THE SCIENCE BEHIND





Xplode shot: helps physical and mental performance

Key Points:

- Caffeine is unquestionably a potent ergogenic aid for high intensity and prolonged exercise bouts.
- Caffeine stimulates mental performance too in particular alertness, promoting improved reaction time, and attenuating RPE.
- The dose of caffeine needs to be 2-6mg/kg body mass for efficacy. More is not better!
- Caffeine should be taken between 30-60 minutes before exercise.
- The effect of caffeine lasts for many hours after ingestion.
- Caffeine can be taken with carbohydrate for beneficial effects on prolonged exercise activities.
- Caffeine can be used to 'fat burn' when ingested in the absence of carbohydrate using trained (not sedentary) persons.
- Xplode shots contain 200mg caffeine which is enough for most individuals since the dose will fall into the 2-6mg/kg range. In order to achieve 2-6mg/kg then taking 1-2 shots of Xplode should be considered. We do not advise taking more than 2 shots at a time.
- Xplode shots should be taken 30-45 minutes before the activity bout. If taken prior to a match, the period just before warm-up is ideal.
- For greater efficacy, athletes should not take any caffeine-containing foods in the 24-h before Xplode shot ingestion (i.e. no coffee, chocolate, green tea etc). This means the body is likely to be more sensitive to the caffeine.
- Xplode shots can be used first thing in the morning before a good 'fat burning' training session but NO carbs please (at least not until after the training bout).



Introduction

The key component in **Xplode** shots is caffeine, although other compounds such as taurine, arginine, tyrosine, and vitamin B6 are also evident. There is research evidence concerning the efficacy of these other ingredients on physical and mental performance, and as such they will be briefly explored in a section at the end of this article. However, the major focus of this article concerns caffeine.

Caffeine

Caffeine is probably the most common drug ingested, with coffee being the main source. It is a mild stimulant that occurs naturally in a number of plant species. Significant amounts of caffeine can be found in coffee, tea, chocolate and soft drinks such as Red Bull and Coca-Cola, although it also occurs in other products such as prescription medications, diuretics, and pain relievers. Because caffeine is a drug and yet is part of a normal diet, the IOC had placed a limit on the amount that could be consumed before exceeding 'doping' limits. However, since January 2004 caffeine was removed from the banned list, although the IOC continue to monitor it's use. Probably the reason for lifting the ban was the fact that the ergogenic effects of caffeine occur when ingested in doses of 2-6mg/kg body mass; an amount which results in urine values of caffeine lower than the previous IOC limit. There has been a wealth of information on studies reporting the positive effects of caffeine ingestion both for endurance and high intensity exercise. The ability of caffeine to stimulate the central nervous system (CNS) is another important feature of its ingestion. The effect of caffeine on the cerebral cortex results in a clearer thought process, a reduced rating of perceived exertion (RPE), and an attenuation of fatigue. The net effect is an enhanced ability to concentrate, thereby aiding athletes competing in sports where quick thinking and rapid reactions are necessary. Since caffeine stimulates the release of fatty acids from adipose tissue, there is a potential to promote 'fat burning'. This article provides an overview of some of the reported effects of caffeine on performance and fat oxidation.

Mechanism of action

Caffeine may act in a number of different ways depending on whether the activity is intense or prolonged. With regard to intense exercise bouts, caffeine has been considered to stimulate the release of calcium ions and so enhance muscle force. With regard to prolonged exercise, caffeine is known to stimulate the release of fatty acids from adipose tissue so that muscles are capable of using them for energy at an earlier time than would be normally expected. This results in sparing the limited stores of muscle glycogen and so fatigue is offset. Furthermore, it is well established that caffeine stimulates the CNS and enables subjects to perceive the exercise as being easier and maintains arousal and mental alertness – this is mediated via the fact that caffeine inhibits adenosine uptake by cells and thereby induces a state of alertness rather than lethargy. Whatever



the mechanism for enhancement of performance, it is clear that caffeine is a positive ergogenic aid for performance.

Timing and dose

Most studies which have reported the positive effects of caffeine ingestion have given a dose of at least 2-6 mg per kg body weight (2-6mg/kg) an hour before exercise. For a 70 kg person this amounts to a dose of 140-420 mg. Caffeine is rapidly absorbed by the intestine, and peak concentrations in blood are seen at approximately 60 minutes after ingestion (See Figure 1). Evidence from many laboratory-based studies has shown no advantages of taking doses greater than 6 mg/kg body weight. More is not better in this regard.



Figure 1. Blood caffeine concentrations 60 minutes after ingestion of varying doses of caffeine and throughout subsequent exercise (after Passman et al., 1995).



Form of caffeine

Table 1 shows that caffeine is present in various drinks and foods. Drinking 3 paper cups (approximately 150 ml each) of dripped coffee will ensure nearly 350 mg of caffeine is ingested. This is equivalent to 5 mg/kg body weight for a 70 kg person. Caffeine is also present in significant amounts in various analgesics such as exedrin and anacin, and of course, caffeine can be purchased in chemists as 'ProPlus' tablets (50 mg per tablet).

Product	Serving	Caffeine
Coffee - instant	150 ml	60 mg
dripped	150 ml	115 mg
espresso	150 ml	100 mg
Tea - black teabag	150 ml	40 mg
loose leaf	150 ml	30 mg
Milk chocolate	50 mg	35 mg
Hot chocolate	250 ml	11 mg
Chocolate cake	1 'slice'	25 mg
Coca cola	330 ml	45 mg
Pepsi	330 ml	35 mg
Red Bull	250 ml	80 mg
Xplode shot	60ml	200mg

Table 1. Caffeine content of various foods and drinks.

Pure caffeine is the form normally ingested in successful laboratory investigations as opposed to trials with decaffeinated v caffeinated coffee. Generally, coffee has been equated with caffeine, although in some recent studies this has proved contentious. For example (Figure 2) a dose of 4.5 mg/kg body weight of caffeine was administered either as caffeine in a capsule or in coffee. Only the pure form of caffeine produced a positive effect by enhancing time to exhaustion (41 minutes vs 32 minutes) at 85% VO_{2max}. There were no differences between the coffee and the placebo treatments or indeed the decaffeinated coffee with added caffeine. For some reason coffee does not always produce the same ergogenic effect as pure caffeine. On the other hand drinking Red Bull or Coca



Cola or a caffeine-containing drink (such as Xplode) or eating some dark chocolate can result in positive ergogenic effects.



Figure 2. Effect of caffeine or coffee on time to fatigue at 85% VO_{2max} when provided as decaffeinated coffee (DC), decaffeinated coffee with added caffeine (DC+C), regular coffee (RC), placebo (PI), and caffeine (CAF).

Caffeine and endurance exercise

A trawl of over 40 published studies using caffeine and endurance exercise over the last 25 years has demonstrated the positive effects on both time to fatigue and in time trials. Improvements with caffeine have varied between 2 and 44%, with an average of 20% being achieved – as a rule of thumb the longer the event the greater the % improvement. The overall conclusion is that caffeine ingestion promotes prolonged activities when using either recreational or competitive athletes.

The proposed mechanism by which caffeine is purported to positively affect prolonged activity is via stimulating the adrenal gland to secrete adrenalin (epinephrine to those in N. America), which in turn targets adipose tissue to release fatty acids into the blood. The resulting increase in fatty acids can be taken up by muscle and used as a source of energy during the exercise bout and so preserve the limited muscle glycogen stores (Figure 3).





Figure 3. Theoretical model proposed to show how caffeine may positively affect prolonged performance (after Costill et al., 1978).

For example, in a study we undertook in the 1990's we provided 6mg/kg body mass of caffeine to university football players who then underwent a simulated football match lasting 90 minutes using the Loughborough Intermittent Shuttle test (Figure 4). The average sprint times in each Block of activity was significantly faster following caffeine ingestion from Blocks 3-5 i.e. end of 1st half and into the 2nd half. Furthermore, the time to fatigue after Block 5 (i.e. after 75 minutes) was significantly longer following caffeine ingestion.

Figure 4. Effects of caffeine ingestion on average Sprint Time in a Block and on Time to Exhaustion. (Unpublished data from MacLaren)

In a study on cycling to fatigue at 70% VO_{2max} , the efficacy of caffeine ingestion can clearly be recognised (Figure 5). In fact this study illustrates another finding in so far as doses above 5-6mg/kg body mass of caffeine are NOT more effective. More is not better!





Figure 5. Effects of caffeine dose on Time to Exhaustion during cycling exercise. All caffeine trials enhanced the time significantly although there were no differences between caffeine trials. (Passman et al., 1995).

Numerous studies on prolonged exercise activities have used carbohydrate co-ingested with caffeine. In all cases the results have proved positive. To illustrate with one example, eight rugby union players underwent a rugby orientated shuttle running protocol involving four 21-minute blocks of activity having ingested either a placebo, a carbohydrate, or a carbohydrate + caffeine (Roberts et al., 2010). The 15-m sprint times were faster in the carb+caf trials; the motor skills were performed more quickly with carb+ caf; and the RPE was lower in the carb+caf.

Caffeine and high intensity exercise

Although fewer studies have been undertaken in this field, a few recent studies have demonstrated the beneficial effects of caffeine on high intensity exercise. Such activities have invariably resulted in fatigue between 1 and 7 minutes and include cycling, treadmill sprinting, and 2km rowing. Events in which fatigue is reached in less than 1 minute are equivocal, although repeated bouts of sprinting are likely to benefit.

Figure 6 highlights two key findings regularly shown in research on caffeine and performance – i.e. caffeine significantly improves prolonged exercise and also that doses greater than 5-6mg/kg body mass are NOT more useful. In this investigation the participants rowed 2-km after placebo or caffeine (6 or 9mg/kg) or placebo ingestion. The caffeine dose of 6mg/kg produced the fastest times followed by the 9mg/kg dose – both were superior to the placebo.



Figure 6. Positive effects of caffeine ingestion on 2-km rowing performance (after Bruce et al., 2000)

In another study using treadmill running at $120 \text{ %VO}_{2\text{max}}$, participants ingested 5mg/kg caffeine or placebo 60 minutes prior to the run. Figure 7 shows that of the 9 participants, eight ran for longer (participant 7 was better on the placebo) and the average improvement in time to exhaustion was 208.2-s compared with 181-s after placebo – a 27-s enhancement.



Figure 7. Differences in time to exhaustion after caffeine ingestion when compared with placebo – values above the line show improvements in TTe whilst below the line show a detriment in performance.

Finally, in a study which used cycling as the mode of exercise, participants had to undertake four 2min bouts of cyling at a power output corresponding to their maximal aerobic capacity followed by an all-out 1-min power sprint. Figure 8 demonstrates that caffeine ingestion resulted in a significantly higher power output (794 watts) for the power sprint compared to placebo (750 watts).





Figure 8. Power output during 4 bouts of cycling at maximal aerobic capacity followed by an all-out power sprint for 1-min (after Doherty et al., 2004).

The findings clearly show that caffeine can have a significant effect on high intensity bouts of activity. If this is the case, then the proposed mechanism of stimulating fatty acid release and use is unlikely to be important here. The proposed mechanism for caffeine enhancing high intensity performance can be seen in Figure 9. Two key factors may be involved – (a) caffeine stimulates greater release of Calcium (Ca²⁺) ions from the sarcoplasmic reticulum and so leads to greater generation of force by the muscle, and (b) caffeine affects the brain to increase alertness, enhance motor recruitment of muscle fibres, and also to reduce the perception of effort (i.e. RPE is reduced).







Caffeine and fat oxidation

As previously mentioned, ingestion of caffeine results in a significant elevation of blood fatty acid levels, and this occurs within 60 minutes after ingestion (Figure 10). The reason for this has also been presented i.e. caffeine stimulation of adrenalin release and the effect of adrenalin in stimulating adipose tissue lipolysis to release fatty acids normally bound as triglycerides within the cells (see Figure 3)..



Figure 10. Fatty acid concentrations following caffeine and placebo ingestions at rest and during exercise (after Giles & MacLaren, 1984).

It is possible that this elevation of fatty acids from adipose tissue results in greater oxidation of fats during subsequent exercise. In our study (Giles & MacLaren , 1984) we observed such by measuring the respiratory exchange ratio (RER) during exercise. The RER reflects the amount of fat and/or carbohydrate oxidised – values close to 0.7 reflect greater fat oxidation and those close to 1.0 reflect carbohydrate oxidation. Figure 11 shows that following caffeine ingestion the RER is significantly lower at the start of exercise and throughout exercise when compared with placebo.

So it appears that caffeine may boost fat oxidation (fat burning) when ingested before an exercise bout. Two sentences of caution however – (a) do not take caffeine with any carbohydrate since the carbohydrate negates the efficacy of caffeine, and (b) sedentary individuals are unlikely to benefit from the fat burning effect of caffeine whereas trained persons are likely to do so (sedentary persons will stimulate the release of fatty acids from fat cells somewhat but are not capable of oxidising the fatty acids sufficiently).





Figure 11. RER during 120-min of running at 65% VO2max after caffeine or placebo ingestion (after Giles and MacLaren, 1984).

Caffeine and mental function

The ergogenic effect of caffeine can also be mediated by its effect on the central nervous system (CNS). Adenosine is a potent adenosine antagonist (See Figure 12), and is a CNS stimulant which can easily cross the blood brain barrier (Davis et al., 2003). Caffeine has been shown to counteract the inhibitory effects of adenosine neuro-excitability, neurotransmitter release, arousal, and spontaneous activity. In fact, the role of caffeine as an adenosine inhibitor is the main reason for its efficacy as a CNS stimulant (Spriet, 2014). Figure 12 illustrates that caffeine competes with adenosine for the same binding site on the cell membrane and so reduces the uptake of adenosine into the cell. Remember that adenosine once inside a cell (such as a brain cell) causes an inhibition of cellular processes linked with stimulation and thereby results in 'our little mouse' feeling lethargic. When caffeine is present, there is a greater chance that it attaches to the adenosine receptor (so blocking adenosine from attaching) and results in increased alertness/activation.



Figure 12. Schema illustrating the effect of caffeine on adenosine uptake by a cell.

One of the ways mental processes can be assessed during exercise is by using the Borg scale of rating of perceived exertion (RPE). Almost without exception, all studies in which RPE has been assessed when comparing caffeine with a placebo demonstrate an attenuated response (Doherty & Smith, 2005). Just to illustrate the point Figure 13, taken from a study by Giles & MacLaren (1984), shows consistently lower RPE scores for trained runners who ran on a treadmill at 65%VO_{2max} for 2-h when given 5mg/kg body mass of caffeine 1-h before exercise. These findings are not unusual when comparing caffeine with a placebo. In other words, caffeine ingestion invariably results in the exercise/work seeming easier compared with placebo.





Figure 13. Rating of Perceived Exertion (RPE) during a 2-h run on a treadmill when given caffeine or placebo (after Giles & MacLaren, 1984).

Miscellaneous considerations

Caffeine is a diuretic and as such may lead to increases in urine production, which could have a negative effect on performance due to dehydration. However, most studies have found no effect of caffeine ingestion on either urine volume or plasma volume. This is quite possibly due to the impact of exercise on urine production overriding the stimulus of caffeine. Another consideration is that caffeine is a mental stimulant and so when taken at night will lead to insomnia and a disturbed sleep. This is not advisable if a match is played on the following day.

Conclusion

Caffeine is classified as a drug, yet it is a normal part of many individuals dietary intake. The ergogenic properties of caffeine are best realised when ingesting the pure form in a capsule or in drinks such as **Xplode** or Red Bull. As such, improvements in endurance and high intensity performance can be realised by ingesting a dose of 2-6 mg/kg body weight 60 minutes before the activity. If taking Xplode shots then 1-2 shots would provide caffeine in the range of 2-6mg/kg caffeine for most individuals. The diuretic effects of caffeine are not realised in exercise, although possible gastrointestinal problems may occur in some individuals. Caffeine may also be employed to aid 'fat burning' in trained individuals.

Caffeine has been shown to be an unquestionable useful ergogenic aid for performance ranging from high intensity bouts to prolonged activities – and not just physically but also mental performance too. Figure 14 provides an overall synopsis for the likely modes of action of caffeine on performance.





Figure 14. Likely mechanisms of action of caffeine on performance.

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Other Ingredients

Caffeine is the most important of the ingredients in Xplode since it has (by far) the greatest amount of positive research findings as to its efficacy for performance. However, the addition of the other compounds is due to the fact that there is (on balance) sufficient evidence of their effectiveness for performance – either physical or in most cases mental. The addition of these 'other ingredients' compliments that of caffeine and (in our view) are useful from an ergogenic perspective. Some examples of the efficacy of these 'other ingredients' is now highlighted.

Arginine-AKG

l-arginine is a "semi-essential" amino acid used by all cells. It plays an important role in cytoplasmic and nuclear protein synthesis, the synthesis of other amino acids, creatine synthesis, and the urea cycle. Arginine is classified as a glucogenic amino acid because it can be metabolized into α ketoglutarate (AKG) and enter the citric acid cycle. In addition, arginine has been reported to improve the immune response and increase the release of growth hormone and insulin. In one of its most important roles, arginine serves as a precursor for the biosynthesis of nitric oxide (NO), an endogenously produced, cellular signaling molecule that is involved in a variety of effects in the vasculature. NO serves as a second messenger to trigger blood vessel dilation and increase blood flow. Research has indicated that exogenously administered arginine promotes NO-mediated biological effects. Studies in animals have reported that acute and long-term administration of arginine improves blood flow and vascular health. In humans, studies have reported beneficial effects after oral arginine supplementation including improved blood flow, a reduction in blood pressure, and improved immune function. These outcomes would potentially be beneficial for athletes undergoing heavy resistance training.



Bailey et al. (2010) undertook a study employing arginine acutely before exercise. They observed that arginine supplementation resulted in significantly elevated NO concentration, reduced systolic blood pressure, a 7% reduction in oxygen uptake during a steady state bout of exercise, and that time to exhaustion was significantly increased from 562-s after placebo to 707-s after arginine ingestion.

 α -Ketoglutarate (AKG) is a five-carbon dicarboxylic acid produced in the citric acid cycle has been reported to modulate protein tissue metabolism in rats with burn injury and muscle-wasting conditions. Theoretically, if oral AKG supplementation influences protein metabolism or catabolism, then it may help athletes undergoing intense training, increase muscle mass, and/or promote positive training adaptations.

Campbell et al. (2006) reported on a study that employed 35 resistance-trained men who were randomly assigned to ingest arginine AKG three times a day or placebo. Participants performed 4 days of resistance training per week for 8 wk. At 0, 4, and 8 wk of supplementation the following tests were performed: clinical blood markers, 1-RM bench press, an isokinetic quadriceps muscle endurance test, Wingate anaerobic power test, aerobic capacity, total body water, body composition, and psychometric parameters tests. Results showed significant differences in the A-AKG group for 1RM bench press, Wingate peak power, blood glucose, and plasma arginine. No significant differences were observed between groups in body composition, total body water, isokinetic quadriceps muscle endurance, or aerobic capacity.

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Taurine

Taurine is a conditionally essential amino acid. It is the most abundant free amino acid in brain, heart, and skeletal muscle (Rutherford et al., 2010), and is suggested to be involved in cell volume regulation, to play a role in the regulation of intracellular free calcium concentration (specifically Ca²⁺- dependent excitation-contraction processes for optimal force development), and as an antioxidant defense from stress responses. Taurine serves a wide variety of functions in the central nervous system, from development to cytoprotection, and is considered by some to be a nerotransmitter (Ripps & Shen, 2012). Because it is one of the few amino acids not used in protein synthesis, taurine is often referred to as a "non-essential" amino acid, or as a "conditionally essential" amino acid.

Oxidative stress plays a major role in a broad range of human diseases. The overproduction of reactive oxygen species and the body's inability to halt the accumulation of free radicals have been implicated in cardiovascular disease, inflammatory disease, and several of the major disorders of the CNS. In each case, taurine, because of its antioxidant activity, has been shown to play a crucial role



as a cell protectant. There is a growing consensus that oxidative stress is linked to mitochondrial dysfunction, and that the beneficial effects of taurine are a result of its antioxidant properties, as well as its ability to improve mitochondrial function by stabilizing the electron transport chain and inhibiting the generation of reactive oxygen species.

Taurine is thought to counteract the harmful effects of oxidative agents, such as free radicals produced during oxidative metabolism, which may cause extensive DNA damage and subsequent cellular death. Taurine may attenuate exercise induced DNA damage (Zhang et al., 2004), and in fact, taurine may enhance the capacity of exercise due to these cellular protective properties (Zhang et al., 2004).

Due to its aknowledged role in modulating the intracellular free calcium concentration, taurine has been shown to increase force production in skeletal muscle and a fall in taurine to decrease force output (Hamilton et al., 2006). The contractile properties and fatigability of extensor digitorum longus (EDL) muscles depleted of taurine (by chemical means) were found to decrease peak twitch force by 23% compared with controls; the force–frequency relationship resulted in significantly less force produced by muscles low in taurine, and also EDL muscles with lowered taurine exhibited significantly slower rates of recovery. Similar findings reported by the same authors found that a fall in taurine decreased force output and increased the endurance of EDL skeletal muscles.

Rutherford et al.(2010) examined whether acute taurine ingestion before prolonged cycling would improve time-trial performance and alter whole-body fuel utilization compared with a control trial and a placebo trial in which participants were told they received taurine but did not. Eleven endurance-trained male cyclists completed 3 trials in which they consumed a noncaloric sweetened drink with either 1.66 g of taurine or nothing added (control and placebo) 1 hr before exercise. Participants then cycled at 65% VO2max for 90 min followed immediately by a time trial (doing 5 kJ of work/kg body mass as fast as possible). There was no difference in time trial performance between the 3 trials, but taurine ingestion resulted in a 16% increase in total fat oxidation over the 90-min exercise period compared with control and placebo.

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β-alanine

Refer to the article on β -alanine on the web.



N-acetyl tyrosine

Tyrosine is a large, nonessential, neutral amino acid present in both animal and vegetable protein. Produced in the liver and to some extent the brain, L- tyrosine is synthesized via phenylalanine, an essential amino acid. Furthermore, L- tyrosine is the precursor of the catecholamine neurotransmitters dopamine, noradrenaline, and adrenaline. High levels of catecholamines, especially noradrenaline, in the blood are elicited by some form of physiological, psychological, and/ or environmental stressors. Depletion of noradrenaline has the potential to impair/ compromise cognitive and physical performance. Interestingly, when tyrosine is systemically administered in pharmacologic amounts before acute exposure to stressful events, it has been shown to increase concentrations and release of catecholamines. Human studies have shown ingestion of such doses is associated with neurochemical, behavioural, and cognitive changes. Notably, tyrosine supplementation has been shown to diminish cognitive and some behavioural deficits associated with stressful conditions. In addition, the administration of tyrosine has been shown to improve performance on stress-sensitive attention tasks.

In a meta-analysis, Attipoe et al. (2015) examined 10 randomized controlled trials and 4 controlled clinical trials. On the basis of the available evidence, they found a weak recommendation in favour of tyrosine for cognitive stress since all studies examined showed a positive effect.

Coull et al. (2015) explored the effect of tyrosine ingestion on cognitive and physical performance during soccer-specific exercise in a warm environment. Their results showed that tyrosine was associated with improved vigilance and reaction time. This suggests that increasing the availability of tyrosine may improve cognitive function during exposure to exercise-heat stress.

There is little doubt that intense or prolonged exercise affects brain chemistry. Therefore, supplements which can possibly maintain a favourable brain chemistry balance could be useful for athletes. It would seem from these results that tyrosine supplementation could limit increases in the fatigue-inducing 5-HT neurotransmitter, keeping both physical energy and mental faculties on top form. The army has been known to use tyrosine as a supplement during hard physical missions, and other research exists to support its use in humans to help combat stress and boost performance. In general, tyrosine should be seen as a stimulatory amino acid supplement.

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