

*Original Research*

## Effects of Caffeine on Repeated Upper/Lower Body Wingates and Handgrip Performance

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### ABSTRACT

*International Journal of Exercise Science* 8(3): 243-255, 2015. Caffeine enhances aerobic performance, but research is equivocal regarding anaerobic performance. This study examined effects of caffeine (7 mg/kg) on anaerobic performance in anaerobically active males (n = 10). Participants completed counterbalanced, double blind caffeine (Caf) and placebo (Pl) trials including a) 6 x 15 s upper body Wingates (UW<sub>ant</sub>), b) 6 x 15 s lower body Wingates (LW<sub>ant</sub>) and c) 6 x 15 s maximal effort static hand grip test (HG) with 3 min recovery between bouts, 30 min between exercises. Peak power (P<sub>peak</sub>), mean power (P<sub>mean</sub>), and heart rate (HR) as well as perceptual measures included ratings of perceived exertion (RPE), muscle pain perception (MPP), and perceived recovery status (PRS) were recorded per bout. Session RPE (S-RPE) (15 min post) for each exercise mode and trial RPE (T-RPE) [10 min post relative to testing period for each treatment (Caf vs. Pl)]. A series of 2 (trial) x 6 (bout) ANOVA's assessed differences and Tukey's LSD post hoc test were used when necessary. Results showed increased performance (main effect) (UW<sub>ant</sub>) for P<sub>peak</sub> (Caf: 6.72 ± 1.2 W/kg vs. Pl: 6.41 ± 1.0 W/kg); and P<sub>mean</sub> (Caf: 5.39 ± 0.8 W/kg vs. Pl: 5.18 ± 0.8 W/kg); however no significant main effect for LW<sub>ant</sub> or HG was observed. No significant differences were observed for perceptual measures. Caf improved anaerobic performance in repeated UW<sub>ant</sub> (bouts 1-4) but not LW<sub>ant</sub> or HG. Further studies are warranted to examine Caf ergogenic properties in repeated exercises dominated by anaerobic metabolic pathways given the equivocal results.

KEY WORDS: Session RPE, heart rate, ergogenic aid, perceived recovery scale

### INTRODUCTION

Caffeine (Caf) is a central nervous stimulant and a common constituent of supplements and energy-drinks based on the premise of improved physical performance with associated short term side effects including headache, nausea, and tremors with mild consumption. However, long term caffeine use is not well understood. The National

Collegiate Athletic Association (31) mandates urinary levels > 15 µg/ml as illegal for competing athletes. This equates to 13 mg/kg or roughly 8 cups of coffee (12, 17). Current literature has documented caffeine as an ergogenic aid in endurance exercise (For reviews see; Goldstein (14) and Magkos (29)). However, a review by Davis (9) indicates that research investigating caffeine's impact on anaerobic

performance has produced equivocal results, potentially attributable to testing of untrained individuals and habitual vs. non-habitual caffeine users, diverse modes of testing (wingates vs. traditional weight lifting), muscle mass tested, and administration of varying caffeine dosages (2-10mg/kg).

Lower body Wingate anaerobic tests ( $W_{ant}$ ) are an accepted model to assess anaerobic capacity (23). However, the efficacy of caffeine on  $W_{ant}$  performance remains unclear. Greer (20) and Williams et al. (37) found caffeine (6-7 mg/kg of body mass) resulted in no change in repeated (20) or single effort (37) Wingates ranging from 15-30 s. However, the authors attributed results to the untrained status of participants. Woolf et al (39) found caffeine (5 mg/kg) significantly increased total weight (reps x resistance) lifted for bench press and peak power for Wingate for highly fit males. However, total weight lifted for leg press only approached significance ( $p = 0.09$ ). Anselm et al. (2) showed ingestion of 250 mg of caffeine prior to exercise increased peak anaerobic power in anaerobically untrained participants during a 6 s  $W_{ant}$ . Additionally, Kang et al. (26) found caffeine ingestion of both 2.5 and 5 mg/kg improved  $W_{ant}$  performance in untrained individuals. Conversely, Greer et al. (19) showed caffeine (6 mg/kg) had no ergogenic effect during repeated (4 x 30 s)  $W_{ant}$  with some evidence caffeine hindered performance in later bouts.

Bugyi et al. (6) showed no significant difference in hand grip (HG) strength after caffeine ingestion of 167, 324, and 500 mg. However, caffeine dosage was not based on

participant's body mass and some participants likely failed to reach critical levels of caffeine ingestion (i.e. > 3-9 mg/kg of body mass) previously shown to elicit an increase in performance (17, 9). Further, with HG testing, the lack of mode specific trained participants could mitigate potential ergogenic properties of caffeine. Bellar et al. (5) observed a mild (not significant) difference in HG to time to failure (Caf:  $104.98 \pm 57.95$  s; Pl:  $99.85 \pm 78.39$  s) with a concurrent significant reduction in subjective pain response (Caf:  $3.45 \pm 2.95$ , Pl:  $4.84 \pm 2.92$ ). It is inconclusive if the small volume of muscle involved in HG testing creates a paradigm less conducive to observing a performance-enhancing effect from caffeine.

Caffeine's ergogenic properties were originally theorized due to enhanced free fatty acid mobilization consequent to glycogen sparing, however this provides minimal impetus for enhanced performance (with acute caffeine supplementation) during exercise dominated by oxygen-independent metabolic pathways justifying further investigation. However, caffeine has been shown to have analgesic properties and a consistent blunting of perceptual pain responses in steady state exercise (27). Far less investigation into caffeine's analgesic properties in repeated high intensity efforts has occurred. Caffeine buffers pain (vs. placebo), evidenced by perceptual and pain responses being blunted when similar work is completed or unaltered when greater work is performed (3, 18, 22, 34). Additionally, reduction in perceived levels of pain may be dose dependent per individual (32). While analgesic potential of caffeine offers a reasonable mechanism, discrepancies among previous studies

make further research warranted to clarify caffeine's effect on anaerobic performance. Additionally, there appears to be a dose response effect with caffeine to potentially observe ergogenic benefits (3-7mg/kg). Therefore, this study examined effects of caffeine ingestion at 7mg/kg on repeated upper and lower body  $W_{ant}$  and hand grip performance in anaerobically trained males.

**METHODS**

*Participants*

Participants were males (ages 19-45) who frequently engaged in high-intensity training (HIT) methods involving both upper and lower body exercises >3 days/week. Participants completed an informed consent form and were screened for apparent chronic disease risks using the PAR-Q (38) and a health questionnaire. Participants also completed questionnaires concerning daily caffeine use (33) and training history. Height (cm) and body mass (kg) were determined using a stadiometer (Detecto, Webb City, MO) and a calibrated digital scale (Tanita BWB-800, Tokyo, Japan). Body fat percentage was estimated using skinfold calipers (Lange, Cambridge, MD) and the three site method for males (chest, abdomen, thigh) (24). During each trial, peak heart rate was obtained using Team2 system heart rate monitors (Polar Electro Oy, Kempelee, Finland). Participants were instructed to report to the lab for testing well-hydrated, having avoided consumption of caffeine, alcohol, and any heavy physical exertion 24 h prior to all trials. Participants documented their dietary intake 24 h prior to the first session which served as a familiarization trial and were instructed to duplicate the diet for each trial.

Participants followed these instructions and replicated dietary intake for all subsequent trials. Descriptions of exercise testing are included below and were replicated for the familiarization session and both treatment sessions. All procedures were approved by the institutions internal review board (IRB) and in followed procedures in accordance with the ethical standards of the Helsinki Declaration.

**Table 1.** Descriptive characteristics for participants (n = 10).

Variable	Mean	SD
Age (yrs)	23.9	5.4
Height (cm)	179	6
Body mass (kg)	86.0	10.4
BMI	26.8	2.3
Body Fat (%)	10.9	2.4
Daily caffeine use (mg/day)	189.0	119.7
Daily caffeine use (mg/kg)	2.21	1.3

*Protocol*

Participants completed a 5 min upper body cycle specific warm-up on an ergometer (Ergomedic 828E, Monark Inc., Varberg, Sweden) equipped with handgrips and mounted on a table designed for arm ergometry. Participants completed 6 x 15 s upper body  $W_{ant}$  ( $UW_{ant}$ ). Resistance was individualized at a ratio of 0.062 kp/kg of body mass (23) and the weight basket was automatically dropped upon reaching 120rev/min. Each bout was separated by 3 min of passive recovery. Peak power ( $P_{peak}$ ), mean power ( $P_{mean}$ ), and fatigue percentage (Ftg%) were collected. Subjective data included rating of perceived exertion (RPE) for each bout 1 min into recovery. Session (S-RPE) was collected 15 min after completion of the last  $UW_{ant}$  using the Omni Pictorial 0-10 scale (36). The Perceived Recovery Scale (PRS) (28) was used to assess participant's subjective feelings of readiness 15 s prior to the start

of each  $W_{ant}$  on a 10cm visual analog scale Muscle-pain perception (MPP) was assessed using a previously validated scale (8). MPP was assessed after 2 min recovery following each 15 s Wingate on a 10cm visual analog scale. Upon completion of the  $UW_{ant}$ , participants arm cycled (20-30 rev/min with no resistance) as an active "cool-down" period for 5 min.

Participants recovered in a seated position for 30 min upon the completion of the last  $UW_{ant}$  before the lower body  $W_{ant}$  ( $LW_{ant}$ ).  $LW_{ant}$  were conducted in the exact manner as  $UW_{ant}$ . Participants completed a 5 min warm-up at a standardized 50-60 rev/min (with no resistance) on a cycle ergometer (Ergomedic 894E, Monark Inc., Varberg, Sweden). Participants completed 6 sets of 15 s  $LW_{ant}$ . Resistance was a ratio of 0.087 kp/kg of body mass (23) and the weight basket was automatically dropped upon reaching 120rev/min. Upon completion of the last  $LW_{ant}$ , participants cycled 5 min at 30-40 rev $\cdot$ min<sup>-1</sup> with no resistance as an active "cool-down".

Participants completed 6 x 15 s maximal effort hand grip strength tests (HG) with each hand (Baseline Hydraulic Hand Dynamometer, Fabrication Enterprises Inc., Irvington, NY) after completion U and L  $W_{ant}$ . Participants completed the trials in a standing position, arms at sides, and dominant hand first. Each bout was separated by 3 min passive recovery. Peak and minimum power, RPE, MPP, PRS, and S-RPE were recorded in the same manner as with Wingate testing. Trial (T-RPE) was collected 20 min after completion of trial (Caf and Pl) using the Omni Pictorial 0-10 scale (36).

Participants ingested either caffeine (Caf) (7 mg/kg) or matched placebo (Pl) pills (lactose) in soft capsule form, 1 h prior to reporting to the laboratory. The primary investigator prepared paired sets of pills (Caf and Pl) in clear storage bags and received verbal confirmation of ingestion 1 hr prior to trial by the participant. Caf and Pl trials were completed in a counterbalanced order and in a double-blind manner.

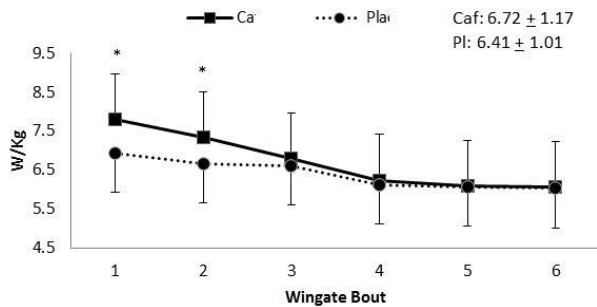
#### *Statistical Analysis*

Data was analyzed utilizing SPSS and an alpha of 0.05 was set a priori. A series of 2 (trial) x 6 (Wingate) repeated measures ANOVA's were used to compare dependent measures including performance ( $P_{peak}$ ,  $P_{mean}$ , Ftg%) and perceptual data (MMP, RPE, and PRS). A two-way repeated-measures ANOVA and Tukey's LSD post hoc test were used to examine differences in HR. Session RPE and Trial RPE were analyzed between Caf and Pl trials using paired samples T-test per exercise mode.

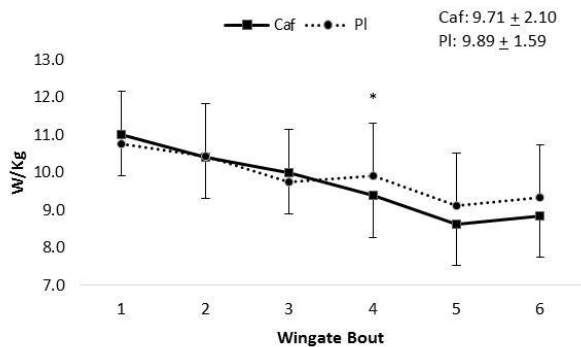
## **RESULTS**

Descriptive data are shown in Table 1. Trial means for performance are shown in Table 2. Upper Body Wingate: A significant main effect for trial was observed for  $P_{peak}$  (Figure 1) and  $P_{mean}$  (Figure 2). Follow up tests showed significantly greater values for CA for  $P_{peak}$  on bouts 1 and 2 (Figure 1) and for  $P_{mean}$  for bouts 1, 2 and 4 (Figure 2). No significant difference was observed for HR (b/min) (Caf:  $158 \pm 16$  vs. Pl:  $158 \pm 10$ ). No Significant differences were found for Ftg% (Caf:  $60.5 \pm 1.4$  % vs. Pl:  $60.5 \pm 3.5$  %), RPE, MPP, PRS, S-RPE. No significant difference

was found in T-RPE (Caf:  $6.2 \pm 0.8$  vs. Pl:  $5.9 \pm 1.4$ ).



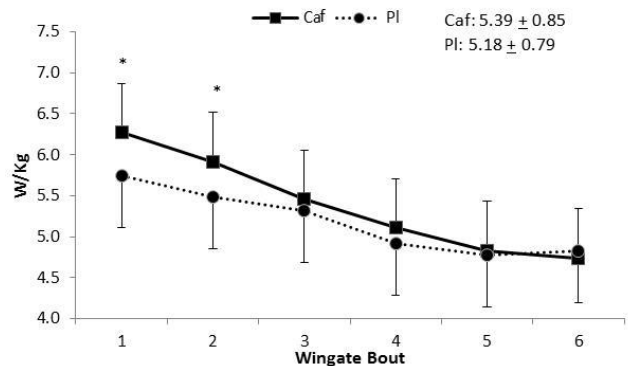
**Figure 1.** Caf vs. Pl UW<sub>ant</sub> P<sub>peak</sub>. \*Caf significantly higher than Pl per bout. Caf significantly higher than Pl,  $p = 0.04$  (main effect).



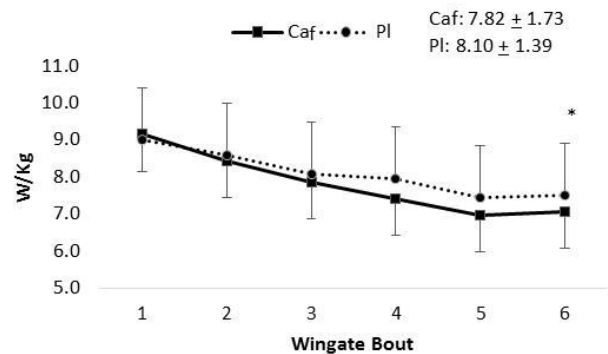
**Figure 2.** Caf vs. Pl LW<sub>ant</sub> P<sub>peak</sub>. \* Pl significantly higher than Caf  $p = 0.04$ .

Follow up tests showed significantly greater values for Pl for PRS on bouts 4, 5, and 6. No significant difference was observed for mean LW<sub>ant</sub> P<sub>peak</sub> (Figure 3). A significant main effect was observed for P<sub>mean</sub> (Figure 4). Follow up tests showed significantly greater values for Pl for P<sub>mean</sub> on bout 6 (Figure 4). HR was significantly different for LW<sub>ant</sub> (Caf:  $163 \pm 7$  b/min vs. Pl:  $158 \pm 8$  b/min) and follow up tests showed significantly greater values for Caf for HR on bouts 2, 3, 4, 5, and 6. Additionally, LW<sub>ant</sub> PRS was significantly higher for Pl (Table 2) No significant differences were observed for LW<sub>ant</sub> Ftg%

(Caf:  $59.2 \pm 2.9$  % vs. Pl:  $59.2 \pm 4.0$  %), RPE, MPP, or S-RPE (Table 2).



**Figure 3.** Caf vs. Pl UW<sub>ant</sub> P<sub>mean</sub>. \*Caf significantly higher than Pl. Caf significantly higher than Pl,  $p = 0.009$  (main effect).



**Figure 4.** Caf vs. Pl LW<sub>ant</sub> P<sub>mean</sub>. \*Pl significantly higher than Caf. Pl significantly higher than Caf,  $p = 0.03$  (main effect).

No significant differences were found between trials for HG peak power (Caf:  $52.7 \pm 1.7$  kg vs. Pl:  $53.0 \pm 1.3$  kg), RPE, MPP, or PRS. HG HR (b/min) was found significantly higher (Caf:  $130 \pm 15$ , vs. Pl:  $122 \pm 16$ ) and follow up tests showed significantly greater values for Caf for HR for all bouts No significant difference was found in HG S-RPE (Table 2).

## DISCUSSION

The major finding of the current study was 7 mg/kg of Caf resulted in a significant

**Table 2.** Perceptual data for Caf vs. Pl. Means and standard deviations.

Variable	Caffeine	Placebo	p value
UW <sub>ant</sub> RPE mean	5.5 ± 1.6	5.2 ± 1.3	0.16
LW <sub>ant</sub> RPE mean	6.1 ± 1.6	5.8 ± 1.5	0.11
HG RPE mean	3.3 ± 1.5	2.7 ± 1.2	0.41
UW <sub>ant</sub> PRS	5.3 ± 2.2	5.7 ± 1.8	0.23
LW <sub>ant</sub> PRS	5.0 ± 2.2	5.7 ± 1.9	0.02
HG PRS	7.1 ± 1.9	7.6 ± 1.3	0.17
UW <sub>ant</sub> MPP	4.1 ± 2.4	3.9 ± 1.8	0.43
LW <sub>ant</sub> MPP	4.8 ± 1.8	4.2 ± 2.2	0.13
HG MPP	2.3 ± 1.5	2.0 ± 1.4	0.21
UW <sub>ant</sub> S-RPE	6.1 ± 1.4	5.8 ± 1.1	0.09
LW <sub>ant</sub> S-RPE	7.0 ± 0.9	6.4 ± 1.5	0.20
HG S-RPE	3.4 ± 1.5	3.4 ± 1.7	0.50

increase in  $P_{peak}$  and  $P_{mean}$  in UW<sub>ant</sub> Caf, while no significant difference was observed in  $P_{peak}$  in LW<sub>ant</sub> and HG performance. However, Caf resulted in a significantly greater  $P_{mean}$  in LW<sub>ant</sub>. Further, RPE for UW<sub>ant</sub> was not significantly different but group means were higher for Caf vs. Pl (Table 2) corresponding with higher  $P_{peak}$  following caffeine ingestion indicating subjective feelings reflected increased work. Caf vs. Pl RPE in UW<sub>ant</sub> 4 was not significant (Caf: 5.8 ± 1.3, Pl: 5.4 ± 0.8) (p = 0.16). Caf vs. Pl UW<sub>ant</sub> S-RPE was not significantly different (Caf: 6.1 ± 1.4, Pl: 5.98 ± 1.1), although performance was greater following Caf ingestion. Collectively, perceptual responses considered concurrent with performance between trials suggests caffeine may blunt perceived exertion and pain response allowing for increased volume of work.

Currently there is limited data on caffeine's ergogenic potential in upper body ergometry. Recent investigation by Aedma et al (1) impact of caffeine supplementation (5mg/kg) utilizing repeated upper body ergometry efforts (4 x 6 min, 30 min

recovery) in attempt simulate match day for wrestlers. Caffeine negatively impacted performance in upper body ergometry (p= 0.05) but not in the placebo trial. Conversely, we found caffeine improved performance in upper body ergometry. However, this could be attributed to the shorter duration (15 s vs. 6 min) of exercise bouts in the current study. Beck et al. (4) examined acute caffeine supplementation on trained individuals between multiple muscle groups in traditional weight lifting exercises (leg extension and bench press) and repeated (2) W<sub>ant</sub>, observing improved performance for upper body, but not lower body. Green et al. (18) found caffeine significantly increased reps to failure during leg press in the third (but not first two) of three sets, but in none of three sets for bench press. Hudson et al. (22) tested trained individuals on upper body (arm curls) and lower body (leg extensions) resistance exercise. Leg extension and arm curl reps to failure significantly improved (attributed to first two of four sets to failure). Current results were similar to Beck et al. (4) in that caffeine's ergogenic properties were limited to upper body exercise and not lower body. Hudson et al. (22) also observed improved performance for upper body exercise that was limited to the first two sets (and not the later sets). However, Green et al. (18) contradict the current study, where significant improvement was observed in the early sets (1<sup>st</sup> and 2<sup>nd</sup>), but not in the later UW<sub>ant</sub> possibly because the intense fatigue-inducing nature of UW<sub>ant</sub>. Additionally, mean RPE for UW<sub>ant</sub> approached significance (p = 0.16) systematically increasing with the increased volume of work performed. Though no significant difference was observed for UW<sub>ant</sub> Caf vs.

Pl, similar peak HR responses ( $158 \pm 16$  b/min vs.  $158 \pm 11$  b/min) with greater power output during Caf may offer evidence of caffeine's ergogenic properties.

The equivocal nature of the literature regarding upper vs. lower body performance and caffeine is difficult to explain; however, muscle volume recruited may be a key factor. Svenningsson et al. (35) found increased motor unit activity in rats via antagonism of adenosine receptors at doses of caffeine of 7.5 mg/kg. Assuming greater muscle volume contains a greater absolute volume of adenosine receptors, it is plausible that individuals with a higher overall volume of muscle mass may potentially respond more positively to caffeine supplementation, although direct evidence is lacking. Given the "trained" (vs. sedentary) status of current participants, the expectation for improved performance would be plausible based on the previous statement. However, various performance tasks may differ following acute caffeine supplementation based on muscle volume involved in testing which is often dictated by testing mode. The results of the current study potentially support the previously stated theory given the improved performance for upper body, similar to Beck et al. (4), but not lower body, contradicting the results of Green et al. (18) and Hudson et al. (22) (see figure 1, 2, 3). Though the results of the studies are contradictory regarding muscle body regions that experienced performance differences; all three studies utilized trained individuals and observed a positive effect from caffeine in at least one body region.

Greater absolute adenosine receptor volume would be anticipated with greater

muscle volume. However, it is also reasonable that highly trained individuals may also be more likely to experience improvement if caffeine's ergogenic benefit is rooted in adenosine receptor antagonism. Because untrained muscle lacks neural adaptation (vs. trained muscle), a lower percentage and absolute number of total fibers may be activated during work. Consequently, there is less opportunity an ergogenic benefit to manifest through adenosine receptor antagonism. This theory has not been directly investigated yet could partially explain observations of improvement in trained and not in untrained participants. Further investigation is needed to clarify the potential role of muscle volume and training status with regard to caffeine, in particular, mechanistic factors.

Current results for  $LW_{ant}$  support Collomp et al. (7), Williams et al. (37), and Greer et al. (19) that Caf had no effect on  $LW_{ant}$  performance. Williams et al. (37) observed caffeine having no effect on mean power in 15 s maximal effort cycling. Greer et al. (19) utilized a similar Caf dose (6 mg/kg) and found peak and mean performance did not improve on the first 3 of 4 consecutive  $LW_{ant}$  but did increase on the final bout. However, it is important to note the participants in Greer et al (19) were unaccustomed to intense exercise. Plausibly no effect was observed because the utilization of untrained individuals prevented potential benefits from caffeine supplementation. Similarly, testing of non-habitual users creates the potential for caffeine-induced nausea which may impair performance at 6 mg/kg of caffeine (21). However the current study included trained individuals while finding a

significant difference between Pl vs. Caf  $P_{\text{mean}} LW_{\text{ant}}$  (Figure 4). Additionally, RPE in the 6<sup>th</sup>  $W_{\text{ant}}$  was significantly higher ( $p = 0.02$ , Caf:  $7.6 \pm 0.8$  vs. Pl:  $6.9 \pm 1.0$ ). Astorino et al. (3) found similar RPE and pain levels during leg extensions (2 sets of 40) Caf (5mg/kg) vs. Pl demonstrating a lack of pain blunting effect of caffeine on RPE similar to the current study. It is unclear why RPE was higher given the lack of differences in performance in the current study.  $LW_{\text{ant}}$  always followed  $UW_{\text{ant}}$  bouts and significantly greater performance was observed for Caf trials during  $UW_{\text{ant}}$  (Figure 2). While remote, it is possible that, because  $LW_{\text{ant}}$  followed the upper body bouts, RPE was higher during Caf because of greater “pre” fatigue resulting from greater work volume during Caf  $UW_{\text{ant}}$  (Table 2). Interesting to note,  $LW_{\text{ant}}$  PRS was significantly higher for Pl vs. Caf ( $5.68 \pm 1.9$  vs.  $5.00 \pm 2.2$ ). Greater PRS demonstrated participant’s subjective feelings of greater recovery and preparedness for ensuing bouts during Pl. This potentially supports the concept that lack of a significant difference for  $LW_{\text{ant}}$  may be attributed to residual fatigue from  $UW_{\text{ant}}$ . A negative reaction to high caffeine dosage can arguably be eliminated as no adverse reactions were observed or reported by participants.

Similar to Bugyi et al. (6), the current study showed no significant difference in mean hand grip performance between Caf vs. Pl trials. However, unlike the current study, caffeine dosage was not based on participant’s body mass in Bugyi et al. (6). Basing dosage on participant body mass plausibly ensured that critical levels of caffeine to induce potential ergogenic effects were reached in those who would

respond positively to caffeine administration. Our mode (repeated maximal static efforts) could negate any potential ergogenic response given the small volume of muscle utilized during hand grip testing. If a critical level of muscle volume is necessary to observe an ergogenic effect from caffeine, detection of improved performance would be difficult in the current (handgrip testing) paradigm. No analgesic effect of caffeine was observed in HG MPP (Table 2) in the current study contradicting Bellar et al. (5) who found pain perception was lowered by caffeine in hand grip to exhaustion Caf even when group means for time to exhaustion were higher Caf. However, the contradiction in perceptual ratings potentially could be attributed to mode of testing (15 s vs. time to exhaustion). Greater exercise duration (~100 s vs. a standardized 15 s) would have the potential to generate greater pain levels creating a situation in which caffeine, as an analgesic, might have greater potential to function. Lack of differences in HG MPP, with similar power, supports the notion that pain perception is influenced by volume of muscle performing work (i.e. relationship between muscle volume and volume of work performed influence on pain perception) and in this paradigm caffeine failed to influence MPP.

During high intensity exercise, caffeine has demonstrated analgesic properties and consistent blunting effect on perceptual responses (10, 30). Although no difference was observed in power levels, RPE approached significance during Pl  $LW_{\text{ant}}$  ( $p = 0.11$ ), failing to provide strong support for caffeine’s hypoalgesic effects of a lower RPE at the same workload (Caf:  $3.42 \pm 1.50$  vs. Pl:  $3.27 \pm 1.71$ ). It is plausible that



caffeine buffers pain as similar or increased work is performed (caffeine vs. placebo trials); yet perceptual responses are blunted or unaltered respectively (3, 18, 22, 34). Furthermore, current results show perceptual pain measures systematically increased with increased muscle volume utilized (Table 2). Additionally, reduction in reported levels of pain may be dose dependent due to individual variability required to reach critical levels (32). Killen et al. (27) observed significantly lower Session-RPE for Caf vs. Pl ( $6.1 \pm 2.2$  vs.  $6.8 \pm 2.1$ ) following 30 min of sub-max cycling equated for intensity and total work volume. In the current study, S-RPE was not significantly different for any exercise, nor was T-RPE significantly different for Caf. However, group means were higher for all 3 exercises Caf vs. Pl RPE (Table 2). The contradictory results between studies may potentially be explained by mode of exercise (aerobic vs. anaerobic). As stated earlier,  $LW_{ant}$  PRS was significantly higher for Pl (vs. Caf), but no significant difference was observed for PRS in  $UW_{ant}$  or HG; however, group means were higher for all exercises during Pl (Table 2). This indicates the high caffeine dosage may have impacted some participants negatively given the subjective measures show participants anticipated performing better on the ensuing bout (when not on caffeine). Laurent et al. (28) demonstrated a correlation between expected performance using the PRS scale and actual performance for the ensuing exercise bout. In that study recovery (PRS) was estimated prior to an entire exercise bout following variable days of recovery. In the current study, PRS was taken repeatedly between bouts essentially reflecting participant's perceived readiness for the next bout. Results were inconclusive

given participants performed better during the  $UW_{ant}$  Caf trial but PRS group means were higher during Pl;  $LW_{ant}$   $P_{mean}$  was higher during Pl and correlated with a higher PRS, while HG max performance was not statistically different nor was PRS (though group mean was higher during Pl). PRS responses and performance in the current study was potentially disrupted by the acute caffeine supplementation and the way in which the PRS scaled was applied. The PRS scale was intended to assess recovery status prior to exercise in a global manner and has not been validated to function as a readiness scale between acute repeated bouts. Even so, it is conceivable that the subjective scale (PRS) lacked sensitivity to detect the small differences in performance in the current design. Further investigation is needed to determine how caffeine ingestion may alter perceptual feelings regarding recovery status.

Because there appears to be inter-individual variability regarding caffeine's' ergogenic potential, conclusions based solely on analysis of mean data may be misleading. It is therefore important to consider individual responses. In the current study 7/10 individuals consumed caffeine daily at rates  $> 1.8\text{mg/kg}$ . Of these 7 habitual users, 6 showed mean improvement following Caf (vs Pl) ingestion for  $P_{eak}$  for  $UW_{ant}$  ( $0.61\text{W/kg}$ ) and  $LW_{ant}$   $0.44\text{ W/kg}$ ), while the single non-responder (Pl  $>$  Caf performance) possessed the highest BF % in the study and produced the lowest W/kg  $UW_{ant}$ ,  $P_{mean}$  and  $LW_{ant}$ ,  $P_{mean}$  in the current study (supporting earlier speculation based on Svenningsson et al. (35)). Although, aggregate data show no significant difference in  $LW_{ant}$   $P_{peak}$ , individual responders to acute caffeine

supplementation improved  $LW_{ant}$   $P_{peak}$  between 0.22-1.34 W/kg (~ 21-131 Watts). Additionally, the responders improved  $UW_{ant}$ ,  $P_{peak}$  (0.34-1.04 W/kg) or (~ 33-102 Watts). Comparatively the 3 designated non-responders demonstrated average values reflecting impaired performance for  $UW_{ant}$ ,  $P_{peak}$  (-0.07 W/kg or -6.65 Watts) and  $LW_{ant}$ ,  $P_{peak}$  (-0.14 W/kg or -13.3 Watts) Caf vs. Pl. Woolf et al. (39) tested collegiate football players after ingestion of 5 mg/kg on 40-yard sprint, 20-yard shuttle test, and bench press reps to failure, showing no significant difference between placebo and caffeine trials when comparing group means. However, 59% of participants improved 40-yard sprint time; 59% improved 20-yard shuttle time, and 47% improved bench press reps to failure. Similar to current results, Woolf's study emphasizes that it is imperative to examine 'responders vs. non-responders' (i.e. individual data) to provide an in-depth evaluation regarding effects of caffeine on anaerobic exercise. Similarly, Jordan et al. (25) examined acute caffeine supplementation at 6 mg/kg on repeated sprints (12 x 30 m) between caffeine naïve (<50 mg per day) and habitual (>300 mg per day) college age males and females. No significant differences were found between Caf consumers and non-consumers in performance. However fastest individual sprint time for all participants followed caffeine consumption. Additionally, mean sprint times for the caffeine trial were faster compared to mean sprint times for the placebo trial and mean RPE was higher during the caffeine trial compared to the placebo trial ( $13.9 \pm 1.5$  vs.  $13.3 \pm 1.6$ ). While significant differences in sprint times were not identified based on common values used to restrict type I error rate, it is

emphasized that seemingly minor differences in anaerobic athletic competitions can be of great practical importance.

More research is required to definitively determine the effect of caffeine on intense anaerobic exercise. One flaw of acute caffeine supplementation research is a clear consensus on what constitutes a habitual caffeine user.. Future studies should define habitual users using daily intake relative to body mass (mg/kg) rather than daily absolute caffeine ingestion. Caffeine should be administered during a multitude of activities in dosages at or above levels previously shown to enhance performance. The ability to mask caffeine supplementation is difficult, but 6 out of 10 participants incorrectly guessed which treatment they received first and was considered but a direct analysis was omitted. However, one constant that should remain is the utilization of anaerobically trained individuals. In Davis' (9) review, of 18 studies showing an ergogenic effect of caffeine, 15 utilized trained individuals, 2 untrained and 1 did not provide training background. Further investigations should focus on more practical approaches reflecting athletic competitions and training, utilizing a strength-to-weight ratio. It is also critical to assess individual responses rather than drawing conclusions based solely on analysis of aggregate data.

The current study demonstrated caffeine's ergogenic properties in certain paradigms (upper body wingates during early sets) and failure to influence performance in others (lower body wingates and static hand grips). Plausibly, caffeine's ergogenic properties may be limited to trained

individuals targeting appropriate muscle mass (mode and set dependent) following ingestion of a required critical dosage. Given a large dose of caffeine, acute negative side effects are plausible which could hinder performance. Additionally, research should not focus only on performance variables; instead continue to investigate perceptual responses to caffeine during exercise. Admittedly, one confounder of caffeine research is the dynamic variability in methodology making it problematic to compare findings to identify a definitive, global answer regarding caffeine's potential impact on exercise bouts dominated by anaerobic metabolic ATP production. Therefore research should continue to focus on the responder vs. non-responder concept in attempts to identify the parameters that create a responder to caffeine's ergogenic properties.

## REFERENCES

1. Aedma M, Timpmann S, Ööpik V. Effect of caffeine on upper-body anaerobic performance in wrestlers in simulated competition-day conditions. *Int J Sport Nutr Exerc Metab* 23(6):601-9, 2013.
2. Anselm F, Collomp K, Mercier B. Caffeine increases maximal anaerobic power and blood lactate concentration. *Eur J Appl Physiol* 65: 188-191, 1992.
3. Astorino TA, Terzi MN, Roberson DW, Burnett TR. Effect of caffeine intake on pain perception during high intensity exercise. *Int J Sport Nutr Met* 21: 27-32, 2011.
4. Beck TW, Housh TJ, Schmidt RJ. The acute effects of caffeine-containing supplement on strength, muscular endurance, and anaerobic capabilities. *J Strength Con Res* 20(3): 506-510, 2006.
5. Bellar D, Kamimori GH, Glickman EL. The effects of low-dose caffeine on perceived pain during a grip to exhaustion task. *J Strength Cond Res* 25(5): 1225-1228, 2011.
6. Bugyi GJ. The effects of moderate doses of caffeine on fatigue parameters of the forearm flexor muscles. *Am Corr Ther J* 34: 49-53, 1980.
7. Collomp K, Ahmaidi M, Audran M. Effects of caffeine ingestion on performance and anaerobic metabolism during the Wingate test. *Int J Sports Med* 12: 439-443, 1991.
8. Cook BC, O'Connor PJ, Oliver SE, Lee Y. Sex differences in naturally occurring leg muscle pain and exertion during maximal cycle ergometry. *Int J Nuero Sci* 95(34): 183-202, 1998.
9. Davis JK, Green, JM. Caffeine and anaerobic performance: ergogenic value and mechanisms of action. *Sports Med* 39 (10): 813-832, 2009.
10. Doherty M, Smith PM, Hughes MG, Davison RC. Caffeine lowers perceptual response and increases power output during high-intensity cycling. *J Sport Sci* 22(7): 637-643, 2004.
11. Dotan R, Bar-Or. Load optimization for the wingate anaerobic test. *Eur J Appl Physiol* 51:409-417, 1983.
12. Ellender L, Linder MM. Sports pharmacology and ergogenic aids. *Clinics Office Care* 32: 277-292, 2005.
13. Essig D, Costill DL, Van Handel PJ. Effects of caffeine ingestion on utilization of muscle glycogen and lipid during leg ergometer cycling. *Int J Sports Med* 1: 86-90, 1980.
14. Goldstein ER, Ziegenfuss T, Kalman D. International society of sports nutrition position stand: caffeine and performance. *J Inter Society Sports Nutr* 7(5): 1-15, 2010.
15. Graham TE, Spreit LL. Metabolic, catecholamine, and exercise performance responses to various doses of caffeine. *J Appl Physiol* 78(3): 867-874, 1995.
16. Graham TE, Hibbert E, Sathasivam P. Metabolic and exercise endurance effects of coffee and caffeine ingestion. *J Appl Physiol* 85(3): 773-774, 1998.

## CAFFEINE AND UPPER BODY WINGATE PERFORMANCE

17. Graham TE. Caffeine and exercise: metabolism, endurance and performance. *Sports Med* 31(11): 785-807, 2001.
18. Green JM, Wickwire JP, McLester JM. Effects of caffeine on repetitions to failure and ratings of perceived exertion during resistance training. *Int J Sport Phys Per* 2: 250-259, 2007.
19. Greer F, McLean C, Graham TE. Caffeine, performance, and metabolism during repeated Wingate exercise tests. *J Appl Physiol* 85(4): 1199-1200, 1998.
20. Greer F, Morales J, Coyles M. Wingate performance and surface EMG frequency variables are not affected by caffeine ingestion. *Appl Physiol Nutr Metab* 31: 597-603, 2006.
21. Hetzler RK, Warhaftig GN, Thompson DL. Effects of acute caffeine withdrawal on habituated male runners. *J Appl Physiol* 76: 1043-1048, 1994.
22. Hudson GM, Green JM. Effects of caffeine and aspirin on resistance training performance, RPE and pain perception. *Med Sci Sports Exerc* 39: S298, 2007.
23. Inbar O, Bar-Or O, Skinner JS. The wingate anaerobic test. Champaign, Illinois, 1996.
24. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. *Br J Nutr* 40(3): 497-504, 1978.
25. Jordan BJ, Farley RS, Caputo JL. Caffeine and sprint performance in habitual and caffeine naïve participants. *Int J Exerc Sci* 5(1): 50-59, 2012.
26. Kang H, Kim H, Kim B. Acute effects of caffeine intake on maximal anaerobic power during the 30s wingate cycling test. *J Exer Physiol Online* 1(3), 1998.
27. Killen LG, McIntosh JR, Green JM, Hornsby J, O'Neal EK, Coates TE. Effects of caffeine on session ratings of perceived exertion. *Eur J Appl Physiol* 113:721-727, 2013.
28. Laurent CM, Green JM, Bishop PA. A practical approach to monitoring recovery: development of a perceived recovery status scale. *J Strength Cond Res* 25(3): 620-628, 2011.
29. Magkos F, Kavouras S. Use of caffeine in sports, pharmacokinetics in man, and cellular mechanisms of action. *Food Sci Nutr* 45(7): 535-562, 2005.
30. Motl RW, O'Conner PJ, Dishman RK. Effect of caffeine on perceptions of leg muscle pain during moderate intensity cycling exercise. *J Pain*, 4: 316-321, 2003.
31. National Collegiate Athletic Association. NCAA banned-drug classes 2012-13. [www.ncaa.org](http://www.ncaa.org). Accessed January 21, 2013
32. O'Conner PJ, Motl RW, Broglio SP. Dose-dependent effect of caffeine on reducing leg muscle pain during cycling exercise is unrelated to systolic blood pressure. *J Pain* 109: 291-298, 2004.
33. Richardson NJ, Rogers PJ, Elliman NA, O'Dell RJ. Mood and performance effects of caffeine in relation to acute and chronic caffeine deprivation. *Pharmacol Biochem Behav* 52: 313-320, 1995.
34. Schneiker KT, Bishop D, Dawson B. Effects of caffeine on prolonged intermittent sprint ability in team sport athletes. *Med Sci Sports Exerc* 38: 578-585, 2006.
35. Svenningsson P, Nomikos GG, Ongini E, Fredholm BB. Antagonism of adenosine A<sub>2A</sub> receptors underlies the behavioral activating effect of caffeine and is associated with reduced expression of messenger rna for ngfi-a and ngfi-b in caudate-putman and nucleus accumbens. *NeuroSci* 79(3): 753-764, 1997.
36. Utter AC, Kang J, Nieman DC, Dumke CL, McAnulty SR. Validation of omni scale of perceived exertion during prolonged cycling. *Med Sci Sports Exerc* 38(4): 780-786, 2006.
37. Williams JH, Signoile JF, Barnes WS. Caffeine, maximal power output and fatigue. *British Journal of Sports Med* 22: 132-134, 1988.
38. Whaley MH. American College of Sports Medicine, Guidelines for Exercise Testing and Prescription (8th ed.). Baltimore: Williams and Wilkins, 2009.

## CAFFEINE AND UPPER BODY WINGATE PERFORMANCE

39. Woolf K, Bidwell WK, Carlson AG. The effect of caffeine as an ergogenic aid in anaerobic exercise. *Inter J Sport Nutr Exerc Metabol* 18(4): 412-429, 2008.