The effect of caffeine on high intensity upper body exercise performance
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Introduction
Caffeine has consistently been shown to be effective at improving lower body exercise involving 75-85% VO$_{2\max}$ cycling to exhaustion (Van Soeren and Graham 1998; Dendai and Dendai 1998; Greer, Friars and Graham 2000; Bell and McLellan 2002; Bell and McLellan, 2003; Hoffman et al. 2007; Hogervorst et al. 2008). Improvements of between 17.6 – 43.6% have been found. The only that has failed to show an effect of caffeine on time to exhaustion during lower body exercise is that of Bell, Jacobs and Zamecnik (1998).

Despite the large amount of research looking at time to exhaustion during lower body exercise, a comparatively small amount of work has been undertaken examining the upper body. Of the previous studies that have looked at caffeine’s effect on upper body exercise to exhaustion, the conclusions on caffeine’s efficacy have been mixed. Jacobs, Pasternak and Bell (2003) and Williams et al. (2008) found no effect of caffeine on upper body exercise to exhaustion. Beck et al. (2006) and Astorino and Rohmann (2008) found a 5% and 11% improvement in upper body exercise to exhaustion, respectively. However, these improvements did not reach significance. Forbes et al. (2007) found significant increases in upper body exercise to exhaustion of 6.3%. However, Forbes et al. (2007) used an intermittent exercise design, whereby total lever chest press repetitions were performed over three sets to exhaustion each separated by one minute rest, whereas all the other research cited in this current study has used a continuous exercise protocol. The only study to find a significant effect of caffeine on upper body exercise to exhaustion is that of Woolf, Bidwell and Carlson (2008), who found caffeine to be effective at increasing repetitions to exhaustion on a pneumatic eccentrically resisted chest press ergometer by 18.3%.

When looking at the studies closely related to the high intensity theme of this current study a dose of 5-6mg.kg$^{-1}$ of caffeine, 60 to 90 minutes prior to exercise, has been shown to be efficacious at increasing time to exhaustion in both upper and lower body exercise (Jackman et al. 1996; Bell, Jacobs and Ellerington 2001; Woolf, Bidwell and Carlson 2008). All used caffeine anhydrous, however Woolf, Bidwell and Carlson (2008) administered this in a drink artificially
sweetened with sucralose. Graham, Hibbert and Sathasivam (1998) also found 4.45 mg.kg$^{-1}$ of caffeine anhydrous administered in 7.15 ml.kg$^{-1}$ of water to be efficacious at increasing time to exhaustion. It would therefore seem a dose of 5 mg.kg$^{-1}$ would be optimal to increase performance. In these studies the subjects varied widely in their ages, training status and caffeine consumption. Jackman et al. (1996) recruited fourteen subjects (3 women and 11 men) with a mean age of 23.5 (± 2) who were recreational or varsity athletes of endurance activities. The level to which the subjects were already accustomed to caffeine was not stated. Bell, Jacobs and Ellerington (2001) recruited eight healthy male subjects with a mean age of 32 (± 5). It is reported that all habitually consumed the equivalent of at least one cup of brewed coffee a day (>100mg). Woolf, Bidwell and Carlson (2008) recruited eighteen male athletes with a mean age of 24.1 (± 5.8). Subjects were required to be performing over twelve hours of planned physical activity per week to be eligible for the study. The mean dietary caffeine intake of subjects was 40.8 (± 51) mg per day. From this it can be seen that in studies where caffeine was found to be effective subjects were physically active males in their mid twenties with a low to moderate habitual caffeine intake.

Despite the fact that time to exhaustion during high intensity exercise of the upper body has largely been found to be unaffected by caffeine, this may be due to multiple methodological flaws. In the study by Jacobs, Pasternak and Bell (2003), leg press repetitions to exhaustion were performed immediately before testing bench press repetitions to exhaustion. This may have resulted in central nervous system (CNS) and muscular fatigue from the leg press exercise that may have masked the ergogenic effect of the caffeine. Williams et al. (2008) only administered a 300mg dose of caffeine, compared to the 5-6 mg.kg$^{-1}$ that has been found effective in other studies this is significantly less and hence possibly why no ergogenic effect was found. Beck et al. (2006) used a similar low absolute dose of caffeine of 201mg from ‘mixed herbal sources’ (2.1-3.0 mg.kg$^{-1}$ in their subjects). Despite this a 5% improvement in 80% one repetition maximum (1RM) bench press repetitions to failure was found, however, as stated before this improvement was not significant. This may have been due to the low dose of caffeine from the questionable ‘mixed herbal sources’, but was more likely due to the fact the subject’s 1RM was tested immediately prior to performing repetitions to failure. This in turn could have produced significant CNS fatigue and resulted in a significant effect of caffeine being masked. Astorino
and Rohmann (2008) administered a proven, effective, 6mg.kg$^{-1}$ dose of caffeine, and found an 11% increase in 60% 1RM bench press repetitions to exhaustion, however, this improvement was not significant. This may have been in part due to the subjects being highly resistance trained. Subjects may have been experienced in how to improve mechanics of lifting as fatigue set in such as raising the hips off the bench to shorten range of movement, bouncing the bar off the chest, or not fully flexing or extending the arms.

In all of the above studies, the exercise protocol was one of the main methodological flaws. Implementing a previously successful lower body model such as that of Jackman et al. (1996) could possibly have aided in finding a significant effect of caffeine on exercise to exhaustion more readily in the upper body. Such a model could be more readily transferred to arm crank ergometry. Arm crank ergometry has been studied frequently as an upper body equivalent to cycling ergometry (Tesch and Lindberg 1984; Hopman et al. 1995; Miller, Mattacola and Santiago 2004; Bottoms 2008). Arm cranking has been successfully used as a well controlled high intensity upper body exercise protocol (Bottoms 2008), however, never in conjunction with caffeine. One advantage of arm cranking is that it is novel, even to trained athletes and therefore differing exercise experience between subjects can be eliminated as being a compounding variable.

The aim of this study was to further examine caffeine’s effects on high-intensity upper body exercise. The study was designed to examine the effect of an established acute dose of caffeine on an established upper body exercise protocol: arm-crank ergometry.

The research hypothesis proposed that similar improvements would be shown for upper body arm-cranking ergometry as for cycle ergometry TTE of a comparable intensity.

The research hypothesis is that similar relative improvements in time to exhaustion will be shown for high intensity arm cranking as for high intensity cycle ergometry.
Methods

Subjects
Six physically active males volunteered to take part in the study. Subjects regularly undertook between three to six hours of planned physical activity per week. Written informed consent was obtained using participant information sheets (Appendix 1) and PAR-Q (Heyward, 2002) forms. Subjects were excluded from the study if they had or developed any contraindications to caffeine usage or exercise testing during the study. Subjects were asked to abstain from caffeine, nicotine and alcohol for the 24 hours preceding all testing sessions and were also asked to keep their breakfast meal consistent before each of the three testing sessions. It was suggested to consume this meal with plenty of water at least two hours before testing. The descriptive characteristics of subjects, including the incremental test data is presented in Table 1 below.

Table 1. Characteristics of Subjects (n = 6).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>23 ± 3</td>
</tr>
<tr>
<td>Height (metres)</td>
<td>1.77 ± 0.1</td>
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<tr>
<td>Weight (kg)</td>
<td>77.9 ± 11.2</td>
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<tr>
<td>Arm Crank HR_{max} (bpm)</td>
<td>177 ± 8</td>
</tr>
<tr>
<td>Arm Crank VO$_{2max}$ (ml.kg.min$^{-1}$)</td>
<td>32 ± 7</td>
</tr>
<tr>
<td>Arm Crank W_{peak} (watts)</td>
<td>121 ± 27</td>
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Instrumentation
Subjects’ weight and height were recorded using a Seca 761 mechanical weighing scale and Seca 216 mechanical stadiometer (Seca, Birmingham, UK).
The ergometer was set-up in a similar fashion to that detailed by Bohannon (1986). A Monark 874 cycle ergometer was modified with free spinning rubber handgrips from a Monark 881 arm ergometer (Monark Exercise AB, Varberg, Sweden). The bike was then placed lengthways on a 3x6x3’ table and secured using g-clamps on the posterior feet of the ergometer. To allow the subjects to self monitor their own revolutions per minute (rev.min\(^{-1}\)) a web camera was set up on the handlebars which was attached to a laptop to stream the digital bike display on the left hand side of the bike. A fixed base chair was positioned at the end of the table and the distance was adjusted for each participant. Two 20kg Olympic barbell plates were positioned behind the chair to prevent it moving during the testing. Multiple fifty and one hundred gram cradle weights were modified by attaching coins using tape ensuring a weight of 83-84g and 166-167g, respectively. These equated to 5 and 10 watts each at 60 rev.min\(^{-1}\) arm cranking. During all testing subjects wore a Polar WearLink+ heart rate (HR) monitor (Polar Electro Oy, Kempele, Finland) and this was automatically recorded in conjunction with VO\(_2\) and RER using the MetaMax system (Cortex Biophysik GmbH, Leipzing, Germany).

**Experimental Design**

Subjects were required to attend three lab-based sessions. All sessions took place between 9 and 11 am. The initial session involved recording height and weight after which the arm cranking incremental protocol was explained to subjects. Subjects were also familiarised with the equipment and procedures and once they were confident each subject undertook the incremental protocol, primarily to determine their maximum attainable wattage (\(W_{peak}\)) and secondarily to attain arm cranking VO\(_2\)\(_\text{max}\) and HR\(_\text{max}\).

Familiarisation involved adjusting the equipment for each individual subject. The central axis of rotation of the arm-crank ergometer was horizontally orientated with the central axis of the shoulder joint. The chair was adjusted to allow for an approximate five degree elbow angle at full extension. Once this had been completed, subjects rested in a seated position for five minutes and baseline heart rate, VO\(_2\) and RER were recorded after which the incremental protocol began at 60 watts. The protocol required the subjects to maintain 60 rev.min\(^{-1}\) while the workload was increased 10 watts every minute until 60 rev.min\(^{-1}\) could not be sufficiently maintained or
volitional exhaustion occurred. During the incremental protocol subject’s heart rate, \( \text{VO}_2 \) and RER were recorded every minute until exhaustion. The wattage at which the protocol was terminated was considered the subject’s \( W_{\text{peak}} \).

The second session took place seven days after the initial familiarisation and incremental protocol. Subjects were randomly divided into two groups and assigned either caffeine anhydrous (MyProtein.co.uk, Cheadle, Cheshire, UK) at 5mg.kg\(^{-1}\) or placebo (nothing) diluted in 500ml of artificially sweetened (sucralose) orange flavour drink, made up with a ratio of 1:4 concentrate to water. This volume of water was used as Graham, Hibbert and Sathasivam (1998) found that a 7.15ml.kg\(^{-1}\) (this was equivalent to 557ml for the current study, based on mean weight) volume of water to be effective at improving time to exhaustion when consumed along with caffeine anhydrous. Subjects consumed the drink one hour before initiation of the testing session as caffeine is rapidly absorbed from the gastrointestinal tract, with plasma levels peaking at 60-90 minutes post ingestion (Jeukendrup and Gleeson 2004, Ruxton 2008). Subjects were also advised to consume additional water to maintain hydration before the testing.

Subjects were then seated at the ergometer for 5 minutes, at which point the baseline heart rate, \( \text{VO}_2 \) and RER were recorded. The subjects then commenced a 3 minute warm-up at 60 rev.min\(^{-1}\), after which the cradle was lowered having already been loaded with 90% \( W_{\text{peak}} \) resistance. Subjects were required to maintain 60 rev.min\(^{-1}\) for as long as possible and were verbally encouraged to do so. Heart rate, \( \text{VO}_2 \) and RER were recorded every minute until the subject reached volitional exhaustion. Subjects were then allowed to recover and were monitored until heart rate reached baseline measurement.

The third session took place 7 days later and involved the same procedures as the second session, except the crossover design was implemented and subjects received the opposite treatment to that they had received for the second session.

**Statistical Analysis**

Means and standard deviations were calculated for all variables and normality was assessed. A two way repeated measures ANOVA was used to assess for significant differences in \( \text{VO}_2 \), RER
and heart rate across treatments and time and Tukey post hoc tests were used to determine where any significant differences lay. Independent samples t-tests were used to examine VO₂, RER and heart rate at exhaustion between trials, to examine trial order and differences in mean time to exhaustion between trials. Statistical significance was accepted at the $P < 0.05$ level.

**Results**

Figure 2 shows a comparison of time to exhaustion for the caffeine and placebo trials. Caffeine ingestion was associated with a significant increase ($P < 0.05$) in time to exhaustion compared with placebo. Time to exhaustion (mean ± SD) was 4:53 ± 1:20 and 5:57 ± 1:54 minutes for the placebo and caffeine trials, respectively. There was no significant effect on time to exhaustion due to the order of the trials ($P > 0.05$).

Figure 3 shows VO₂ during the 90% Wpeak exercise to exhaustion. VO₂ increased rapidly from rest in both trials and continued to increase throughout exercise ($P < 0.01$; main effect for time). There was no significant effect on VO₂ at rest and during exercise due to caffeine ($P > 0.05$) however, caffeine resulted in a significantly higher VO₂ at exhaustion than placebo ($P < 0.01$). Subjects reached 92.7 ± 18.2% and 101.5 ± 21.6% of their previously established VO₂max during the placebo and caffeine trials respectively.

Figure 4 shows RER during the exercise at 90% Wpeak to exhaustion. RER increased rapidly from rest in both trials and continued to increase throughout exercise ($P < 0.01$; main effect for time), however began to plateau at two minutes ($P > 0.05$). There was no significant effect on RER at rest, during exercise or at exhaustion due to caffeine ($P > 0.05$).

Figure 5 shows heart rate during 90% Wpeak exercise to exhaustion. Heart rate increased rapidly from rest in both trials and continued to increase throughout exercise ($P < 0.01$; main effect for time). There was no significant effect on heart rate at rest and during exercise due to caffeine however, caffeine resulted in a significantly higher heart rate at exhaustion than placebo ($P < 0.05$). Subjects reached 98.7 ± 5.6% and 101.6 ± 6.1% of their previously established HRmax during the placebo and caffeine trials respectively.
Figure 2. Time to exhaustion at 90% $W_{\text{peak}}$ after caffeine or placebo ingestion.
* Significantly longer time to exhaustion.

Figure 3. Oxygen consumption (L.min$^{-1}$) during exercise at 90% $W_{\text{peak}}$ to exhaustion.
* Significantly higher VO$_2$ at exhaustion ($P < 0.01$). ♦ = placebo, ■ = caffeine.
**Figure 4.** Respiratory exchange ratio during exercise at 90% $W_{\text{peak}}$ to exhaustion.

♦ = placebo, ■ = caffeine.

**Figure 5.** Heart rate (bpm) during exercise at 90% $W_{\text{peak}}$ to exhaustion.

* Significantly higher heart rate at exhaustion ($P < 0.05$). ♦ = placebo, ■ = caffeine.
Muscular RPE at exhaustion (mean ± SD) was 18 ± 1 and 18 ± 2 for the placebo and caffeine trials, respectively. Cardio respiratory RPE at exhaustion (mean ± SD) was 15 ± 2 and 15 ± 3 for the placebo and caffeine trials, respectively. There was no significant effect on either Muscular RPE or Cardio respiratory RPE at exhaustion due to caffeine ($P > 0.05$).

**Discussion**

The effectiveness of caffeine as a performance enhancer during lower body exercise is well documented. The results of the current study extend earlier observations by demonstrating that caffeine can also enhance upper body performance as reflected by the increased time to exhaustion during high-intensity arm cranking exercise. This is the first study to document such a finding. The current study is the first to provide evidence that caffeine can indeed enhance physical performance of a nature that would likely be advantageous during competitive sports where upper body exercise to exhaustion or near exhaustion is commonplace.

The order in which the subjects undertook the trials proved to be insignificant, indicating there was no training effect produced from arm cranking exercise. Therefore the increase in time to exhaustion was due to caffeine consumption.

In this study, the acute ingestion of caffeine resulted in a significant mean improvement of 1:04 minutes or 21% in time to exhaustion during arm cranking exercise. Only one study examining 90% $W_{\text{peak}}$ arm cranking has been conducted by Bottoms (2008), however unfortunately a fixed exercise time of five minutes was used and it therefore cannot be used to compare to the current study’s time to exhaustion.

The only study to find a significant effect of caffeine on upper body exercise to exhaustion is that of Woolf, Bidwell and Carlson (2008) who used a pneumatic eccentrically resisted chest press ergometer and examined maximal repetitions to exhaustion. The 18.3% improvement is very similar to the 21% found in the current study. This may be due to several similarities in the methodology. The upper body exercise protocol used is similar to arm cranking in that it is only eccentrically loaded and in a fixed pattern of movement, which is governed by the ergometer mechanics. Also, the $5\text{mg.kg}^{-1}$ dose of caffeine was administered, in a drink artificially
sweetened with sucralose, sixty minutes prior to exercise, identical to the current study. Despite these similarities this exercise test was not the main focus of the study. However, the exercise mode and intensity used was still very different from that of the current study.

Jackman et al. (1996) found that time to exhaustion was increased by 19.7% with a 6mg.kg\(^{-1}\) dose of caffeine 60 minutes prior to a 100% VO\(_{2\text{max}}\) cycle exercise trial to exhaustion. This exercise intensity is comparable to that of the current study as it was observed that at exhaustion subjects were performing at their relative VO\(_{2\text{max}}\). Times to exhaustion were 4:07 and 4:56 minutes for the placebo and caffeine trials respectively. These durations are very similar to those of the current study and therefore it can be assumed that the relative intensity is similar.

Taking into account the similar methodologies and relative exercise intensities of Jackman et al. (1996) and Woolf, Bidwell and Carlson (2008) the current study compares favourably with regards to time to exhaustion. However, obviously these similarities are speculation as the exercise modalities are very different.

Oxygen consumption and heart rate increased with exercise, however, no significant effect on oxygen consumption or heart rate during exercise was found from caffeine supplementation. At exhaustion however subjects were found to have a significantly higher oxygen consumption and HR with caffeine.

Bottoms (2008) states that at the cessation of 90% W\(_{\text{peak}}\) exercise heart rate was 92 ± 4% of HR\(_{\text{max}}\) and oxygen consumption was 97 ± 19% VO\(_{2\text{max}}\). These values are very close to the values at exhaustion for the placebo trial in the current study.

Of those studies that did find a significant effect of caffeine on exercise to exhaustion Woolf, Bidwell and Carlsson (2008) found a main effect for time on heart rate, with the highest values being at exhaustion. However, no effect was found for trial at rest or throughout exercise. Unfortunately no gasses were collected during these upper body studies.
The findings of the current study are similar to those of Horgevorst et al. (2008) with regards to increased heart rate as exhaustion with caffeine, however the methodology is very different. This increase observed in heart rate and \( \text{VO}_2 \) at exhaustion is commensurate with the longer duration of exercise when compared to placebo. At the intensity of exercise employed in this study, it is known that both the oxygen consumption and heart rate increase the longer an individual exercises (Schneider, Wing and Morris 2002). Thus it is unlikely the ergogenic effects of caffeine were due to an increased \( \text{VO}_2 \text{max} \) or \( \text{HR}_\text{max} \). This theory is supported by subjects \( \text{VO}_2 \) and HR at exhaustion not being significantly different from their \( \text{VO}_2 \text{max} \) and \( \text{HR}_\text{max} \).

No significant effect of caffeine on RER was observed. RER was found to increase with exercise and plateau after two minutes in both caffeine and placebo trials to between 1.3-1.4 until exhaustion. This indicates a very strong reliance on anaerobic respiration. The studies in which RER was measured at workloads of 75-85% no effect of any dose of caffeine was found (Bell, Jacobs and Zamecnik, 1998; Van Soeren and Graham, 1998; Greer, Friars and Graham, 2000; Bell and McLellan, 2002; Bell and McLellan, 2003). These studies methods do not directly relate to the methods of the current study, but the finding that caffeine does not result in significant changes in RER is a common feature.

RPE was not significantly different at exhaustion with caffeine supplementation. Bottoms (2008) used a similar system of central RPE (cardio respiratory) and local RPE (muscular) and states that after a work load of 90% \( W_{\text{peak}} \) for 5 minutes duration RPE values were 16 and 18 for cardio respiratory and muscular respectively. For both trials in the current study at exhaustion the muscular value was identical, however the cardio respiratory value was 1 point lower when compared to Bottoms (2008).

It has been demonstrated that if recorded during lower body exercise to exhaustion RPE is significantly lower with caffeine (Bell and McLellan 2002; Bell and McLellan 2003). It has been consistently demonstrated that RPE is not significantly influenced by caffeine at exhaustion in both upper (Woolf, Bidwell and Carlson 2008; Astorino and Rohmann, 2008) and lower (Bell and McLellan 2002; Hogervorst et al. 2008) body studies. The fact the RPE values showed no significant difference at exhaustion in the current study indicates subjects perceived to give equal
levels of effort in both trials. An observation that was made is when subjects received placebo instead of caffeine they usually followed up their RPE scores with a statement to the effect of an increased or decreased perception of lactate build-up.

Caffeine acts as an adenosine receptor antagonist. When caffeine is consumed, the resultant cascade of cellular events that follow adenosine receptor blockade, including increased dopamine and nor-adrenaline release have been proposed as key regulatory mechanisms to explain the ergogenic effect of the drug (Bell and McLellan 2002).

Both Jackman et al. (1996) and Woolf, Bidwell and Carlson (2008) theorize that caffeine’s ergogenic effects are the result of the effects on the central nervous system (CNS) by acting as a potent adenosine antagonist, thus blocking adenosine receptors or interacting with dopamine receptors, as adenosine inhibits the release of most excitatory neurotransmitters in the brain, particularly dopamine, and might reduce dopamine synthesis. Because adenosine receptors are abundant in many areas of the brain, caffeine might inhibit adenosine from blocking neuronal transmission and thus alter perception of pain, sympathetic activity, motor recruitment, and fatigue. It is in this capacity that caffeine may increase time to exhaustion. Because caffeine is a dopamine agonist, it could possibly reduce RPE. However, as in the current study, Woolf, Bidwell and Carlson (2008) note that no reduction in RPE was seen with caffeine. One important factor to note however, is that RPE values were recorded at exhaustion. Since caffeine was found to increase time to exhaustion in both the current study and that of Woolf, Bidwell and Carlson (2008) and RPE was similar for both caffeine and placebo groups, it would be reasonable to assume both groups perceived working equally as hard at exhaustion despite the prolonged effort of the caffeine groups. This would lead one to speculate that had RPE been taken mid exercise it may have been found to be reduced with caffeine, as it was in several aerobic lower body studies (Bell and McLellan 2002; Bell and McLellan 2003). However, Jackman et al. (1996) states that the majority of the subjects could not correctly identify the caffeine trial and that any effect of the caffeine on the CNS was not obvious to the subjects themselves.

It is also likely adenosine receptors within the muscle could be antagonized (Woolf, Bidwell and Carlson 2008). Silinsky and Redman (1994) state that at the myo-nucleal junction ATP is
released concurrently with acetylcholine. The ATP is degraded, producing adenosine, which
binds to the motor neuron’s A1 receptors to inhibit further neural transmitter release. Caffeine is
known to be an adenosine antagonist and therefore could disinhibit this response. Svenningson et
al. (1997) found that caffeine doses as low as 7.5mg.kg\(^{-1}\) may result in an increase in motor
activity in rats primarily through the antagonism of adenosine receptors. These findings are
supported by the more recent work of Davis et al. (2002) who found that, in rats, treadmill run
time was significantly increased with caffeine, despite concurrent administration of the
adenosine receptor agonist (5’-N-ethylcarboxamidoadenosine), indicating that caffeine can delay
fatigue through CNS mechanisms, at least in part by blocking adenosine receptors. Woolf,
Bidwell and Carlson (2008) also theorise the more trained an athlete is the more muscle mass
they will carry and therefore the more adenosine receptors available to antagonize within the
muscle when compared to an untrained individual, likely producing a more ergogenic effect.

As the finding’s of the current study were similar to those of Jackman et al. (1996) and Woolf,
Bidwell and Carlson (2008) it is proposed adenosine receptor antagonism was likely responsible
for the significantly increased time to exhaustion. This was likely manifested as a decreased
perception of fatigue.

As the current study was the first to examine caffeine’s effects on high intensity arm crank
ergometry there were multiple limitations and consequent opportunities for improvement. The
subjects used for the study were all active, young, university students however, there was no
requirements for a specific upper body training status. This variation in training status manifested
itself in the variation in times to exhaustion, despite subjects performing experimental trials at
identical workloads (90% \(W_{\text{peak}}\)). The minimum time to exhaustion was 3:10 minutes and the
maximum was 9:03. This is likely to have been caused by variation between subjects in their
respective lactate thresholds (LT) and lactate turn point’s (LTP), which can be influenced by
training status (Schneider, Wing and Morris 2002). This variation in LT and LTP would have
resulted in differing \(\text{VO}_2\) slow components and therefore differing times to exhaustion. The
effect of the \(\text{VO}_2\) slow component on time to exhaustion is further compounded by the fact arm
cranking to exhaustion results in significantly more type II muscle fibres being recruited when
compared to lower body exercise to exhaustion (Schneider, Wing and Morris, 2002; Bernasconi
et al., 2006) as the upper body contains a larger percentage of these fibres. These are likely the most frequently trained fibres through resistance training of the upper body. In future studies the training status and LT and LTP should be closely matched between subjects.

A major limitation of the current study was that it was single blind only. The researcher knew who was taking the caffeine and consequently consciously or subconsciously may have encouraged the subjects who had taken caffeine to perform for longer through motivational talk. A double blind study could be implemented in the future as this would avoid the chance of any psychological intervention to aid performance, from the researchers.

The RPE should have been recorded during exercise to help confirm the adenosine receptor theory presented above. This was not done during this current study as subjects were occupied with their upper body and therefore couldn’t point to a Borg scale, but also were wearing the gas analysis equipment so would have found it hard to be understood mid exercise. A suggestion might be to take a pause at absolute intervals during exercise to establish RPE. In addition to this the perception of which condition subjects felt they had been allocated to after each trial would provide information to either confirm or dispute the findings of Jackman et al (1996) that subjects could not successfully determine if they had been administered caffeine or not.

Some other general considerations for future research are including females in the research, however, making sure that all subjects are not taking oral contraceptives, as this will double the clearance time of the caffeine (Lorist and Tops, 2004). Subjects were instructed to consume a normal breakfast prior to morning testing, however dietary logs would also have been useful for at least 24 hours before each testing session. Included with this could have been a caffeine questionnaire to determine if subjects were habitual caffeine consumers or not. This could then be accounted for in future research to better understand the dose-response relationship for caffeine users and non-users. In future studies a comparison of upper body ergometry and cycling time to exhaustion at the same relative workloads would make for easier comparison to past research examining cycling time to exhaustion.
The current study has found that exhaustive high intensity upper body exercise can be significantly prolonged with caffeine supplementation. Implications of these findings at this early stage of research are difficult to make. However, these findings would indicate that athletes such as kayakers and wheelchair athletes would benefit from caffeine supplementation prior to exhaustive training bouts.

**Conclusion**

In conclusion this study was the first to investigate the effect of caffeine on high intensity upper body ergometry to exhaustion. The research hypothesis was accepted and the finding of a 21% improvement in time to exhaustion is consistent with many lower body studies and may be due to the antagonism of adenosine receptors causing a lower RPE during exercise.

**References**


Bell, D. G., McLellan, T.M. 2002.’Exercise endurance 1, 3 and 6 h after caffeine ingestion in caffeine users and nonusers’. *Journal of Applied Physiology* 93, 1227-1234.


