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Research article

Effects of caffeine on exercise performance in sedentary females

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Abstract
The purpose of the study was to examine the effect of caffeine ingestion on total work, average power, oxygen consumption (VO_2), respiratory exchange ratio (RER), ratings of perceived exertion (RPE), heart rate (HR) and energy expenditure (kJ) during stationary cycling at a standardised power output, as well as during a set time period where participants were required to cycle as fast as they could. Ten healthy, sedentary, female, non-regular caffeine users completed 15 min of stationary cycling at a standardised power output equating to 65% HR_{max} (Phase A), followed by 10 min of stationary cycling where they were required to cycled as fast as they could (Phase B) after ingesting 6.0 $mg \cdot kg^{-1}$ of caffeine or placebo 60 min prior to exercise. VO_2 and energy expenditure were significantly higher at the end of Phase A (p = 0.008 and p = 0.011, respectively). All other variables examined in Phase A were similar between trials. In Phase B, there were no significant differences found for any variable assessed. While caffeine ingestion resulted in significant increases in VO_2 and energy expenditure during steady-state exercise, it did not improve cycling performance during a 10 min trial where participants were required to cycle as fast as they could.

Key words: Sub-maximal exercise, rating of perceived exertion, energy expenditure, weight maintenance.

Introduction

The common belief that caffeine improves both mental and physical performance, combined with the removal of caffeine from the banned substances list on the 1st of January 2004 by the World Anti-Doping Agency (WADA, 2008) has led to its widespread use amongst athletes competing in many sports. To date, many research trials have demonstrated caffeine to be an ergogenic aid for exercise of varying intensities, durations and modalities in an athletic population (Bell and McLellan, 2002; Bridge and Jones, 2006; Bruce et al., 2000; Doherty and Smith, 2005; Graham, 2001; Plaskett and Cafarelli, 2001; Schneiker et al., 2006; Stuart et al., 2005). Benefits associated with caffeine ingestion in this population include delayed feelings of fatigue (Anselme et al., 1992; Jackman et al., 1996), reduced sensations of pain and exertion (Bell and McLellan, 2002, increased time to exhaustion (Graham and Spriet, 1991), increased fatty acid oxidation (Chad and Quigley, 1989), increased mean power output (Anderson et al., 2000), decreased times to complete a set amount of work (Bridege and Jones, 2006), stimulation of motor activity (Fisone et al., 2004), as well as an increase in alertness, feelings of subjective energy and ability to concentrate (Keisler and Armsey, 2006; Lorist et al., 1994). While the precise reasons underlying the ergogenic effects of caffeine remain equivocal, it is generally accepted that the most likely mechanism is adenosine receptor antagonism (Graham, 2001). When the body is under increased metabolic demand, circulating adenosine is increased in order to decrease activity. By attaching to adenosine receptors, caffeine is able to counteract many of the inhibitory effects of endogenous adenosine on neuro-excitability (Kalmar and Cafarelli, 2004), neurotransmitter release (Fredholm et al., 1999) and arousal (Porkka-Heiskanen, 1999).

The effects of caffeine on physical performance (walking, cycling and aerobic-dance bench stepping) have also been assessed to a lesser degree in non-athletic but recreationally active populations (Ahrens et al., 2007a; 2007b; Engles et al., 1999). These studies reported that caffeine significantly increased the rate of energy expenditure (REE), oxygen uptake (VO_2: Ahrens et al., 2007a; Engels et al., 1999), and the percentage of maximal VO_2 uptake reserve (Ahrens et al., 2007a), but did not alter sense of effort (Ahrens et al., 2007a; 2007b), heart rate (HR; Ahrens et al., 2007a; 2007b) or respiratory exchange ratio (RER: Ahrens et al., 2007a; 2007b; Engels et al., 1999). Of relevance, various limitations were associated with these studies that may have affected results. For example, there was no fasting period prior to the exercise protocols (Ahrens et al., 2007a; 2007b), cohorts consisted of mixed genders (Engels et al., 1999) and menstrual cycle phases were not controlled for (Ahrens et al., 2007a; 2007b; Engels et al., 1999), with the luteal phase being associated with greater pain sensitivity (Fillingrim and Ness, 2000). Furthermore, none of these studies assessed the effects of caffeine on the ability to perform more work.

Of importance, the ability to perform more work after caffeine ingestion, as demonstrated in athletic populations, can result in greater caloric expenditure and the possibility of improved fitness if the exercise is maintained over an extended period of time. These benefits, combined with the proposed increase in fatty acid oxidation associated with caffeine ingestion (Chad and Quigley, 1989), are pertinent to populations characterised by sedentary lifestyles that in turn can result in weight gain and consequent health issues. Of relevance, there has been only one published study to date known to the authors that has assessed the effects of caffeine on exercise in a sedentary population (Engels and Haymes, 1992). These researchers reported significantly higher minute ventilation, as well as an increase in pre and post exercise free fatty acids associated with a 60 min walking protocol in

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sedentary males following caffeine ingestion. However, the ability to perform more work during this time period was not assessed. Consequently, further well-controlled studies are needed to assess the effects of caffeine in a sedentary population, with particular focus on caffeine's ability to increase fatty acid oxidation, lower effort sensation and improve work performance.

Therefore, the first aim of this study was to examine the effect of caffeine ingestion on VO_2, RER, RPE and HR during 15 min of stationary cycling at a standardised power output in a sedentary, female population. Secondly, the effect of caffeine ingestion on 10 min of self-paced stationary cycling, where participants were required to cycle as fast as they could during this time period, was also assessed. Based on the reported ergogenic benefits of caffeine on exercise, it was hypothesised that compared to a placebo, caffeine ingestion would result in lower RPE values and greater fatty acid oxidation as indicated by a decrease in RER values during stationary cycling at a set power output. It was also hypothesised that participants would cycle at a significantly higher intensity overall and complete more total work during the 10 min stationary cycling trial after caffeine ingestion.

Methods

Ten healthy, sedentary, females who were non-regular caffeine users (<100 mg per day) volunteered for this study (mean ± SD: age 22 ± 2 yr; height 1.65 ± 0.11 m; body-mass 61.9 ± 14.8 kg; BMI 22.7 ± 3.0). The participants were initially screened using the PAR-Q, as well as a detailed questionnaire that assessed caffeine consumption habits. Individuals who participated in more than 20 min of exercise performed on three or more days per week were considered ineligible for the study. Prior to testing, participants gave informed consent and the study was approved by the human ethics committee of the University of Western Australia (UWA).

Each participant reported to the Exercise Physiology Laboratory at UWA on three separate occasions: once for a familiarisation session, followed by two experimental sessions involving stationary cycling following the administration of either caffeine or placebo in a double-blind, randomised, crossover design. The participants were instructed to refrain from consuming caffeine for 48 h prior to each testing session, as well as to avoid exercise for 24 h prior. The same instructions applied to all visits. A list of food and beverages containing caffeine was provided to each participant in order to inform them of what products they should avoid. Participants were also asked to keep a diary documenting their diet 24 h prior to the first testing session and to replicate this diet before each subsequent visit.

The familiarisation session required participants to complete a graded exercise protocol on a bicycle ergometer (Exertech EX-10 front access cycle ergometer, Repco Cycle Company, Huntingdale, Victoria) in order to determine the power output required to elicit a target HR equating to 65% of individual age-predicted maximum HR (220 – age). Percentage HR_{max} was chosen over $\%VO_{2max}$ in an effort to make the study less onerous for the sedentary participants. This procedure involved a starting workload of 25 Watts (W) that increased by 25 W increments every three minutes, until the target HR was reached. Once the target HR was attained, the participants continued to cycle at the power output that equated to this HR value for a duration of 15 min. During this time, the participants breathed through a mouthpiece attached to a Hans-Rudolf valve connected to a computerised gas analysis system comprising of a Morgan ventilation monitor (Morgan, Reinham, Kent, UK), an oxygen analyser (Ametek SOV S-3A11, Applied Electrochemistry, Ametek, Pittsburgh, PA) and a carbon dioxide analyser (Ametek COV CD-3A, Applied Electrochemistry, Ametek, Pittsburgh, PA). Both the Morgan ventilometer and gas analysers were calibrated before testing using a 1 L syringe and a certified gravimetric β standard gas mixture (BOC Gases, Chatswood, Australia) of known composition, respectively. After this initial 15 min of cycling, the participants were allowed 5 min of passive rest before commencing another 10 min bout of cycling where they were required to cycle as fast as they could within this time frame. During this second phase of the exercise, participants were constantly motivated by the researcher and regularly (every minute) advised of the time remaining. Of importance, the time periods of 15 and 10 min were selected as a result of pilot testing that indicated that sedentary participants had difficulty in coping with longer exercise periods that were comprised of the two phases of cycling used in this protocol. The 10 min time period selected for the second phase of the protocol is similar to that used in other studies that assessed caffeine ingestion in recreationally active adults and used set time trials of only 8 mins (Ahrens et al., 2007a; 2007b).

The experimental trials were then conducted between days 3 and 11 of each participant's menstrual cycle (follicular phase) with approximately 4 weeks between trials. Prior to each testing session, participants were given a capsule containing either caffeine (6.0 $mg \cdot kg^{-1}$ body mass; Nō-Dōz®, Key Pharmaceuticals Pty Ltd, Rhodes, NSW, Australia) or placebo (6.0 $mg \cdot kg^{-1}$ body mass; Sucaryl sweetener) and were instructed to ingest the given capsule at 6.30 am on the day of testing whilst at home. The dose of 6.0 $mg \cdot kg^{-1}$ was selected based on previous studies that used this dose and reported performance benefits (Carr et al., 2008; Schneiker et al., 2006; Stuart et al., 2005). The participants were then required to arrive at the Exercise Physiology Laboratory at UWA by 7.15 am in a fasted state, having not ingested food or beverages besides water for the previous 12 h period. At 7.30 am, baseline HR was recorded using a Polar HR monitor (Polar Electro Oy, Kempele, Finland) and exercise testing began. The exercise testing was divided into two phases. The first phase (Phase A) involved 15 min of stationary cycling performed at a constant power output that equated to 65% of the individual's age predicted HR_{max}, as determined during the familiarisation session. During this period, the participants breathed through a mouthpiece connected to the previously described computerised gas analysis system for the analysis of VO_2, RER and energy expenditure (kJ and $kJ.min^{-1}$). In addition, HR and RPE (6 – 20 scale: Borg, 1982) were recorded at 5 min intervals throughout the exercise

Table 1. Effect of caffeine or placebo on the total amount of work done, mean power and energy expenditure during 15 min of steady state cycling (Phase A). Values are means (±SD, n = 10).

	Time	Placebo	Caffeine	Mean change (%) ± 90% confidence limits	P
Work done ($J \cdot kg^{-1}$)	5 min	281 (86)	271 (66)	-.11 (.19)	
	10 min	279 (74)	273 (64)	-.07 (.11)	
	15 min	277 (73)	280 (81)	.03 (.09)	
	Total	837 (232)	825 (210)	.05 (.08)	.39
Mean power (Watts)	5 min	55 (15)	54 (15)	-.06 (.20)	
	10 min	56 (15)	56 (14)	-.03 (.09)	
	15 min	56 (16)	56 (15)	-.01 (.10)	
	Overall	57 (16)	55 (14)	-.12 (.14)	.80
Energy ($kJ \cdot min^{-1}$)	5 min	20 (5)	21 (4)	.20 (.20)	
	10 min	21 (5)	21 (4)	.08 (.19)	
	15 min	20 (5)	22 (5)	.48 (.25)	
Energy (kJ)	Total	304 (296)	322 (315) *	.25 (.14)	.07

P indicates interaction effect of time and trial. * indicates a significant difference (p = 0.01) in total energy (kJ) values between trials based on results from a one-way ANOVA analysis.

protocol. Upon the completion of Phase A, participants were required to rest passively for 5 min and then to cycle for a further 10 min as fast as they could (Phase B, see details described earleir). During this phase, the average power output and total work completed were recorded using the software Cyclemax (School of Sports Science, Exercise and Health, UWA). Of importance, the participants were not provided with any visual or verbal feedback regarding their work intensity. Heart rate and RPE, were recorded every 5 min while VO_2, RER and energy expenditure were calculated at 30 s intervals by software associated with the metabolic cart (META2000, UWA, Perth Australia). A slow, active warm down was then conducted prior to participants leaving the laboratory.

Data analysis

Average results for total work done, overall mean power output and total energy expenditure recorded between trials during both Phase A and Phase B of the experimental trials were analysed separately using one-way ANOVAs. Two-way repeated measures ANOVA's were also used in order to assess the results for individual 5 min blocks of the above measures, as well as HR, RPE, VO_2 and RER during both phases of the protocol (time and trial). Statistical significance was set at $p \leq 0.05$ and Bonferroni post hoc comparisons were made where appropriate. All data was was analysed using SPSS (Version 13.0 for Windows; SPSS Inc, Chicago, IL).

Results

Phase A

The effect of caffeine on overall mean power output and the total amount of work performed during Phase A is summarised in Table 1. There were no significant differences between these two variables for the two trials (p = 0.26 for total work done and p = 0.14 for overall mean power output; Table 1). Further, when the total 15 min period of Phase A was divided into 5 min blocks, there was no significant interaction effect of time and trial for either of these variables (Table 1). Caffeine ingestion did however have a significant effect on total energy expenditure during Phase A (p = 0.01; Table 1). Further, when Phase A was divided into 5 min blocks, the interaction of time and trial on energy expenditure approached significance (p = 0.07; Table 1). Post-hoc analysis revealed that there was a significant increase in energy expenditure at the 15 min mark of the exercise trial after caffeine ingestion compared to the placebo trial (p = 0.007).

While there were no significant interaction effects of time and trial for HR, RPE or RER, changes in VO_2 approached significance (p = 0.08; Table 2). Post-hoc analysis revealed that VO_2 was significantly higher at the 15 min mark of the caffeine trial compared to the placebo trial (p = 0.008; Table 2).

Table 2. Effect of caffeine or placebo on heart rate (HR), rating of perceived exertion (RPE), oxygen uptake (VO_2) and respiratory exchange ratio (RER) during 15 min of steady state cycling (Phase A). Values are means (±SD, n=10).

	Time	Placebo	Caffeine	Mean change (%) ± 90% confidence limits	P
HR	5 min	130 (12)	129 (10)	-.07 (.57)	
	10 min	134 (10)	136 (13)	-.37 (.68)	
	15 min	138 (12)	140 (12	.23 (.61)	.81
RPE	5 min	11 (2)	10 (2)	-.04 (.56)	
	10 min	11 (2)	11 (2)	-	
	15 min	11 (2)	11 (2)	-	.74
VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	5 min	16.05 (3.92)	16.93 (4.03)	.22 (.24)	
	10 min	16.79 (3.91)	17.27 (3.33)	.12 (.19)	
	15 min	15.85 (3.64)	17.78 (3.93)	.53 (.28)	.08
RER	5 min	.95 (.08)	.94 (.08)	-.21 (.52)	
	10 min	.89 (.05)	.91 (.07)	.43 (1.18)	
	15 min	.90 (.04)	.88 (.08)	-.35 (.73)	.31

P indicates interaction effect of time and trial

Table 3. Effect of caffeine or placebo on the total amount of work, mean power and energy expenditure during 10 min of self-selected intensity cycling (Phase B). Values are means (±SD, n=10).

	Time	Placebo	Caffeine	Mean change (%) ± 90% confidence limits	P
Work done ($J \cdot kg^{-1}$)	5 min	423 (85)	470 (95)	.55 (.52)	
	10 min	410 (74)	406 (49)	-.05 (.67)	
	Total	833 (142)	876 (109)	-.06 (.38)	.19
Mean power (Watts)	5 min	86 (29)	92 (29)	.19 (.36)	
	10 min	84 (23)	90 (28)	.28 (.45)	
	Overall	85 (25)	90 (26)	-.14 (.60)	.92
Energy ($kJ \cdot min^{-1}$)	5 min	28 (7)	31 (8)	.34 (.40)	
	10 min	30 (8)	32 (8)	.33 (.47)	
Energy (kJ)	Total	289 (280)	314 (302)	.17 (.50)	.93

P indicates interaction effect of time and trial

Phase B

No significant differences were found for the total amount of work performed, overall mean power or total energy expenditure during the 10 min period of self-paced cycling between the two conditions ($p = 0.27$, $p = 0.32$ and $p = 0.16$, respectively), or when individual 5 min blocks were analysed (Table 3). Further, there were no significant interaction effects (time and trial) for HR, RPE, VO_2, or RER (Table 4) during the 10 min time trial of self-paced cycling.

Six of the ten participants correctly identified their exposure to caffeine, due to feelings of restlessness, hyperactivity and shaky hands and legs. The remainder believed that they had not received the caffeine treatment, based on the fact that they did not feel any effects associated with caffeine ingestion.

Discussion

While the ergogenic benefits of caffeine have been well established in athletes, this is not the case in a sedentary population. Of importance, benefits associated with caffeine ingestion, i.e. the ability to perform more work, as well as a reduced sense of effort associated with exercise could be used to promote initial exercise performance in sedentary individuals who are prone to weight gain and associated health issues. Therefore, this study assessed the effect of caffeine on physiological variables in 10 sedentary females during a 15 min steady state aerobic exercise, as well as during a 10 min exercise protocol where participants were required to cycle as fast as they could. Overall, results from this study showed that caffeine resulted in significant increases in energy expenditure and VO_2 uptake during the steady state protocol (phase A) only.

These results were surprising considering the convincing evidence for significantly greater work being performed in set times after caffeine ingestion in an athletic population (Bridge and Jones; 2006; Collomp et al., 1992; Ivy et al., 1979; Schneiker et al., 2006). Furthermore, a meta analysis by Doherty and Smith (2005) reported that when compared to placebo, caffeine ingestion resulted in a 6% reduction in RPE during constant rate exercise. Lack of significant differences in exercise performance and other physiological variables may be due to the use of exercise durations (15 min for phase A and 10 min for phase B) which were considerably shorter than those undertaken in athletic studies that reported significant performance benefits (i.e. ≥ 30 min; Costill et al., 1978; Dodd et al., 1993; Graham and Spriet, 1991; Ivy et al., 1979). This conjecture is further supported by results from Ahrens et al. (2007a) and Ahrens et al. (2007b) that showed no significant change in RPE, HR and RER following caffeine ingestion during exercise protocols that lasted for only 8 min in recreationally fit women. Possibly a longer exercise duration is needed for the mechanisms that underlie the ergogenic effects of caffeine to result in significant differences. Further, use of $\%VO_{2max}$ rather than $\%HR_{max}$ as a guide for exercise intensity during phase A of the exercise protocol may have resulted in different physiological outcomes due to variability associated with HR values. Nonetheless, every effort was made to perform the exercise trials under similar conditions in order reduce HR variability (see methods section).

Training status may also play a role in eliciting exercise performance benefits associated with caffeine ingestion. To date, the majority of studies that reported significant improvement in exercise performance following caffeine ingestion involved well-trained athletes, whereas the effects of caffeine may be different in a non-

Table 4. Heart rate (HR), rating of perceived exertion (RPE), oxygen uptake (VO_2) and respiratory exchange ratio (RER) during 10 min of self-selected intensity cycling (Phase B). Values are means (±SD, n=10).

	Time	Placebo	Caffeine	Mean change (%) ± 90% confidence limits	P
HR	5 min	159 (17)	169 (14)	.59 (.50)	
	10 min	167 (21)	178 (10)	.53 (.49)	.69
RPE	5 min	13 (2)	13 (2)	-.06 (.38)	
	10 min	15 (2)	14 (2)	-.14 (.60)	.79
VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	5 min	21.74 (3.41)	23.59 (4.28)	.54 (.69)	
	10 min	22.97 (4.43)	25.22 (4.31)	.51 (.69)	.75
RER	5 min	.99 (.08)	1.01 (.09)	.23 (.59)	
	10 min	.94 (.05)	.95 (.06)	.08 (.63)	.43

P indicates interaction effect of time and trial

athletic population. This premise is supported by studies that assessed the effects of caffeine ingestion compared to placebo in sedentary males (Engels and Haymes, 1992), as well as in recreationally active individuals (Engels et al., 1999, Ahrens et al., 2007a; 2007b) and reported no significant differences in RPE (Ahrens et al., 2007a; 2007b; Engels et al., 1999), HR (Ahrens et al., 2007a; 2007b; Engels et al., 1999) or RER (Ahrens et al., 2007a; 2007b; Engels et al., 1999) during exercise. Of relevance, only steady-state exercise was performed in these studies and total work output was not assessed. It can be speculated that adaptive physiological changes that occur in trained athletes may result in greater responsiveness or sensitivity to caffeine uptake and its consequent effects. This may further imply that a higher dose of caffeine may be needed in a sedentary population in order to induce physiological benefit. To date, apart from the current study, only a 5 $mg \cdot kg^{-1}$ dose of caffeine has been trialed in a sedentary population (Engels and Haymes, 1992), while 3 – 6 $mg \cdot kg^{-1}$ doses have been used in studies involving recreationally active individuals (Ahrens et al., 2007a; 2007b; Engels et al., 1999). Further, the significantly lower RER values associated with caffeine ingestion reported in many endurance studies involving athletes (Bell and McLellan, 2002; Chad and Quigley, 1989; Costill et al., 1978; Graham and Spriet, 1991) may in part be due to the greater reliance fitter individuals have on fatty acids as a fuel source (McArdle et al., 2001) combined with caffeine's purported ability to mobilise free fatty acids (Engels and Haymes, 1992). Conversely, deconditioning, which in turn results in greater reliance on CHO as an energy source, may partly explain the insignificant difference found between RER values in sedentary participants in the current study. Again, adaptive processes that accompany regular exercise training may be required to induce significantly greater fatty acid use as a fuel (reflected by lower RER values) during endurance exercise. Further studies are needed to assess the effect of caffeine ingestion in untrained versus trained participants.

In addition, it is possible that caffeine ingestion may only have an ergogenic effect in a sedentary population when exercise is performed at higher exercise intensities. For instance, Engels et al. (1999) reported no significant change to VO_2 and RER in sedentary males after caffeine ingestion whilst walking at intensities equivalent to 30% and 50% VO_{2max}. Of relevance, the higher intensity exercise employed by Engels and Haymes (1992) is similar to the intensity used during steady-state cycling in the current study that equated to 65% individual HR_{max} (McArdle et al., 2001), which also resulted in similar results between caffeine and placebo trials. Conversely, a review by Graham (2001) noted that the majority of studies that reported improvement in exercise performance in an athletic population following caffeine ingestion used exercise intensities between 75-85% VO_{2max}. Another explanation for the results of this study may pertain to participants not being accustomed to regular exercise and consequently being reluctant to extend themselves during phase B of the protocol due to fear of injury or lack of confidence in their capabilities. The inclusion of extra exercise sessions may boost exercise confidence, which in turn may result in more work being performed during a set time, self-paced exercise routine.

Significantly higher VO_2 uptake associated with steady state exercise (phase A) after caffeine ingestion was also reflected by significantly higher energy expenditure. Previous studies assessing the effect of caffeine ingestion in a non-athletic population have reported mixed findings on VO_2 uptake during exercise. Engels et al. (1999) reported a significant increase in VO_2 and energy expenditure, without change in RER, after caffeine ingestion in recreationally active adults during a 60 min cycle exercise and suggested that these results were due to a number of mechanisms involving caffeine's stimulatory effects. Another study by Ahrens et al. (2007a) reported a significant 4% increase in VO_2 with just 8 min of treadmill walking performed at a moderate intensity, while Engels and Haymes (1992) reported no significant differences in VO_2 values following either caffeine or placebo ingestion during 60 min of walking at either 30% and 50% VO_{2max}. The increase in VO_2 reported by Ahrens et al. (2007a) was proposed to be due to an increase in stroke volume considering that HR values were not significantly different between trials (based on the Fick equation). This conjecture was also proposed by Hartley et al. (2004), who reported an increase in VO_2 as a consequence of a higher stroke volume (but not HR) in women at rest and during mental stress 45 min after a 3.3 $mg \cdot kg^{-1}$ ingestion of caffeine. As HR values were similar between trials in the current study, it is possible that an increase in stroke volume may have been responsible for the higher VO_2 and hence energy expenditure reported after caffeine ingestion during the steady-state cycling phase of the exercise. Other possible mechanisms responsible for these increases may relate to the stimulatory affects associated with caffeine ingestion (i.e. increased catecholamine release; Graham, 2001).

Conclusion

This study demonstrated that a moderate dose of caffeine did not improve cycling performance undertaken over a 10 min period in sedentary females, however caffeine ingestion significantly increased VO_2 and energy expenditure during steady state exercise. While this increase in energy expenditure represented only a 22 kJ difference between trials, this increase could be magnified if exercise duration and frequency were increased. It is possible that initial small improvements seen in the ability to exercise via caffeine ingestion may motivate sedentary individuals to make exercise a regular habit. This in turn could result in positive implications for weight management, fitness and health. Of importance, the addictive nature of caffeine, combined with possible associated health issues, such as sleep disorders (Landolt et al., 1995), caffeine induced anxiety (Fredholm et al., 1999), and raised mean arterial blood pressure (James et al., 1991), should be clearly described to individuals, with caffeine only used as a motivating tool during the initial stages of an exercise program. Further studies investigating the effect of caffeine in a sedentary population should

use extra exercise sessions, longer exercise durations and higher intensities, as well as higher doses of caffeine.

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Key points

- A 6.0 $mg \cdot kg^{-1}$ dose of caffeine did not improve work done ($J \cdot kg^{-1}$) or mean power (W) during 10 min of self-paced stationery cycling in sedentary female participants.
- A 6.0 $mg \cdot kg^{-1}$ dose of caffeine significantly increased VO_2 and energy expenditure (kJ) during 15 min of steady-state stationery cycling in sedentary female participants.
- A 6.0 $mg \cdot kg^{-1}$ dose of caffeine did not significantly affect RPE, RER or HR during 15 min of steady-state cycling or 10 min of cycling performed as fast as the participant could achieve, when compared to placebo, in sedentary female participants.

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