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NUTRITIONAL ERGOGENIC AIDS IN THE HORSE - USES AND ABUSES

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What is an Ergogenic Aid?

If one imagines two horses with identical genetic makeup with equal physiological ability, ridden by jockeys with equal riding skill and with the same way of riding a race, being raced against each other, all things being equal the two horses should finish at exactly the same time. But what if one of the horses were given 'formula X' to improve its performance, enabling it to win outright. 'Formula X,' in this case, is an ergogenic aid. The term ergogenic comes from the Greek 'ergon' meaning work and 'genic' meaning producing. An ergogenic aid therefore can be used to describe any factor which can increase or improve work production. This could result in an increase in speed or endurance or strength.

A number of factors could have caused the improved performance in our example and have been shown to be important in man, including:

1. Psychological factors
2. Mechanical or biomechanical factors including improved equipment (shoes, track design, etc.)
3. Pharmacological agents
4. Physiological improvements (including most importantly those obtained through training)
5. Nutritional supplements.

The key to optimal sport performance has been said to be the proper production and control of energy. To this end the appropriate biomechanical, psychological and physiological training specific to the nature of the athletic event will improve the control and utilization of the various energy systems and maximize energy efficiency and production. The adaptations that occur in the body cells, tissues and organs in response to chronic exercise training are fairly specific to the imposed demands and are fairly well documented, at least in man.

In man, nutritional ergogenic aids have been suggested to be far less efficient, in general, in improving physical ability than an appropriate training regimen. This is likely to be true also for the horse. However, athletes training at levels close to their upper limit require relatively large increases in training effort in order to achieve even a small increase in performance. Very small variations in performance, whether achieved through significant increases in training effort or by addition of 'formula X' among elite athletes could make the difference between finishing first or in the middle of the pack. The comparative ease of taking 'formula X' compared with the significant increase in training effort

required explains why there has been such a drive for effective nutritional ergogenic aids.

Possible ways that ergogenic aids, apart from equipment, etc., could improve performance in the horse could include (see also Harris, 1994):

1. Psychological effects
2. Improved coordination or recruitment of muscle fibers
3. Provision of a supplementary fuel source or the feeding of a feed with a higher energy content
4. Increased levels of available stored energy
5. Improved efficiency of conversion of the chemical energy of the feed, or stored energy, to mechanical energy for work
6. Improved ATP/ADP homeostasis in contracting muscle fibers
7. Decreased substrate depletion
8. Decreased end product accumulation including improved intracellular acid base regulation.

These could result in increased mechanical energy for work and/or a delayed onset of fatigue or improved neuromuscular coordination. Many substances have theoretical ergogenic properties.

Nutritional ergogenic aids may be considered to be compounds or elements that can be administered orally and have a nutritionally oriented function. This paper will concentrate on these postulated nutritional ergogenic aids, but it will not consider pharmaceuticals such as steroids or other agents that may fundamentally affect underlying metabolic and structural elements within the muscle fibres.

How Could One Design the Ideal Nutritional Ergogenic Aid?

There are three basic steps to producing an ideal nutritional ergogenic aid.

1. Identify an aspect of exercise physiology that is limiting to competition (of a particular type).
2. Identify a nutritional compound or element that will positively affect this aspect.
3. Finally and most importantly, provide evidence that the feeding or administration of the compound or element is effective in the field (as well as the laboratory); this will necessarily include evidence in the target species of (Harris, 1994):
    - absorption from the gut
    - uptake into the target tissue
    - demonstrably improved tissue function
    - improvement in performance.

Unfortunately too many substances are marketed without adequate understanding of their function, little or no evidence that the metabolic or physiological

mechanism which they contribute to is limiting, and no evidence that they will affect performance in the field. The outcome may be a short term financial advantage for the manufacturer but a disappointed rider/owner and on occasions an unhappy horse. Often a compound's efficacy is based on anecdotal evidence, owner hopes or expectations, and results based on work in other species. On the other hand potential benefits should not be dismissed out of hand simply because of the difficulties in demonstrating an effect on performance. This may prevent justification of the compound or element on scientific grounds.

Why Are There Difficulties in Confirming the Efficacy of Ergogenic Aids?

There are a large number of potential reasons why there have been difficulties in confirming the efficacy of a variety of ergogenic aids. Some of these are outlined below:

1. Studies in horses are very expensive so tend to use only small numbers of animals.
2. Individual variability in response means that it may be difficult to assess scientific significance if small numbers are used. If only one out of five horses shows a marked improvement this may not show up as a statistical effect in studies, but nonetheless could be highly significant to the individual (responding) athlete in the field.
3. The effect on performance may be so small as to be masked by the normal within-subject variability intrinsic within any testing program and the sensitivity of the procedures employed, but in competition could make the difference between winning or being placed.
4. Alternatively, laboratory studies could show statistically significant effect of a compound or element on some physiological or biochemical function but which in the field is not manifest in any significant improvement in performance.
5. Extrapolation from other species is often used but not always appropriate, e.g. creatine is not well absorbed in the horse compared with the dog or man.
6. Dose response effects need to be taken into account.
7. Study design can be difficult especially regarding:
   - randomization
   - handlers/assessors/riders/drivers etc. being blind to treatment
   - pattern of running over the test distance varying between test periods.
8. How do we assess performance?
9. How representative is the treadmill to the racetrack or show arena?
10. What may be suitable for a sprint race horse may be contraindicated in the endurance horse.

Results of differing studies are often contradictory and in practice there are very few credible studies available.

What About the Ethics Behind Ergogenic Aids?

In our three step example one could have added a fourth step: that the product is not banned as an illegal substance. For this we may have to establish:

1. What is allowable?
2. What should be allowable?
3. When is an ergogenic aid an advance in optimal feeding and therefore legal and acceptable?
4. When is an ergogenic aid a prohibited substance?

The IOC (International Olympic Committee) doping legislation stipulates that any physiologic substance taken in abnormal quantities with the intention of artificially and unfairly increasing performance should be construed as doping, violating the ethics of sport performance.

But what if an ergogenic aid would help for example with the welfare of horses undergoing prolonged endurance events? Should it be allowable even if it improves performance? If one takes this to its logical conclusion then the feeding of water and electrolytes to horses in endurance rides could be considered use of ergogenic aids!

What about creatine? Will there be questions over the legality of its use in horses if, in the future, a way of improving absorption and utilization is found? Creatine is not a normal component of horse diets, especially not at the levels likely to be required (although the horse synthesizes it within its own body), but then neither is fat in large amounts a normal component of horse diets. What if feeding creatine just enables the horse to be restored to its potential capacity as may arguably be the case in man?

Dietary Manipulation and the Feeding of Supplemental Fat

The horse evolved as a grazing animal which escaped predators by flight and was adapted to an almost constant supply of forage, which was predominantly digested in the hindgut. Today the horse might be expected to carry a rider and undertake fairly exhaustive repetitive work. The horse therefore often needs more energy than it would have required in the wild. The gross (chemical) energy (GE) of the feed is decreased by the energy lost with ingestion, foraging, chewing and then fermenting the feed (often included in the heat of maintenance) and via losses in the feces. The amount left is the digestible energy (DE) content of the diet. Potential energy is also lost via gas production from the fermentation processes and via urea in the urine. The conversion of the residual chemical energy, or metabolizable energy, to mechanical energy of movement is substantially less than 100%, with most energy being lost as heat. Work efficiency or Kw is only 20 - 25%. The amount of net energy available from the diet depends obviously on the feed given; for example corn has more net energy than barley and oats. Barley has around twice that of good hay which in turn is over twice that of wheat straw. Note: that barley in Grecian times was referred to as aiding performance

by providing additional energy over and above forage. Is this the first ergogenic aid?

The relative energy contents for a kg of three different types of diet based on the partitioning system of Kronfeld (1996) are shown below. Traditionally energy in horse feeds is depicted as digestible energy, but the DE system tends to overestimate the energy potential of a high fiber feed compared with a highly hydrolyzable carbohydrate feed, as fiber predominantly produces volatile fatty acids, which are not as efficiently used as glucose (Harris, 1997).

Relative energy contents (MJ/kg) for three diets:

	Hay only	**Hay:Oats (50:50)**	**Hay:Oats:Fat (45:45:10)**
GE	15.6	16.3	18.5
DE	8	9.8	12
ME	7.5	9.3	11.6
NEm[1]	5.4	7.1	9.3

[1]NEm = Net energy for maintenance (The net energy will be considerably lower for work.)

Consider a 500 kg three day eventer on the cross-country day that needs approximately 60 MJ of net energy to live and compete. If it is assumed that all this energy is to be supplied by its daily ration, a horse would need to eat around 22 kg of hay which would not be possible (appetite being restricted to around 10 - 12 kg DM). This explains why, even though animals vary in their energy efficiency, it would be difficult if not impossible to maintain weight and energy output in regularly competing horses fed only on hay. If oats were substituted as part of the diet then the ration would be more realistic, but it would still be unlikely that the horse could eat all provided. However, if energy dense fat was added to the diet then it could be possible to match this energy requirement with intake (Kronfeld, 1996).

Feeding Fat:

1. reduces the amount of concentrates that need to be fed to maintain energy intake, which effectively means that often a horse can retain a healthy fiber intake despite a high energy requirement. May have behavioral advantages.
2. increases the energy density of a feed so that a horse can effectively take in more energy even if appetite decreases.
3. decreases heat load as more efficient in conversion to mechanical energy than fiber or carbohydrate. Useful under hot and humid conditions.
4. potentially helps reduce bowel ballast and possibly water requirements.

Feeding fat supplemented diets to horses has resulted in a range of effects on a variety of physiological and metabolic parameters as well as on performance. These variations may result from the variances in the study protocols and horses

used in these trials. Because of the variable results, a consensus view of the benefits on performance beyond those listed above is not yet available. Long term fat supplementation in combination with appropriate training, however, has been suggested to result in the following adaptations which could result in improved performance (see also Harris, 1997; Potter et al., 1992):

1. increased mobilization of free fatty acids (FFA) and increased speed of mobilization.
2. increased speed of uptake of FFA into muscle (Orme et al., 1997) - often considered to be rate limiting.
3. a glycogen sparing effect so that fatigue is delayed and performance improved - could be especially important in endurance activities.
4. increased high intensity exercise capacity (Eaton et al., 1995).
5. increased pre-exercise muscle glycogen levels (Meyers et al., 1989; Scott et al., 1992; Hughes et al., 1995).

The profile of FFA contained within a fat supplemented diet may also be important since this may in turn influence the FFA profile of the fats stored within the body, and therefore the FFA mobilized with exercise. FFA constitute a broad spectrum of molecules, from the so-called short to long fatty acids, which may well show different rates of diffusion through cells affecting their rate of utilization with exercise. Although techniques are available for the loading of plasma with FFA with a predetermined profile (Orme & Harris, 1997), and for the investigation of their use during exercise (Orme et al., 1995), no studies have been undertaken in the horse to examine specifically the utilization of the different FFA during exercise. Medium chain triglycerides (MCT) have been proposed as a potential ergogenic aid during exercise in man. In humans MCT are emptied more rapidly from the stomach (Beckers et al., 1992), rapidly absorbed and hydrolyzed by the small intestine, and secreted directly into the systemic circulation. Furthermore, MCT do not require the acetyltransferase system to cross the inner mitochondrial wall in order to undergo oxidation. Orally supplied MCT, however, do not lead to a sparing of muscle glycogen use during moderate to intense exercise (Jeukendrup et al., 1995 & 1996; Massicotte et al., 1992) and their effect on performance is questionable.

Dietary Ergogenic Strategies for Racing

An analysis of the metabolic events occurring in the muscles during intense racing exercise indicates four areas where improved metabolic function could theoretically be beneficial to performance. These are:

1. increased availability of locally stored muscle glycogen
2. an increased rate of oxidative metabolism
3. improved intracellular acid-base regulation
4. increased phosphagen support of ATP metabolism.

I. Increased Availability of Locally Stored Glycogen

Aerobic and anaerobic utilization of carbohydrate constitutes the major fuel store for the regeneration of ATP in muscle during racing. Endurance rides of 100 km or more may bring about total depletion of the glycogen store in some muscle fibers (Snow et al., 1981; Snow et al., 1982), but over normal racing distances the decrease is probably no more than 30-40% (Hodgson et al., 1984; Harris et al., 1987; Snow et al., 1987). With normal non-exercise muscle glycogen content of around 600 mmol/kg dry muscle (dm) glucose units, glycogen is unlikely to be limiting in flat or harness racing, although over longer National Hunt distances local depletion within some muscle fibers is a possibility (Snow et al., 1981). Glycogen loss during training is accommodated easily by a normal diet containing concentrates (Snow and Harris, 1991) and is unlikely to lead to depletion prior to racing.

The use of "glycogen loading" stems from early work in man (Bergström and Hultman, 1966; Bergström et al., 1967). The normal glycogen content of rested human muscle is approximately 300 mmol/kg dm but may increase twofold by a combination of exercise and diet. This strategy is now followed ardently by human middle distance and endurance runners. There have been claims both for (Topliff et al., 1983; 1985) and against (Topliff et al., 1987; Snow et al., 1987) a similar effect in horses fed a carbohydrate supplement. However, no effect on either the rate of muscle glycogen resynthesis, or the final level reached, was seen when increased levels of starch were added to the feed (Topliff et al., 1987; Snow, 1992). Glycogen repletion, following exercise resulting in a 40% decrease in the muscle store, was approximately the same in horses fed a low carbohydrate (LC) diet consisting of hay, a normal carbohydrate (NC) diet consisting of pelleted concentrate plus hay, or a high carbohydrate (HC) diet which was the NC diet supplemented with intravenous infusions of 0.45 kg glucose (circa 0.9 g/kg bwt) on each of the first 2 days (Snow et al., 1987). However, an increased rate of glycogen resynthesis was observed by Davie et al. (1995) when 6 g/kg bwt dextrose was infused intravenous following exercise resulting in a 50% decrease in the muscle content. Muscle contents after 24 h recovery, however, were no longer significantly different with or without dextrose infusion and therefore it would appear that any advantage gained in the repletion of the muscle glycogen stores, even by this extreme procedure, is short-lived.

In general, there seems to be little or no case for adding high carbohydrate supplements to feed in preparing horses for racing, in contrast to the well described use of such supplements by human athletes to bring about "glycogen loading" of the muscles.

II. An Improved Rate of Oxidative Metabolism

Undoubtedly the best way to achieve an increase in the oxidative capacity of muscle is through training. However, a number of nutritional strategies have been used in an attempt to increase this still further.

Q10

One example is ubiquinone or Q10 but, apart from its known role in mitochondrial electron transport, there is scant evidence that it in any way limits oxidative metabolism in normal, well-fed animals. Similarly, there are no studies showing uptake from the gut and into the muscles of the horse, and no evidence of any effect upon tissue function or physical performance. Q10 has been shown in rats to be absorbed from the gut but orally supplied Q10 exhibits a restricted distribution within tissues (Zhang et al., 1995; Zhang et al., 1996). Studies in humans have generally failed to find any effect upon performance (Laaksonen et al., 1995) or the attenuation of muscle lactate formation (Porter et al., 1995) with exercise performed close to the onset of blood lactate accumulation (OBLA).

L-CARNITINE

L-carnitine is an essential cofactor for the transport of long chain fatty acids across the inner mitochondrial membrane and has been suggested to be rate limiting to fat utilization during exercise. This, however, seems unlikely in view of the very high concentration of L-carnitine that is found in tissues such as muscle (circa 6-7 mM) (Foster and Harris, 1992; Harris et al., 1995) relative to its affinity for mitochondrial bound carnitine-palmitoyl transferase. Of possibly more relevance to racing is the role of L-carnitine in carbohydrate oxidation in buffering the mitochondrial concentration of acetyl CoA (Alkonyl et al., 1975; Foster and Harris, 1987; Harris et al., 1987) and the regulation of pyruvate dehydrogenase activity (Carlin et al., 1990, Constantin-Teodosiu et al., 1991). Accumulation of acetylcarnitine provides a metabolic sink for the temporary storage of 2-carbon acetyl units formed from pyruvate and ß-oxidation of FFA, as well as preventing the local depletion of coenzyme A, the effect of which would be to inhibit functioning of the TCA cycle. The accumulation of acetylcarnitine following a warm-up exercise may account for 90% of the available L-carnitine pool in the muscle, providing a valuable source of 2-carbon units during the early stages of racing. The high concentration of L-carnitine found in muscle is clearly of advantage in respect of this role, and a further increase in concentration could in theory enhance its overall function still further. As in man, however, L-carnitine is only poorly absorbed in the horse (Harris et al., 1995) although oral supplementation with 10-60 g L-carnitine per day will effect a doubling or more of the plasma concentration (Foster et al., 1988). Despite this, prolonged oral supplementation at these levels for 58 days had no effect on the muscle content. In a further study, Harris et al. (1995) administered 10 g L-carnitine intravenously daily for 26 days. Despite plasma concentrations 30 times higher than normal immediately after infusion, and 3 times higher still after 6 h, no change was observed in the muscle content. Thus the case for manipulation of the muscle L-carnitine (by any means) is very tenuous, and this is probably true also for the human. Despite this, L-carnitine supplements continue to be popular among athletes and are often included in compound supplements claimed to enhance “fat burning.” L-carnitine supplements have been available for horses for some years.

DICHLOROACETATE AND 2-CHLOROPROPIONATE

The activation of pyruvate dehydrogenase through the use of dichloroacetate (DCA) or 2-chloropropionate (2-CP) does, however, appear to be effective in increasing the oxidation rate of pyruvate during the early stages of exercise, and to attenuate the accumulation of lactate under these conditions. This has been demonstrated in man (Mercier et al., 1994, Timmons et al., 1998) and dogs (Timmons et al., 1996), following oral and intravenous administration, but has yet to be shown to be an effective method of limiting lactate under race conditions. No studies of either DCA or 2-CP have been undertaken in the horse, although anecdotal evidence indicates that the former has been used in racing. Gannon and Kendall (1982) reported a positive effect of DCA on the performance of greyhound dogs when this was administered as the diisopropylammonium salt in conjunction with N,N-dimethylglycine.

BRANCHED CHAIN AMINO ACIDS (BCAA)

Dietary supplied BCAA may affect performance by increasing the concentration of TCA intermediates (anaplerosis) available for condensation with acetylCoA enabling an increase in the turnover rate of the cycle, as well as an effect on factors contributing to central fatigue. While BCAA supplements are available to the human athlete, data on their ergogenic effect following oral ingestion is contradictory (e.g. Bigard et al., 1996; Blomstrand et al., 1996). In the study of Blomstrand et al. (1996), a positive effect on metabolism (increased alanine synthesis, reduced fall in muscle glutamate, and lower glycogen utilization during exercise) was apparent and was favorable to an increase in endurance performance. Little or no quantitative data on the uptake, metabolism and effect on performance of BCAA are currently available for the competitive horse, although once again BCAA supplements are available.

III. Improved Intracellular Acid-Base Regulation

It is inevitable during racing that lactate accumulation will occur in muscle, decreasing the intracellular pH. As the race continues, loss of adenine nucleotide may commence, signifying a breakdown in the normal mechanisms regulating ADP homeostasis at the myosin-actin cross-bridge interface. It is probable that the two events are linked, i.e. the onset of adenine nucleotide loss and pH decrease (Sewell and Harris, 1992; Sewell et al., 1992), while the consequential rise in ADP may initiate a loss of performance arising from local muscle fatigue. It is essential, if performance is to be maintained, that H^+ ions released with lactate are either transported out of the muscle fibers or neutralized by physicochemical buffering.

SODIUM BICARBONATE

Sodium bicarbonate has been used widely in racing to effect an improvement in intracellular acid-base regulation. It is cheap, easily obtained, relatively simple to administer and has proved effective in facilitating H^+ removal from the working muscle fibers. Results from different studies, however, have been contradictory, possibly reflecting the wide range of doses used and inadequate information on the changes with time in plasma bicarbonate and pH following ingestion. Greenhaff et al. (1990) reported a peak increase in plasma acid-base excess (ABE) of 6.8 mmol/l 6 h following intubation of 0.6 g $NaHCO_3$/kg bwt. A lower response was seen using 0.3 g/kg bwt, a dose used in the horse by Lawrence et al. (1987) where it resulted in improved maintenance of plasma pH during exercise. Subsequent studies (Greenhaff et al., 1991b) using 0.6 g/kg bwt showed a reduced loss of muscle adenine nucleotide to IMP during a 2- min standardized treadmill exercise test resulting in a blood lactate concentration of approximately 20 mmol/l. These results are indicative of an improvement in *intracellular* pH control during the 2 minutes of exercise. Lactate efflux from muscle into plasma appeared to be increased with bicarbonate. The effect of $NaHC0_3$ administration upon actual race performance, however, remains unresolved. Lloyd et al. (1993) observed a detrimental effect of 1.0 g/kg bwt during treadmill exercise at approximately 110% VO_2max. No effect of 0.6 g/kg bwt was observed by Greenhaff et al. (1991a) during a 1000 m field test confirming reports of Kelsö et al. (1987). However, field studies are particularly difficult to undertake and the intrinsic high within-horse variance in performance time (measurable on test and retest), as well as differences in the pattern of the exercise performed, could easily obscure any small effects upon performance. Thus, although there is a clear rationale for the use of $NaHCO_3$, and its effect in the target tissue (plasma) has been established, there is inadequate evidence of any effect on performance under field conditions.

INTRACELLULAR PHYSICOCHEMICAL BUFFERING

Sodium bicarbonate, however, provides only an indirect approach to improving intracellular acid-base status, necessitating as it does the efflux of H^+ ions out of the cell. The primary defense against intracellular H^+ increase is afforded by the cell's own physicochemical buffers made up of organic and inorganic phosphates, amino acids and proteins, and the histidine dipeptide (of which the predominant species in equine muscle is carnosine). In equine muscle carnosine may account for 30% or more of the physicochemical buffering in type II fibers (Sewell et al., 1991) where it is found in highest concentrations (Dunnett and Harris, 1995; Harris et al., 1998). Although its importance to intracellular acid-base regulation is evident, the physiology governing its synthesis and metabolism remains to be elucidated. Addition of 0.4% L-histidine to the feed over 14 days resulted in a 19.2% increase in muscle carnosine content, compared to horses fed a diet containing 0.14% L-histidine (Powell et al., 1991). The increase, however, was not statistically significant. The alternative is that carnosine synthesis is

limited by the availability of ß-alanine. However, administration of this is known to result in the efflux of the beta amino acid, taurine, from tissues including heart and skeletal muscle and in humans is associated with symptoms of paresthesia, even at low doses (R.C.Harris, M.Dunnett, J.Fallowfield and J.Coakley, unpublished).

Phosphate potentially constitutes the next largest physicochemical buffer in muscle, with the majority of this in the resting state being combined with creatine to form phosphorylcreatine (PCr). PCr is a relatively weak buffer of H^+ ions at normal physiological pH. However, a much bigger buffering effect is observed only after release of the PCr bound phosphate, ultimately to form inorganic and sugar phosphates. Increasing the PCr content of resting muscle by creatine (Cr) "loading" (discussed in the next section) may (at least in the human) increase the potential buffering from this pool by as much as 10%.

IV. Increased Phosphagen Support of ATP Metabolism

The continued accumulation of lactate in working muscle fibers will ultimately exceed the capacity of the physicochemical buffers and the cell's capacity to transport H^+ ions. Intracellular pH will fall affecting both the contractile process and the mechanisms regulating ADP removal at myosin-actin cross-bridge sites. Failure to maintain normal ATP/ADP homeostasis is a major threat to the continuation of the cross-bridge cycling rate necessary to maintain work output. Although improvements in the pyruvate oxidation rate (see previous) and H^+ buffering and transport (see previous) will delay the point when this is reached, improvement in the support given to ATP/ADP homeostasis is an important last step where metabolic intervention may be used to improve performance, in particular manipulation of the PCr content, which functions in the cell as the "low ADP threshold sensor."

The comprehensive role of PCr in muscle as a buffer to ADP accumulation, and high energy phosphate transfer and integration within the cell, is summarized in Figure 1. As already noted, PCr also constitutes the largest pool of metabolically active phosphate able to contribute to acid-base regulation when this is released from PCr (with exercise leading to a net fall in PCr). The size of the PCr store at the start of exercise will be important both to buffering ADP accumulation as well as H^+ accumulation. A greater concentration of free Cr will also contribute to a faster resynthesis, in absolute terms, in PCr (Greenhaff et al., 1994) which will be especially important in events requiring intensive intermittent exercise.

Studies in man have shown that Cr administered orally is absorbed rapidly from the gut and taken up into muscle (Harris et al., 1992). Supplementation of the diet with 4 x 5g CrH_2O for 5 days resulted in a 30% increase in the muscle [Cr + PCr] store in some individuals, with a proportionate increase occurring in both PCr and Cr. A maximum content in human muscle of around 160-180 mmol/kg dm was indicated in these studies. Lower doses in humans of 2-3 g taken daily, but over a longer period, will bring about a similar increase in the muscle [PCr + Cr] content. Uptake into muscle is by means of a transporter and is greatest in vegetarian subjects who do not regularly encounter Cr in their diet. Creatine

uptake appears depressed in subjects regularly taking Cr supplements (R.C. Harris, unpublished). It is facilitated locally by exercise (Harris et al., 1992), and in the non-exercised state by insulin (Haughland and Chang, 1975; Green et al., 1995). Elevation of the muscle Cr and PCr contents has been shown in numerous studies to increase the capacity for sustained or intermittent hard exercise (Balsom et al., 1993a; Greenhaff et al., 1993; Harris et al., 1993; Birch et al., 1994) and may also exert an anabolic effect increasing peak strength (Earnest et al., 1995; Vandenberghe et al., 1997). Muscle Cr elevation does not appear to enhance the capacity for prolonged submaximal exercise (Balsom et al., 1993b; Stroud et al., 1994) although it is probable that an effect would be seen with changes in pace with exercise continued to the point of marked glycogen depletion (affecting ATP/ADP homeostasis). The increased capacity with Cr loading to undertake short term exhaustive exercise is associated with a reduction in adenine nucleotide loss (Greenhaff et al., 1993; Balsom et al., 1993a).

Creatine is rapidly absorbed in the dog (Harris and Lowe, 1995) where in the feral state up to 5 g may be ingested in a single meal (calculated for a dog of 35 kg). In contrast, in the horse Cr is only poorly absorbed (Sewell and Harris, 1995). Intubation of 50 mg/kg bwt resulted in an increased plasma concentration from 40 to 100 Fmol/l after 4-6 h; the same dose resulted in an increase to 800-1000 Fmol/l in the human. Greater absorption in the horse may occur when Cr is coadministered with feed (R.C. Harris, unpublished), but is still far below that observed in man and dog. Administration of 3 x 50mg/kg bwt added to the drinking water over 13 days had no effect on the muscle [PCr + Cr] content (Sewell and Harris, 1995). Thus although there is again a clear rationale for an effect on performance by Cr and effects have been established in man (and partially in dog), adequate evidence for an effect of Cr supplementation is lacking in the competition horse.

Concluding Remarks

Several issues here will, inevitably, be viewed with some misgivings by bodies governing equestrian sports. In some areas the edges between what is and what is not acceptable are blurred by the fact that many ergogenic aids are found naturally in the body. For example, in the case of carnosine, would high levels of either of its precursors be regarded as acceptable in the knowledge that, if this were to result in an increase in muscle carnosine, this could affect performance? One of these precursors, L-histidine, occurs naturally in the free or protein bound form in feeds, but not so ß-alanine. If this defies the rules of equine competition, can we justifiably administer thiamine, which is again a metabolic regulator, in this case a cofactor of pyruvate dehydrogenase? Certainly it would be difficult to see any greater justification for the latter compared, for instance, with carnitine which functions in the same relative metabolic area and which like thiamine is found in the normal diet.

Because the horse is herbivorous, Cr is not a natural component of the equine diet, in contrast to man and the dog. There is no doubting the role of Cr and PCr in energy metabolism in muscle and other tissues (e.g. brain, heart, spermatozoa,

retina) and that a low content may not only impair performance but also enhance the degradation of adenine nucleotide to IMP and ultimately to uric acid. Degradation of IMP to uric acid is one of several routes by which highly reactive free radicals are formed and which during exercise may challenge the antioxidant defenses (Mills et al., 1997). A high Cr (and carnosine content) may in this case be regarded as beneficial to the horse, helping to limit the damage resulting from intensive exercise. Finally, in humans some of the most encouraging reports of the effectiveness of Cr have been in the supplementation of the elderly. While veteran races for horses and dogs have yet to be introduced, should we not be considering investigating the use of dietary Cr in, for instance, elderly working dogs? How different should we now regard the feeding of compounds such as Cr, carnitine and carnosine, or their precursors, to the feeding of vitamins, selenium or carbohydrate supplements?

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