Like in other studies [8,12,17,21,32-34], caffeine significantly increased the systolic blood pressure especially in obese subjects, who are known to be at higher risk of hypertension than the lean ones [29]. Blood pressure may also increase due to physical exertion [4,34]. As follows from our results, the combined rise in blood pressure induced by exercise and caffeine did not significantly increase the SBP compared with the post-exercise value in obese men receiving placebo.

In conclusion, caffeine seemed to activate metabolism without inducing major changes in fat/carbohydrate oxidation. The differences in body fat content seem not to affect the caffeine-induced effects in a submaximal exercise.

References


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