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Effects of oral L-carnitine supplementation in racing Greyhounds

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Abstract

L-Carnitine supplementation can stimulate erythropoiesis, reduce exercise-induced plasma lactate concentrations and decrease post-exercise muscle damage. Next to horses, Greyhounds represent the premier animal racing species and perform short-duration, very high-intensity exercise that has the potential to incur substantial muscle damage. Under resting and standard racing conditions (5/16 mile), we tested the novel hypotheses that L-carnitine supplementation in Greyhounds would: (1) elevate haematocrit at rest and immediately post-exercise; (2) reduce peak post-exercise plasma lactate; and (3) reduce indices of muscle damage (plasma creatine phosphokinase, CPK and aspartate aminotransferase, AST). Six conditioned Greyhounds (30.1 ± 1.6 kg) underwent a randomized placebo-controlled crossover study to determine the effects of 6 weeks of L-carnitine supplementation (100 mg kg^{-1} of body weight/day) at rest and following a maximal speed 5/16 mile race. In accordance with our hypotheses, L-carnitine elevated resting and immediately post-race haematocrit (control, 60.1 ± 1.7, L-carnitine, 63.6 ± 1.7; $P < 0.05$) and reduced peak post-race plasma CPK and AST concentrations (both $P < 0.05$). Those dogs with the highest peak post-exercise plasma CPK concentrations under placebo conditions evidenced the greatest reduction with L-carnitine supplementation ($r = 0.99$, $P < 0.01$). However, contrary to our hypotheses, L-carnitine did not change peak post-exercise plasma lactate concentrations (control, 27.0 ± 2.1, L-carnitine, 27.7 ± 1.3; $P > 0.05$). We conclude that L-carnitine supplementation increases the potential for oxygen transport and reduces plasma indicators of muscle damage, CPK and AST in racing Greyhounds.

Keywords: haematocrit; oxygen transport; muscle damage; aspartate aminotransferase; creatine phosphokinase; lactate; maximal exercise

Introduction

Carnitine, first extracted from muscle in 1905[1,2] and named from the Latin *carnis* (flesh or meat), is best known for its role as a cofactor (L-carnitine) of carnitine acyltransferase, which transports long-chain fatty acids across the mitochondrial inner membrane. Without L-carnitine, the mitochondrial inner membrane would be impermeable to long-chain fatty acids and fatty acyl CoA esters[2]. Other important functions of carnitine include maintaining the acetyl CoA/CoA ratio, which acts to control pyruvate dehydrogenase (PDH) and thus lactate production and accumulation. Carnitine also reduces the catabolism of purines, free radical formation and sarcolemmal disruption and is associated with decreased muscle soreness[3]. Skeletal muscle constitutes the principle reservoir of carnitine in the body. However, during high-intensity exercise, intramuscular carnitine concentrations plummet, decreasing to values approaching that required for half-maximal activity of carnitine acyltransferase for carnitine (0.25–0.45 mM l^{-1})[4].

Given the potential to impact human athletic performance, L-carnitine supplementation has been widely investigated and there is scientific support, some of it equivocal, for L-carnitine supplementation resulting in: (1) improved maximal oxygen uptake[5–6] (not)[7]; (2) improved exercise performance[8] (not)[9]; (3) elevated erythropoiesis[10]; (4) reduction of muscle damage and soreness after maximal exercise[11,12]; and (5) reduced lactic acid accumulation[8,13] (not)[14].

L-Carnitine supplementation increases the plasma carnitine concentration and, in species as diverse as mice and pigeons, this leads to an elevation in heart and skeletal muscle L-carnitine pools[15,16]. However, even in the horse where increased plasma concentrations are not observed, L-carnitine supplementation is still associated with capillary and possibly oxidative enzyme adaptations to training[17].

Racing Greyhounds engage in very intense exercise of short duration (typically 5/16 mile, ~28–32 s) and there are several putative mechanisms by which L-carnitine supplementation may benefit these athletes. Specifically, sprint exercise places disproportionate demands on substrate-level phosphorylation, i.e. phosphocreatine breakdown and anaerobic glycolysis leading to lactate accumulation. If it is possible to increase the participation of fat as an energy source, rapid glycogen depletion within type II fibres[18] may be avoided. Any slowing of glycolysis would also be expected to retard lactic acid production and thus reduce the accumulation of hydrogen ions, thereby constraining any deleterious effects on the contractile machinery. Greyhounds also have an extraordinarily well-developed oxygen transport system with very large hearts (approaching 2% body mass)[19] and presumably, therefore, high cardiac outputs and potential for oxygen delivery to the working muscles. During whole body exercise, it is generally recognized that humans and animals at maximal oxygen uptakes evidence a supply limitation to maximal oxygen utilization[20,21]. If L-carnitine has an erythropoietic effect in Greyhounds, this may further increase exercise oxygen transport by elevating arterial oxygen content. The elevated maximal oxygen uptakes reported after carnitine supplementation[5,6] may potentially have resulted from such an increase in arterial haematocrit. Finally, short-term, high-intensity sprint running has the propensity to incur substantial muscle damage in humans[22] and it is likely that this also occurs in racing dogs. Whether L-carnitine supplementation can reduce muscle damage in Greyhound dogs as it does in humans[11,12] has not been determined.

The purpose of the present investigation was to test the novel hypotheses that L-carnitine supplementation in racing Greyhounds would: (1) elevate systemic haematocrit (at rest and immediately post-exercise); (2) reduce peak post-race plasma lactate accumulation; and (3) decrease plasma indicators of muscle damage (creatine phosphokinase, CPK and aspartate aminotransferase, AST).

Methods

Animals

Six healthy Greyhound dogs that had previously been raced on the track were acquired for this investigation. There were four intact females and two intact males ranging in age from 2 to 4 years and weighing 30.1 ± 1.6 kg. They were housed at the National Greyhound Association Park in Abilene, Kansas in individualized standard Greyhound wire crates (approximately 42 inches long × 26 inches wide × 36 inches high) with shredded paper for bedding. They were turned out into a 20' × 30' run three times per day for 20- to 30-min intervals. The dogs were fed Iams (Iams Mini Chunks®, Iams Company, Dayton, OH, USA), adult dog food (3.5–4 cups; minimum of 26.0% crude protein, minimum of 15.0% crude fat, maximum of 5.0% crude fibre and maximum of 10.0% moisture), once daily in the evening and had access to water during turnout times. The Greyhounds were up to date on vaccinations including distemper virus, adenovirus, parainfluenza virus, parvovirus and *Bordetella bronchiseptica*, as well as being on monthly Heartguard Plus. All procedures used were approved by the Kansas State University Animal Care and Use Committee.

Experimental protocol

Each dog completed one simulated race on the 5/16 mile training track after each of the following conditions in a randomized crossover design: no treatment, placebo and L-carnitine supplementation. Either a liquid supplement of L-carnitine at a dosage of 100 mg kg^{-1} or an equivalent amount of water was administered as a top dressing on the feed daily depending on whether the animal was in the treatment or placebo group. Consumption of the entire diet was observed for each animal. Prior to the simulated race, each dog had a custom-made seven French flexible introducer catheter (Polyurethane Peel-Away Catheter, Access Technologies, Skokie, IL, USA) placed aseptically under local anaesthesia (2% lidocaine) in either the right or left jugular vein with a short extension set attached to the catheter and sutured to the skin *via* elastikon butterflies and 2.0 Braunamid. Resting blood samples and rectal temperatures were obtained from the dogs before being transported by a climate-controlled dog trailer approximately 2 miles to a local training track. Greyhounds were divided into three heats with two dogs in each heat and run on the track in a race-simulated timed run (5/16 mile distance) around the 4/16th mile track, chasing a lure. The Greyhounds were caught immediately after reaching the lure, with venous blood samples being obtained within 15–30 s post-race. Additional catheter samples and rectal temperatures were collected from each dog at 10, 20 and 30 min and 1, 3 and 5 h post-exercise. In addition, follow-up samples were obtained at 24, 48 h and 1 week post-race from the jugular vein with 21 G 1" vacutaner needles and processed in a manner identical to the post-exercise samples. A second identical run (run 2) was performed 8 weeks after the first run, with treatment and control groups being reversed.

Blood analysis

At sampling times, both heparinized and ethylenediamine tetraacetic acid (EDTA) samples were obtained and placed immediately on ice. Initial processing of the heparinized samples included spun haematocrits (Micro-capillary Centrifuge, Model MB, International Equipment Company, Needham, Heights, MA, USA) read with a micro-haematocrit reader. All blood samples were centrifuged and the plasma was recovered and frozen at $-70°C$ for storage prior to analysis, as appropriate. A portion of the heparinized plasma was analysed for creatine phosphokinase (CPK; Hitachi 911, Roche Diagnostics, Indianapolis, IN, USA), aspartate aminotransferase (AST; Hitachi 911, Roche Diagnostics, Indianapolis, IN, USA), glucose (GLU; Nova CCX, Nova Biomedical, Waltham, MA, USA) and plasma lactate (La^-; Nova CCX, Nova Biomedical, Waltham, MA, USA). L-Carnitine (Total, Free and Ester) was quantified using a radioisotopic enzymatic method (Metabolic Analysis Labs, Madison, WI)[23]. Plasma NEFAs (FFA) were determined on the EDTA-treated samples using a commercial enzymatic–colorimetric assay (NEFA-C kit number 994-75 409, Wako, Richmond, VA).

Statistical analysis

A paired *t*-test indicated no significant differences between the two control runs and therefore these two trials were averaged. Differences in measured variables over time were analysed using a mixed-effects model where time is a fixed effect and dog and dog × time are random effects. The response equals overall effect + dog effect + time effect + dog × time interaction + error, where dog and dog × time are random effects. When significant differences were found, a least-square means *post hoc* test was used to determine where differences existed. The Statistical Analysis System Program 9.1.2 statistical package (SAS Institute, Incorporated, Cary, NC, USA) was used for the ANOVA. Pearson product moment correlations were used to determine relationships between variables. A paired *t*-test was used to determine whether differences existed between conditions for peak values of AST and CPK. The SigmaStat 3.0 statistical package was used to analyse the correlations and paired data. One-tailed tests were utilized where appropriate directional *a priori* hypotheses were tested as indicated. Significance was accepted at $p \leq 0.05$ level for all variables.

Results

L-Carnitine levels

The resting plasma L-carnitine levels were substantially greater in the treated dogs, as expected. Total, free and ester L-carnitine levels were 27.7 ± 3.8, 23.7 ± 3.2 and $4.0 \pm 0.7\,\mu moles\,l^{-1}$ for control conditions and 185.3 ± 6.6, 152.7 ± 7.7 and $32.6 \pm 5.2\,\mu moles\,l^{-1}$ for treatment trials, respectively (all $p < 0.05$).

Indications of muscle damage

When treated with L-carnitine, the Greyhounds exhibited a decrease in muscle damage ($p < 0.05$, one-tailed) as evidenced by reduced peak concentrations of plasma CPK and AST (Fig. 1; Tables 1, 2). Those dogs with the greatest degree of muscle damage (as assessed by peak CPK concentrations) on the placebo trial evidenced the largest reductions following L-carnitine treatment ($r = 0.99$, placebo CPK *versus* delta CPK with treatment, $p < 0.05$). There were also significant correlations between: (1) peak CPK and AST on the placebo trial ($r = 0.99$); (2) peak CPK and peak AST on the treatment trial ($r = 0.88$) and (3) delta (placebo treatment) peak CPK and delta peak AST ($r = 0.93$).

Metabolic and haematologic variables

Plasma FFAs were significantly reduced in the treatment condition at rest, were not different immediately post-exercise and were higher at 10 min post-exercise in the L-carnitine treatment condition (Table 1 and Fig. 2). Haematocrit was significantly elevated in the treatment *versus* placebo condition at rest and immediately post-exercise (Fig. 3). No differences existed between treatment and placebo conditions at rest or immediately post-exercise in plasma glucose (Tables 1 and 2), plasma lactate (Tables 1 and 2), race times

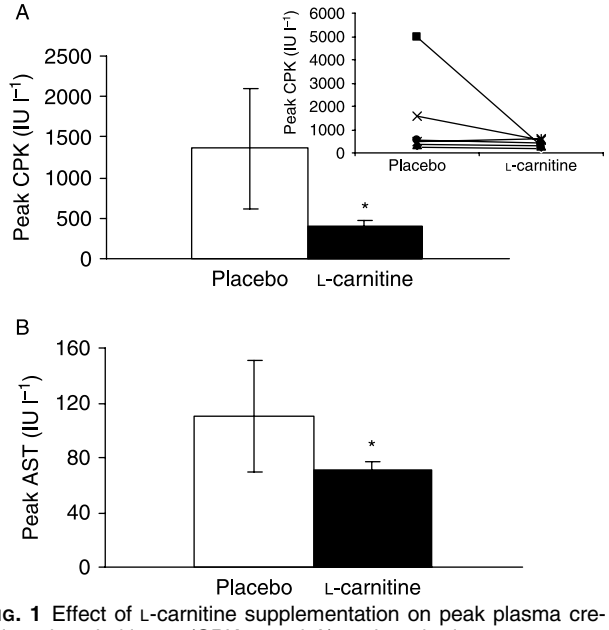

FIG. 1 Effect of L-carnitine supplementation on peak plasma creatine phosphokinase (CPK; panel A) and peak plasma aspartate aminotransferase (AST; panel B) concentrations following a simulated 5/16th mile race (number of dogs = 6). Data are presented as mean ± SE. *Placebo-treated Greyhounds demonstrated higher peak plasma CPK and AST than L-carnitine-treated Greyhounds post-exercise ($P \leq 0.05$, one-tailed). Inset in panel A illustrates individual Greyhound results

Table 1 Measured variables under treatment with placebo

Time period	Haematocrit (%)	Plasma lactate ($mmol\,l^{-1}$)	Plasma FFA ($mmol\,l^{-1}$)	Plasma glucose ($mg\,dl^{-1}$)	Plasma CPK ($IU\,l^{-1}$)	Plasma AST ($IU\,l^{-1}$)
Rest	55 ± 1	1.9 ± 0.3	0.45 ± 0.07	126.7 ± 2.4	104.3 ± 19.4	27.4 ± 3.4
IPE	60 ± 2	23.7 ± 1.7	0.40 ± 0.09	177.3 ± 5.4	219.6 ± 79.0	39.8 ± 7.1
10 min	61 ± 2	25.4 ± 2.3	0.37 ± 0.05	196.7 ± 8.6	200.3 ± 15.2	43.3 ± 5.1
20 min	60 ± 1	19.3 ± 1.7	0.26 ± 0.08	171.4 ± 9.6	198.5 ± 25.0	49.2 ± 6.2
30 min	57 ± 1	9.7 ± 0.7	0.39 ± 0.06	149.8 ± 9.6	193.3 ± 23.8	51.7 ± 6.1
1 h	55 ± 2	2.7 ± 0.4	0.64 ± 0.09	132.0 ± 4.6	238.3 ± 43.0	52.8 ± 6.7
3 h	52 ± 2	1.5 ± 0.2	0.78 ± 0.20	123.9 ± 3.4	626.6 ± 218.0	68.6 ± 11.1
5 h	53 ± 2	1.2 ± 0.1	0.77 ± 0.13	119.7 ± 3.8	816.8 ± 417.1	76.6 ± 14.5
24 h	53 ± 1	1.3 ± 0.2	0.46 ± 0.08	118.9 ± 1.0	947.8 ± 768.3	77.7 ± 40.5
48 h	52 ± 1	1.4 ± 0.3	0.32 ± 0.06	120.4 ± 3.1	460.4 ± 341.7	54.2 ± 26.3
7 days	53 ± 2	1.2 ± 0.2	0.31 ± 0.06	119.9 ± 1.2	108.6 ± 11.7	32.0 ± 2.8

Time period column refers to the time samples following immediate post-exercise (IPE) being drawn at the indicated times, post-exercise. Individual dogs may have demonstrated 'peak' values at different times post-exercise.

(treatment; 33.19 ± 0.24 s: placebo; 33.22 ± 0.47 s: L-carnitine) or rectal temperature (106.4 ± 0.6°F: placebo; 106.3 ± 0.4°F: L-carnitine) (all $P > 0.05$).

Discussion

The principal novel findings of this investigation are that L-carnitine supplementation ($100\,mg\,kg^{-1}$) in racing Greyhound dogs elevates systemic haematocrit at rest and immediately post-exercise and reduces peak plasma CPK and AST indicators of muscle damage. These results suggest that L-carnitine supplementation might facilitate greater oxygen transport during maximal exercise, whilst reducing muscle damage and potentially enhancing post-race recovery. Given previous research in humans[8,13,14] and the putative intracellular actions of L-carnitine with respect to enhancing pyruvate flux through PDH[2,25], the unchanged peak post-exercise plasma lactate concentration was not expected. Considering that plasma lactate accumulation reflects a complex interaction between lactate production and removal processes as well as distribution dynamics amongst different compartments, it would be speculative to consider that these data indicate an unchanged muscle lactate production. Notwithstanding this consideration, however, the present data do not support the hypothesis that L-carnitine supplementation decreases muscle lactate production in racing Greyhounds.

Comparison with previous literature

To our knowledge, there have been no studies to date examining the effects of L-carnitine supplementation in racing Greyhounds. However, there are data in other species supporting that L-carnitine may:

1. *Stimulate erythropoiesis*[10] that may underlie the elevated systemic haematocrit found at rest and following exercise in the present investigation. As discussed below, L-carnitine supplementation has been found to increase maximal oxygen uptake in some[5,6] but not all[7] studies. Because maximal oxygen uptake during large muscle mass exercise is generally limited by oxygen delivery (as demonstrated by the proportional increase in maximal oxygen uptake with elevated oxygen delivery)[26,27, review 20] if the same maximal cardiac output is achieved at a higher haematocrit, and therefore arterial oxygen content, an improved maximal oxygen uptake would be expected.
2. *Reduce muscle damage* consequent to intense exercise[3,11,12 review 2]. The mean peak level of plasma CPK found in the placebo trial in the present inves-

Table 2 Measured variables under L-carnitine treatment

Time period	Haematocrit (%)	Plasma lactate ($mmol\,l^{-1}$)	Plasma FFA ($mmol\,l^{-1}$)	Plasma glucose ($mg\,dl^{-1}$)	Plasma CPK ($IU\,l^{-1}$)	Plasma AST ($IU\,l^{-1}$)
Rest	59 ± 1*	1.9 ± 0.3	0.25 ± 0.07*	127.0 ± 3.4	101.3 ± 9.5	34.7 ± 2.5
IPE	64 ± 2*	25.2 ± 1.7	0.41 ± 0.08	186.3 ± 13.7	165.3 ± 22.6	44.8 ± 3.5
10 min	64 ± 2*	27.7 ± 1.3	0.57 ± 0.10*	195.7 ± 15.4	230.8 ± 33.5	53.8 ± 3.7
20 min	62 ± 1*	17.8 ± 1.2	0.31 ± 0.10	165.6 ± 16.9	239.8 ± 46.4	57.2 ± 4.4
30 min	60 ± 2*	10.3 ± 1.6	0.40 ± 0.04	158.3 ± 15.8	231.2 ± 33.6	60.7 ± 5.5
1 h	57 ± 1*	2.4 ± 0.5	0.60 ± 0.07	139.7 ± 2.6	247.2 ± 31.6	60.3 ± 5.7
3 h	57 ± 2*	1.0 ± 0.2	0.72 ± 0.08	128.8 ± 3.3	339.0 ± 45.8	66.5 ± 5.4
5 h	55 ± 2*	1.0 ± 0.1	0.65 ± 0.05	124.5 ± 3.5	390.7 ± 72.8	68.3 ± 6.3
24 h	57 ± 2*	1.9 ± 0.3	0.50 ± 0.07	136.7 ± 12.4	212.3 ± 50.5*	44.3 ± 4.5*
48 h	58 ± 1*	1.6 ± 0.2	0.42 ± 0.07	124.8 ± 5.1	195.2 ± 90.9	34.5 ± 4.4
7 days	59 ± 1*	1.4 ± 0.2	0.30 ± 0.10	128.7 ± 3.9	148.7 ± 32.5	44.0 ± 8.1

Time period column refers to the time samples following immediate post-exercise (IPE) being drawn at the indicated times, post-exercise.* Indicates L-carnitine-treated animals significantly different from placebo-treated animals. Individual dogs may have demonstrated 'peak' values at different times post-exercise.

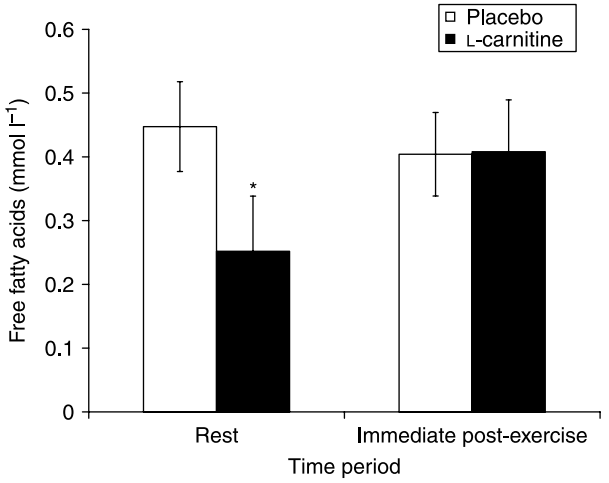

FIG. 2 Plasma free fatty acid concentrations (FFAs) in L-carnitine-treated Greyhounds and placebo-treated Greyhounds at rest and immediately post-exercise (number of dogs = 6). Data are presented as mean ± SE. *L-Carnitine-treated Greyhounds demonstrated lower resting free fatty acids (FFAs) than placebo-treated Greyhounds ($P < 0.05$)

tigation was ~1350 IU l^{-1} and occurred between 5 and 24 h post-exercise. This level is higher than that observed after prolonged moderate exercise in Beagles[28], slightly below those present in endurance-run sled dogs[29] and far lower than that observed following intense eccentric weightlifting exercise in humans (>7000 IU l^{-1})[30]. L-Carnitine supplementation reduced peak plasma CPK levels close to 35% in the present investigation, which is somewhat less than the ~70% demonstrated by Volek *et al.*[3] following intense repetitive squat exercise (subjects supplemented 2 g/day of L-carnitine for 3 weeks).

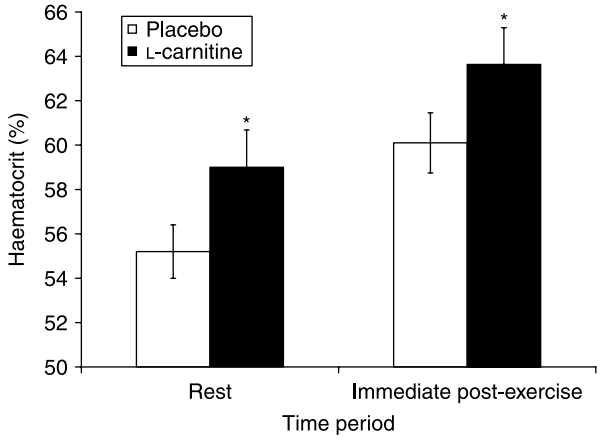

FIG. 3 Haematocrit of Greyhounds treated with L-carnitine and placebo at rest and immediately post-exercise (number of dogs = 6). Data are presented as mean ± SE. *L-Carnitine-treated Greyhounds demonstrated an elevated haematocrit at both rest and immediate post-exercise time points *versus* placebo-treated Greyhounds ($P < 0.05$)

3. *Reduce blood/plasma lactate levels* following intense exercise[8,13]. Although there is also evidence suggesting that a reduction in blood or plasma lactate might not be a universal finding following L-carnitine supplementation in humans[14], it is pertinent that the peak plasma lactate levels found post-exercise in the Greyhounds (~27 mmol l^{-1}) are far higher than reported for humans following maximal exercise (e.g. 8–12 mmol l^{-1})[8,13,14]. Moreover, the theoretical basis for L-carnitine reducing lactate production by exercising muscle has been challenged. Specifically, as mentioned in the introduction, L-carnitine supposedly enhances PDH activity (and therefore provides an increased oxidation of pyruvate and decreased lactate production) by constraining or preventing an increase in the acetyl CoA/CoA ratio[2]. However, the notion that PDH activity is dependent upon the acetyl CoA/CoA ratio is undermined by the fact that, during high intensity exhausting exercise, PDH activity increases simultaneously with the acetyl CoA/CoA ratio[25].

Potential for elevated oxygen delivery and utilization

In dogs[26], as in humans[27], there is sufficient muscle mitochondrial capacity to raise the maximal oxygen uptake when oxygen delivery is enhanced by elevated cardiac output (pericardectomy)[26] or arterial oxygen content review 20,21,27. Hence, if the 6% increase in systemic haematocrit found herein is not accompanied by a decrease in either haemoglobin oxygen saturation or reduced muscle blood flow, there should be an equivalent increase in maximal oxygen uptake. It is pertinent that a fall in cardiac output with polycythemia in exercising dogs does occur during submaximal exercise, but this is consequent to the increased oxygen content[31]. When methaemoglobin was used to prevent the polycythemia from increasing oxygen content, a 15% increase in systemic haematocrit did not elevate systemic vascular resistance. Thus, in the dog as for the horse[32], there is no reason to suspect that the increase in haematocrit found herein will cause any reduction in cardiac output during maximal exercise. Moreover, the elevated systemic haematocrit will likely lead to an increased lung and muscle oxygen-diffusing capacity that may actually accentuate the achievable maximal oxygen uptake above that attributable to increased convective oxygen delivery alone[32,33]. The present investigation did not demonstrate any improvement in running performance associated with the L-carnitine supplementation. However, elevated muscle oxygen delivery in other species is typically associated with improved muscular performance and hence future investigations, possibly on a larger scale involving actively competing Greyhound dogs, may be warranted.

Mechanism for reduced muscle damage

The muscle damage that results from high-intensity exercise, particularly that with a large eccentric component, has a complex aetiology. One putative scenario is detailed below. High-intensity exercise that stimulates the adenylate kinase reaction (2 ADP → ATP + AMP) leads to the oxidation of AMP to hypoxanthine. There is also an increase in intracellular calcium that activates calcium-dependent proteases which convert xanthine dehydrogenase to xanthine oxidase, which subsequently catalyses the formation of hypoxanthine to xanthine and converts it to uric acid during intense exercise[34]. Molecular oxygen is used as an electron acceptor for these reactions that produce the superoxide radical which, in turn, can combine with iron to form hydroxyl radicals and damage the polyunsaturated fatty acid component of the sarcolemmal membrane, causing cytosolic proteins such as CPK, AST and myoglobin to leak into the circulation. That inhibition of xanthine oxidase with allopurinol during exercise decreases reactive oxygen species generation, plasma concentrations of muscle proteins and decreases muscle damage[35] supports the participation of the above schema in exercise-induced muscle damage. L-Carnitine supplementation in humans has been demonstrated to impact several of the steps presented above. Specifically, L-carnitine reduces plasma hypoxanthine, xanthine oxidase, serum uric acid as well as plasma malondialdehyde, a product of lipid peroxidation[3].

Volek and colleagues[3] have considered that L-carnitine supplementation might exert at least part of its beneficial effect on free radical production and muscle damage by enhancing the oxidative regeneration of ATP and thereby limiting the availability of AMP for oxidation to hypoxanthine. The present investigation revealed an intriguing correlation between the increase in systemic haematocrit and decrease in plasma CPK that potentially supports this notion (Fig. 4). An appropriately designed prospective scientific evaluation of this hypothesis would be valuable.

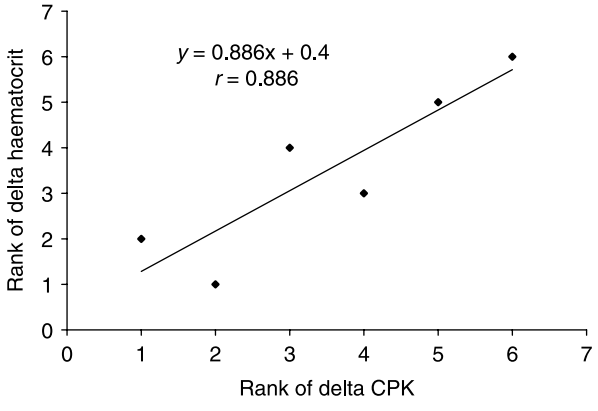

FIG. 4 Relationship between rank changes in haematocrit *versus* rank changes in creatine phosphokinase (CPK) in six Greyhound dogs treated with L-carnitine and placebo ($r = 0.89$; $P < 0.05$)

Conclusions

In racing Greyhounds, L-carnitine supplementation (at 100 mg kg^{-1} of body weight per day) significantly elevated systemic haematocrit and reduced peak plasma CPK concentrations ~70% (Fig. 1). The dose chosen for the current investigation was based on data published for equids and humans. The magnitude of the CPK rise suggested that extensive muscle damage is not incurred during standard racing conditions. However, there is the possibility that prevention or reduction of any such damage may help maintain peak Greyhound performance over the course of the racing season or multiple seasons. Moreover, carnitine supplementation is legal and will not interfere with drug testing. This question and the role of the L-carnitine-induced elevation of haematocrit in reducing muscle damage and potentially improving racing performance deserve to be evaluated in a larger-scale prospective study. No evidence was found for L-carnitine decreasing peak plasma lactate concentrations.

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