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Acute Caffeine Ingestion's Increase of Voluntarily Chosen Resistance-Training Load After Limited Sleep

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Introduction: This study aimed to determine whether caffeine ingestion would increase the workload voluntarily chosen by athletes in a limited-sleep state. ***Methods:*** In a double-blind, crossover study, 16 professional rugby players ingested either a placebo or 4 mg/kg caffeine 1 hr before exercise. Athletes classified themselves into nondeprived (8 hr+) or sleep-deprived states (6 hr or less). Exercise comprised 4 sets of bench press, squats, and bent rows at 85% 1-repetition maximum. Athletes were asked to perform as many repetitions on each set as possible without failure. Saliva was collected before administration of placebo or caffeine and again before and immediately after exercise and assayed for testosterone and cortisol. ***Results:*** Sleep deprivation produced a very large decrease in total load ($p = 1.98 \times 10^{-7}$). Caffeine ingestion in the nondeprived state resulted in a moderate increase in total load, with a larger effect in the sleep-deprived state, resulting in total load similar to those observed in the nondeprived placebo condition. Eight of the 16 athletes were identified as caffeine responders. Baseline testosterone was higher ($p < .05$) and cortisol trended lower in non-sleep-deprived athletes. Changes in hormones from predose to preexercise correlated to individual workload responses to caffeine. Testosterone response to exercise increased with caffeine compared with placebo, as did cortisol response. ***Conclusions:*** Caffeine increased voluntary workload in professional athletes, even more so under conditions of self-reported limited sleep. Caffeine may prove worthwhile when athletes are tired, especially in those identified as responders.

Keywords: sleep deprivation, sleep poverty, testosterone, cortisol, individual responses

Caffeine has long been touted as an ergogenic aid. With the lifting of the partial ban on its use by the International Olympic Committee in 2004, research on its effects in relation to athletic performance has intensified. Indeed, caffeine at appropriate doses appears able to improve time to exhaustion and other indices of endurance in a number of different physical activities including rowing, swimming, cycling, and running and simulated rugby and soccer performances (Carr, Gore, & Dawson, 2011; Davis & Green, 2009; Paton, Lowe, & Irvine, 2010; Stuart, Hopkins, Cook, & Cairns, 2005). Furthermore, a position stand by the International Society of Sports Nutrition has declared that the positive effects of caffeine supplementation are not associated with negative effects on fluid balance that could negatively affect performance (Goldstein et al., 2010).

In terms of the effects of caffeine on strength performance, the existing literature is equivocal. It has been suggested that caffeine can enhance contractile force at submaximal loads (Tarnopolsky, 2008), and Jacobson, Weber, Claypool, and Hunt (1992) observed that a dose of 7 mg/kg body weight of caffeine had a beneficial effect on muscle strength. However, the same benefits were not seen in a similar study design by Bond, Gresham, McRae, and Tearney (1986) using a 5-mg/kg body weight dose. One study (Beck et al., 2006) suggested that upper body strength is enhanced by caffeine consumption with no effect on lower body strength, while others have seen the opposite (Astorino, Martin, Schachtsiek, Wong, & Ng, 2011) or have failed to see any evidence for strength increases (Williams, Cribb, Cooke, & Hayes, 2008). Astorino et al. (2011) concluded that caffeine had a limited practical effect before workouts but conceded that this was highly individual. Many fundamental resistance-training programs progress by adding total load via increased repetitions to set weights before increasing the actual weight load itself (Kraemer & Ratamess, 2005), so motivation to perform additional repetitions, as observed on the leg press by Astorino et al. (2011) after caffeine ingestion, may in fact be an important component in progressive training gain that has received little focus.

Mild sleep deprivation is common across all aspects of modern society (Bixler, 2009) and can affect physical performance and hormonal responses (Mougin et al., 2001; Remes, Kuoppasalmi, & Adlercreutz, 1985; Vgontzas et al., 2004). Indeed, increasing nightly sleep hours by approximately 2 hr from 8 to 10 hr of sleep has

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been shown to improve physical-performance attributes in college athletes (Mah, Mah, Kezirian, & Dement, 2011). Caffeine is commonly used to ameliorate the effects of limited sleep, and caffeine consumption has been associated with enhancements in mood, psychomotor performance, and vigilance (Dawkins, Shahzad, Ahmed, & Edmonds, 2011; McLellan et al., 2005). In terms of improving sporting performance, caffeine has produced clear benefits in cognitive tasks during exercise (Hogervorst et al., 2008) and sport-specific skills in team-sport players (Foskett, Ali, & Gant, 2009; Stuart et al., 2005). Furthermore, our research team has reported that caffeine improved passing skills in rugby during a competitive test scenario while the athletes were sleep deprived (Cook, Crewther, Kilduff, Drawer, & Gaviglio, 2011).

Caffeine ingestion also appears to be associated with increases in free testosterone and cortisol concentrations that may be important in mediating the adaptive benefits of resistance exercise. In professional athletes, caffeine elicited small but significant acute increases in salivary testosterone across a workout accompanied by larger increases in the cortisol response in a dose-responsive manner (Beaven et al., 2008). Similar changes in these hormones were seen across the combined physical and mental stress of a simulated rugby skill (Cook et al., 2011). In contrast, caffeinated chewing gum led to a substantial increase in free testosterone not accompanied by any change in cortisol in competitive cyclists (Paton et al., 2010).

The main aims of the current study, then, were to observe whether caffeine ingestion influenced the voluntary choice of workload and, if so, whether this effect was exaggerated after self-reported poor sleep duration. A secondary aim was to observe whether free concentrations of the hormones testosterone and cortisol varied across these states.

Methods

Participants

Sixteen professional rugby players (age 20.9 ± 0.9 years, height 1.85 ± 0.06 m, and body mass 97 ± 8 kg; *M* ± *SD*) were recruited to participate in the study. The athletes chosen were similar in strength level, with maximum lifts of 170- to 210-kg squat, 130- to 170-kg bench press, and 110- to 130-kg barbell row. All participants were fully informed of the nature and possible risks of the study before giving written consent, and the protocol was approved by the university ethics committee.

Experimental Protocol

All athletes completed a one-repetition maximum (1-RM) test in the week preceding the start of the experiment for back squat, bench press, and bent barbell row, and these data were used to extrapolate each individual's 85% 1-RM loading used in the four test sessions. Briefly, the 1-RM test session consisted of 5 × 50%, 3 × 60%, 2 × 70%, 1 × 80%, 1 × 90%, 1 × 95%, and then 1 × 100% of each individual's previous best effort. If athletes failed, they had one more attempt, and if they still failed, the weight was reduced by 2.5 kg until their best lift was obtained. If they were successful at their 100% lift, the weight was increased by 2.5 kg until two failed attempts indicated they had reached their maximum weight. Three minutes were allowed between efforts.

The study used a randomized, double-blind, placebo-controlled, balanced trial. Each athlete was asked to log his nightly sleep time and quality and to turn up for testing on two occasions when he had obtained 8 hr or more of good sleep and on two occasions when he had slept for less than 6 hr (sleep-deprived state), under the proviso that any two testing sessions be a minimum of 3 days apart. On arrival, the athletes ingested gelatin capsules containing either lactose (placebo) or 4 mg/kg body weight of caffeine 1 hr before exercise. Thus, all participants completed four test sessions over a 4-week period with caffeine or placebo ingested before exercise in both a non-deprived and a sleep-deprived state. They also completed a short questionnaire at the conclusion of training to ascertain their perception of what they had ingested and whether they thought it had had an effect on their training.

All participants were instructed to refrain from ingesting dietary caffeine before exercise on the test days. A dietary log for the preceding 24 hr was collected to assess caffeine, fluid, and food intake, and reminders were given to ensure dietary compliance. The athletes were by their own admission light, occasional caffeine consumers, and this was verified by food diaries that were kept for 2 weeks before the study. The diaries indicated that consumption of caffeine was equal to or less than 120 mg caffeine per day, with most participants not consuming caffeine on a daily basis.

Test Sessions

All exercises were performed at the gymnasium where the participants were accustomed to training. All participants were completely familiar with the training protocol and had undertaken similar sessions over the last 12 months. They all had a minimum of 2 years of fully recorded and instructed resistance training. Resistance-training sessions were performed at 11 a.m., with athletes reporting to the gym at 9:30 a.m. after consuming breakfast and at least 750 ml of fluid. Water was available ad libitum across the sessions.

The athletes performed a warm-up as follows: 5-min warm-up on a stationary cycle, unloaded, at 60 rpm and a nonstop circuit of one set of squats using a 20-kg bar for 10 repetitions (reps), one set of bench press using a 20-kg for 10 reps, one set of barbell rows using a 20-kg bar for 10 reps, and one set of squats, bench press, and barbell row with a 60-kg weight for 10 reps (each with 30 s rest). The subjects then rested for 1 min and began training.

Training consisted of four sets of back squat using 85% of their individually defined 1-RM, four sets of bench press at 85% 1-RM, and four sets of bent row at 85% 1-RM. Athletes rested for 90 s between sets and 3 min between exercises. They were accompanied by a trainer during the workout who verbally encouraged them to perform as many repetitions on each set as they felt they could without failure. No assistance was given, and any failed repetitions were not counted. All sets were attempted. The workload for each exercise was calculated as the product of total repetitions and load lifted. The sum of these workloads was defined as the total workload.

Saliva Samples

Whole saliva samples were obtained from each athlete immediately before caffeine or placebo ingestion, then again before starting and at the end of the resistance-exercise session. For each sample, participants were asked to expectorate 2 ml of saliva into a sterile container. Saliva samples were stored at –20 °C until assay. Salivary steroid samples were taken in this study because they are minimally invasive and have the advantage of reflecting free-steroid concentrations, which are reported to be more physiologically relevant than total blood levels (Obminski & Stupnicki, 1997; Vining, McGinley, & Symons, 1983). To prevent blood contamination of saliva, resulting in an overestimation of hormone concentrations, subjects were advised to avoid brushing their teeth, drinking hot fluids, or eating hard foods such as apples in the 2 hr before providing their sample. Saliva samples were analyzed in duplicate for testosterone and cortisol using ELISA kits per manufacturer's instructions (Salimetrics Ltd.). Detection limits for the assays were 0.1 pg/ml and 0.01 ng/ml for testosterone and cortisol, respectively. The intra- and interassay coefficients of variation were <9% for cortisol and <8% for testosterone.

Statistical Analysis

Changes in the mean of each measure with and without caffeine treatment were used to assess magnitudes of effects by dividing the changes by the appropriate between-participants *SD*s. Pairwise *t*-statistic comparisons were made between conditions, and differences were interpreted in relation to the likelihood of exceeding the smallest worthwhile effect with individual change thresholds for each variable. Hormonal and load data were log-transformed to reduce nonuniformity of error, with effects derived by back-transformation as percentage changes. Magnitudes of the standardized effects were interpreted using thresholds of 0.2, 0.6, and 1.2 for small, moderate, and large, respectively (Hopkins, Marshall, Batterham, & Hanin, 2009). Standardized effects of –0.19 to 0.19 were termed trivial. To make inferences about the large-sample value of an effect, the uncertainty in the effect was expressed as 90% confidence limits. An effect was deemed unclear if the confidence interval overlapped the thresholds for both small positive and negative effects. The significance level was set at $p \leq .05$.

Results

None of the athletes were able to perceive beyond chance whether they had actually received the placebo (which they had been told was another supplement) or the 4-mg/kg body weight caffeine dose. Caffeine administration in the non-sleep-deprived state was associated with substantial and significant increases in bench press (effect size [ES] 0.75, $p = .039$), back squat (ES 0.93, $p = .0122$), bent row (ES 0.81, $p = .0249$), and total workload (ES 1.13, $p = .003$) compared with the non-sleep-deprived placebo condition (Figure 1). Caffeine administration in the sleep-deprived state also produced significant increases in bench press (ES 1.16, $p = .0023$), back squat (ES 1.39, $p = .0004$), bent row (ES 1.07, $p = .0043$), and total workload (ES 1.47, $p = .0002$) compared with placebo sleep-deprived state. With caffeine administration, the total workload completed in the sleep-deprived state was not significantly different ($p = .6402$) than that seen in the non-sleep-deprived state of the placebo condition (Figure 1). Very large increases in the voluntarily chosen workload were observed between the non-sleep-deprived caffeine state and the sleep-deprived placebo state: bench press 2,568 vs. 1,780 kg ($p = 2.31 \times 10^{-5}$), squat 3,316 vs. 2,327 kg ($p = 2.20 \times 10^{-5}$), bent row 1,997 vs. 1,524 kg ($p = .0009$), and total workload 7,881 vs. 5,631 kg ($p = 1.05 \times 10^{-8}$). A lack of sleep was associated with significant decreases in total workload in both the placebo (ES 2.33, $p = 1.98 \times 10^{-7}$) and caffeine (ES 1.03, $p = .0058$) condition.

The group data were biased by large responses among 8 of the 16 athletes who were identified as high caffeine responders. Those participants consistently noted that they believed there was an effect when they were administered the caffeine dose. Total workload in the high caffeine responders was significantly higher than in nonresponders in the non-sleep-deprived (ES 3.46, $p = 8.78 \times 10^{-6}$) and sleep-deprived (ES 3.37, $p = 1.13 \times 10^{-5}$) conditions (Figure 2). Indeed, there was no difference in the voluntarily chosen workload of the nonresponders between the caffeine and placebo conditions ($p = .5329$). There was, however, a clear moderate positive effect of the 4-mg/kg body weight caffeine dose on the workload chosen by nonresponders in the fatigued state (ES 1.01, $p = .0533$).

Baseline testosterone was clearly elevated in the non-sleep-deprived groups compared with the groups that reported a lack of sleep (ES 0.67–1.09; Table 1). The 4-mg/kg body weight caffeine dose produced an increase in testosterone across the hour before the workout in the sleep-deprived (5.8% ± 3.3%, $p = .0083$) and non-sleep-deprived (3.4% ± 1.8%, $p = .005$) groups (Figure 3). In contrast, testosterone significantly decreased over the same period in both placebo groups ($p < .01$), with a small but clear difference between the change in the sleep-deprived (10.6% ± 3.6%, $p = .0001$) and non-sleep-deprived conditions (10.9% ± 2.4%, $p = 2.82 \times 10^{-8}$). The workout elicited significant increases in testosterone in each of the conditions, but the greatest increase was in the

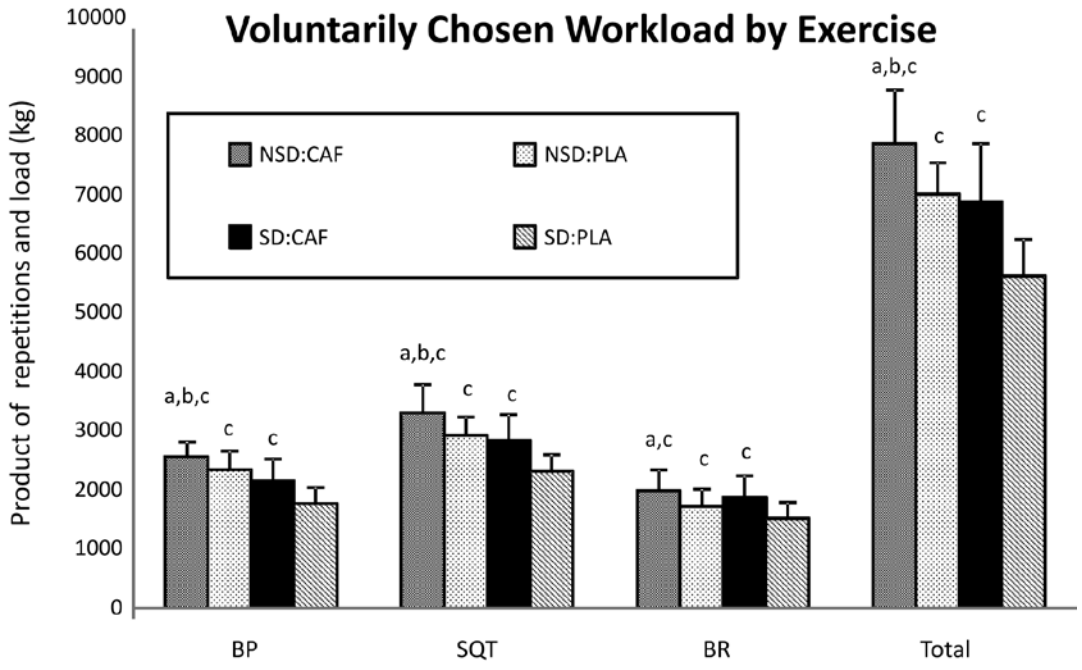

Figure 1 — Workload for each exercise performed, $M \pm SD$. NSD = non-sleep-deprived; SD = sleep-deprived; CAF = caffeine; PLA = placebo; BP = bench press; SQT = back squat; BR = bent row. [a]Significantly greater than NSD:PLA. [b]Significantly greater than SD:CAF. [c]Significantly greater than SD:PLA. The threshold for significance was $p \leq .05$. Workload is defined as the product of repetitions performed and load lifted.

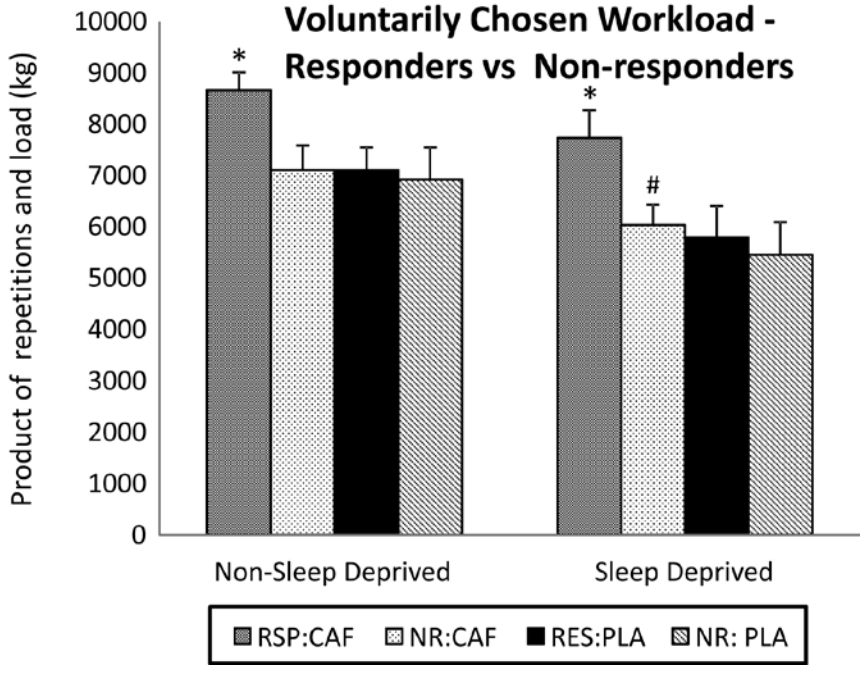

Figure 2 — Total workload for responders and nonresponders with placebo and caffeine administration, $M \pm SD$. RSP = caffeine responders ($n = 8$); NR = nonresponders ($n = 8$); CAF = caffeine; PLA = placebo. *Significantly greater than other conditions; #Clearly greater than corresponding placebo condition. Caffeine dose was 4 mg/kg body weight. The threshold for significance was $p \leq .01$.

Table 1 Group Salivary Hormone Concentrations Across Treatment Groups

	Predose		Preworkout		Postworkout	
	Testosterone	Cortisol	Testosterone	Cortisol	Testosterone	Cortisol
NSD:CAF	136 ± 22[b,c]	2.07 ± 0.21	140 ± 20[a,b,c]	2.23 ± 0.21	183 ± 26[a,b,c]	2.71 ± 0.26[a]
NSD:PLA	134 ± 31[b,c]	2.22 ± 0.51	125 ± 30[c]	2.13 ± 0.49	151 ± 32[c]	2.44 ± 0.49
SD:CAF	113 ± 21	2.44 ± 0.54[a,d]	120 ± 24	2.64 ± 0.46[a,c,d]	143 ± 29[c]	2.98 ± 0.50[a,c]
SD:PLA	115 ± 26	2.40 ± 0.53[d]	110 ± 25	2.30 ± 0.51	123 ± 29	2.60 ± 0.49

Note. NSD = non-sleep-deprived; CAF = caffeine; PLA = placebo; SD = sleep-deprived. Testosterone and cortisol concentrations are in pg/ml and ng/ml, respectively.

[a]Substantially greater than NSD:PLA. [b]Substantially greater than SD:CAF. [c]Substantially greater than SD:PLA. [d]Substantially greater than NSD:CAF.

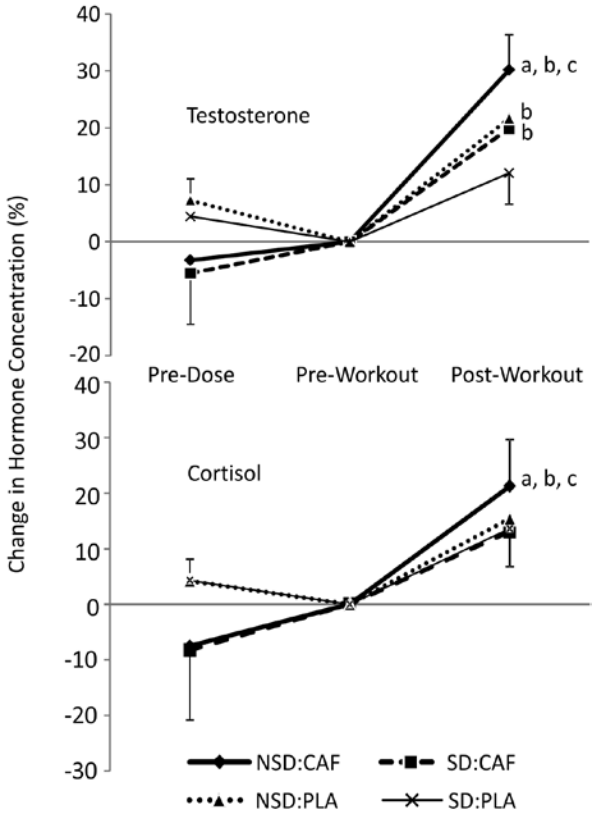

Figure 3 — Salivary hormone responses to caffeine ingestion and resistance exercise sessions, *M* ± *SD*. NSD = non-sleep-deprived; SD = sleep-deprived; CAF = caffeine; PLA = placebo. Caffeine dose was 4 mg/kg body weight. All preworkout testosterone and cortisol values are significantly different from the preceding predose value ($p \leq .01$). All postworkout testosterone and cortisol values are significantly greater than the preceding preworkout value ($p < .01$). [a]Substantially greater than corresponding sleep-deprived:caffeine value. [b]Substantially greater than corresponding sleep-deprived:placebo value. [c]Substantially greater than corresponding non-sleep-deprived:placebo value.

non-sleep-deprived caffeine condition (30.1% ± 6.2%, $p = 2.14 \times 10^{-11}$), which was greater than in any of the other conditions (p £ .0104). The poorest testosterone response was observed in the sleep-deprived placebo condition (Figure 3), which was significantly worse than in any of the other conditions (p £ .0035).

Baseline cortisol was clearly elevated in the sleep-deprived groups compared with the non-sleep-deprived groups (ES 0.41–0.80; Table 1). Caffeine administration produced an increase in cortisol across the hour before the workout in the sleep-deprived (9.0% ± 5.3%, $p = .0107$) and non-sleep-deprived (8.1% ± 2.5%, $p = .0001$) groups. In contrast, cortisol significantly decreased over the same period in both placebo groups ($p < .01$), with a small but clear difference between the change in the sleep-deprived (13.7% ± 5.6%, $p = .0007$) and non-sleep-deprived conditions (12.7% ± 2.7%, $p = 3.67 \times 10^{-7}$; Figure 3). Exercise elicited significant increases in cortisol in each of the conditions, with the greatest increase in the non-sleep-deprived caffeine condition (21.3% ± 8.4%, $p = 1.56 \times 10^{-7}$), which was greater than in any of the other conditions (p £ .0898).

It was apparent that the increase in testosterone across the hour before the workout was driven by the previously identified caffeine responders, with increases of 6.4% ± 1.6% ($p = .0002$) and 12.9% ± 2.8% ($p = .0001$) in the non-sleep-deprived and sleep-deprived conditions, respectively. The corresponding changes in testosterone for the nonresponders were a trivial increase of 0.4% ± 2.1% in the non-sleep-deprived group and a trivial decrease of 0.8% ± 1.7% in the sleep-deprived group. These changes were significantly less than those seen in the caffeine responders ($p = .0012$ and 1.19×10^{-5}, respectively). Furthermore, among the 8 individuals identified as caffeine responders, the change in testosterone from time of caffeine ingestion to immediately before exercise was strongly correlated with the subsequent increase in voluntarily chosen workload ($R^2 = .67$, $p = .0002$).

Discussion

The main finding in this study was that caffeine ingestion significantly improved the total voluntary workload lifted

by professional rugby athletes in a self-reported state of sleep poverty. There was a similar, although smaller, beneficial effect on total workload in a non-sleep-deprived state. As loads were kept constant throughout the testing, the increases in total workload were a direct result of an increase in the number of voluntarily selected repetitions in each set. It is important to state, then, that given the voluntary nature of the repetitions (not forced by coach or lifting partner), this is likely more about motivation to perform than an actual physical-performance increase. As training progression is often based around such increases, caffeine ingestion appears to be a valuable intervention when an athlete is tired. The results also demonstrate that it is important for athletes to avoid states of sleep poverty due to the negative physical and hormonal effects observed.

Previously published work has focused on acute improvements in maximal strength measures in non-sleep-deprived conditions, where a small effect of caffeine was observed (Astorino, Rohmann, & Firth, 2008; Beck et al., 2006). University of California researchers have reported that while caffeine did produce significant effects on repetitions performed on the leg press, it was of limited practical value to the training environment (Astorino et al., 2011). Part of their argument was based on the importance of progression in extrapolated 1-RM strength, which clearly is important, rather than maintenance of repetitions performed across sequential sets. However, herein we focus more on a higher maintenance of repetitions per set at a standard weight, which we suggest is a valid training gain. Indeed, the ingestion of caffeine in the non-sleep-deprived state was associated with an increase in total workload of 2,250 kg in a single session compared with the sleep-deprived placebo condition. This voluntary increase in workload represents a superior adaptive stimulus as a result of caffeine ingestion without an increase in maximal load.

To a large extent our data agree with the findings of Astorino et al. (2011), as we found moderate effects of caffeine ingestion on repetitions performed while non-sleep-deprived, with the largest effect observable on squats rather than bench press or bent row. After sleep deprivation, however, we saw larger effects of caffeine ingestion, potentially offering more practical significance to athletes when programs progress based on added repetitions, especially given that sleep deprivation is not uncommon in many athletic groups. Indeed, high training loads have been associated with sleep disruption in competitive swimmers (Taylor, Rogers, & Driver, 1997).

It should be noted that ours results demonstrated large variability, and individual responses in caffeine susceptibility were apparent. Indeed, some athletes showed clear changes in voluntary load volume with caffeine ingestion compared with placebo, while others were not affected at all. These data were used to identify the high caffeine responders in the current study along with their associated distinctive hormonal profiles. Astorino et al. (2011) also clearly reported an individual responsiveness, with heavier caffeine consumers tending to respond positively to caffeine ingestion. A number of studies now suggest that heavy caffeine consumers are more responsive to caffeine ingestion and that this may in fact simply represent amelioration of caffeine-withdrawal effects induced by the restrictions on caffeine intake that studies demand before testing (James & Keane, 2007; Yeomans, Ripley, Davies, Rusted, & Rogers, 2002). We intentionally chose low consumers of caffeine to avoid any potential confounding effects of withdrawal reversal. We have previously suggested that in low caffeine consumers a much lower dose of caffeine may be more ergogenic than a higher dose (Cook et al., 2011).

In both placebo groups (sleep-deprived and non-sleep-deprived) salivary testosterone and cortisol tended ($p < .08$) to decline from predose to immediately before exercise. This observation is consistent with other data showing a circadian fall across the morning hours (Bird & Tarpenning, 2004; Gachon, Nagoshi, Brown, Ripperger, & Schibler, 2004). However, the expected circadian decrease was absent in the caffeine groups, with notable increases in individuals identified as caffeine responders. The observation that the caffeine-induced increase in testosterone was specific to the athletes who responded positively in terms of increased workload volume suggests that the dose of caffeine sufficient to elicit meaningful training-volume improvements is related to changes in preworkout hormones. Alternatively, our research group has demonstrated a role of testosterone in training gains that may relate to motivation to perform (Cook et al., 2011; Crewther & Cook, 2010). The current study provides some support for this hypothesis, which may be worthy of further exploration in a competitive environment where caffeine may have more significant effects.

Caffeine ingestion was also associated with significant increases in free testosterone and cortisol after the workout compared with the placebo condition. This phenomenon does not appear to have been well studied. However, in professional rugby league players we also saw a small but significant increase in both the testosterone and the cortisol response across the workout after caffeine ingestion (Beaven, Hopkins, et al., 2008). One other study has reported similar, but larger, effects in testosterone but did not observe a marked change in cortisol (Paton et al., 2010). That study in competitive cyclists employed a chewing-gum delivery method, which may account for the differences in hormone response observed. It is also possible that the differential hormonal responses across the workout are a simple reflection of the increased workload, facilitated through other mechanisms by caffeine, as hormone change is known to be influenced by intensity of workout (Kraemer et al., 1990; Kraemer & Ratamess, 2005).

The actual outcome of this caffeine-augmented increase in hormones across the workout remains equivocal. Indeed, it has been suggested that typical changes in testosterone concentration are unlikely to have any real anabolic effect (West et al., 2010). In contrast, others have suggested that testosterone can both directly and indirectly influence muscle and strength gains (Beaven,

Cook, & Gill, 2008; Kvorning, Anderson, Brixen, & Madsen, 2006; Rønnestad, Nygaard, & Raastad, 2011). Furthermore, the importance of a functional androgen receptor has been demonstrated (Inoue, Yamasaki, Fushiki, Okada, & Sugimoto, 1994), suggesting at least a permissive role for testosterone in muscle adaptation. In addition, testosterone has been demonstrated to be dose responsive in priming for a workout, with short-term effects on the neuromuscular system that have the potential to mediate adaptation (Crewther, Cook, Cardinale, Weatherby, & Lowe, 2011).

In conclusion, we found significant positive effects of caffeine ingestion on voluntary workload volume in a resistance-exercise session performed by professional rugby players. We found large beneficial effects of caffeine in athletes who reported a poor sleep duration that could have practical implications in certain responsive individuals. In these individuals, salivary testosterone and cortisol were influenced by caffeine and may in part be linked to the increase in voluntary workload.

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