

The Reduction of Heat Production in Exercising Pigeons after L-Carnitine Supplementation

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ABSTRACT Four groups (CS,CR,PS,PR) of nine trained male racing pigeons were deprived of feed for 1 d and then subjected to a respiration chamber test in order to study the effect of oral l-carnitine supplementation on the energy metabolism during flight. One week before, groups CS and CR were orally supplemented with 90 mg of l-carnitine daily, whereas PS and PR were given a placebo. Groups CS and PS underwent flight

simulation by electrostimulation of the breast muscles. Flight simulation increased heat production, kept respiratory quotient from decreasing, decreased thyroxine levels, and increased weight loss. L-Carnitine decreased the rise in heat production during electrostimulation but did not influence respiratory quotient, weight loss, or thyroid hormones. L-Carnitine supplementation in pigeons improves fatty acid combustion efficiency during heavy exercise.

(Key words: pigeons, carnitine, exercise, heat production, thyroid hormones)

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INTRODUCTION

During races, pigeons prove to have a high endurance capacity. Being transported to the releasing site, they are deprived of feed for several days. Thereafter, they are still capable of performing a flight of more than 1,000 km. In the literature, l-carnitine has already been thoroughly investigated for its role in exercise performance: it enhanced aerobic and anaerobic metabolism in human athletes (Cerretelli and Marconi, 1990), horses (Souffleux, 1994; Foster *et al.*, 1989), and dogs (Grandjean *et al.*, 1993; Dubelaar *et al.*, 1994).

Several decades ago the main biochemical mode of action of l-carnitine was clarified. In the cytosol, l-carnitine binds fatty acid chains from acylated coenzyme A (CoA) by esterification. In contrast, with acyl CoA, the so-formed acylcarnitine can be transported through the inner mitochondrial membrane. Once present in the mitochondria, the acyl groups can be transferred from acylcarnitine to CoA, so that they can be combusted for adenosine triphosphate (ATP) production in the Krebs cycle (Fritz, 1955).

In adult vertebrates, the endogenous production of l-carnitine is sufficient in most cases, but particular situations, such as in certain diseases (Rebouche and

Paulson, 1986) and heavy exercise (Siliprandi, 1986), can lead to suboptimal levels of l-carnitine. Especially in racing pigeons, fatty acid combustion is of critical importance, as lipids are the main source of energy during flight (Bordel and Haase, 1993). By stimulating the inflow of acyl groups into the mitochondrial matrix, exogenous l-carnitine will probably indirectly support aerobic metabolism and thus prevent muscle fatigue.

Previous experiments with l-carnitine supplementation in pigeons have shown a lowering effect on several relevant blood parameters such as fatty acids, lactic acid, and creatinine phosphokinase (Borghijs and De Wilde, 1992). The aim of the present experiment was to investigate whether these significant biochemical changes would be accompanied by improvements in exercise physiology and changes in metabolic rate, and if so, how this effect evolved during exercise.

MATERIALS AND METHODS

Animals and Housing

For 2 mo, 36 male pigeons (*Columba livia domestica*) were fed a mixture of whole grains and seeds² and a fat pellet

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Abbreviation Key: ATP = adenosine triphosphate; coA = coenzyme A; CR = carnitine-supplemented pigeon at rest; CS = carnitine-supplemented pigeon under flight simulation; HP = heat production; PR = placebo-supplemented pigeon at rest; PS = placebo-supplemented pigeon under flight simulation; RQ = respiratory quotient.

TABLE 1. Total diet analysis

Ingredient	Composition	
	(%)	
Energy-Corn® ¹		20
Corn (<i>Zea mays</i>)		28
Pea (<i>Pisum sativum</i>)		18
Wheat (<i>Triticum aestivum</i>)		18
Sunflowerseed (<i>Helianthus annuus</i>)		1.6
Linseed (<i>Linum usitatissimum</i>)		0.2
Rape seed (<i>Brassica napus</i>)		0.2
Millet (<i>Panicum miliaceum</i>)		3.8
Canary seed (<i>Phalaris canariensis</i>)		0.2
Chemical composition of pickstone ^{1,2}		
Moisture		3
Crude ash		88
Calcium		30
Phosphorus		1.5
Sodium		2.8
Magnesium		0.06
Iron		0.1700
Cupric sulphate		0.0025
Manganese		0.0500
Zinc		0.0300
Iodine		0.0010
Cobalt		0.0005
Chemical composition of the mixture of ground shells and stones ^{1,2}		
Moisture		0.3
Crude ash		96.3
Calcium		13.5
Phosphorus		0.03
	Total	Energy-Corn®
Chemical composition		
Moisture	10.95	12
Crude ash	2.73	7.5
Crude protein	13.77	18.8
Crude fat	8.04	17.0
Crude fiber	4.75	4.4
Nitrogen-free extract	59.76	40.3
Apparent ME, kcal/g	3.34	3.30

¹Versele-Laga Ltd., B-9800 Deinze, Belgium.

²Offered for *ad libitum* consumption and was not part of seed diet per se.

supplement (Energy Corn®)² (vol/vol 5:1) (Table 1). A solid mineral supplement and ground stones and shells² were available for *ad libitum* consumption. Apparent metabolizable energy content (kilocalories) was calculated by means of the formula of the European Union for poultry: 37.1 CP + 82 CF + 40 St + 31.1 Su, where CP is grams of crude protein, CF is grams of crude fat; St is grams of starch; and Su is grams of sugars (Larbier and Leclerq, 1992). Feed allotment, which was based on average intakes from earlier experiments involving similar conditions, averaged 27 ± 2 g (SE). The pigeons were housed in four identical group cages of nine individuals. The cages were 150 cm wide, 225 cm long, and 220 cm high, so that they were large enough to fly in. A passageway connected this cage to an outside wired cage,

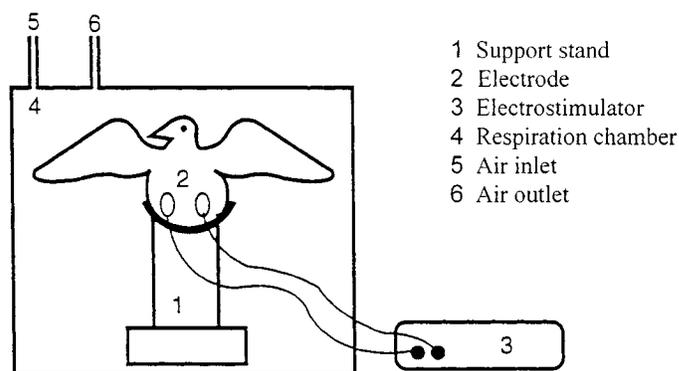


FIGURE 1. The experimental setup of the flight simulation in the respiration chambers. The wires of the electrodes were guided through a special hole in the respiration chamber which was made air-tight by silicone. CS, α l-carnitine-supplemented, flight-simulated; CR, l-carnitine-supplemented, rested; PS, placebo-supplemented, flight simulated; PR, placebo-supplemented, rested.

(90 cm wide, 100 cm long, and 60 cm high), which was freely accessible.

Experimental Procedure

The pigeons were divided into nine groups of four animals each. Each group started at a different moment, but the relative time schedule was the same for each group. All animals had to perform three exercise flights of about 50 km during the last 2 wk of the 2-mo experimental period. The last 7 d were used to give daily oral supplements of l-carnitine or placebo. Two pigeons per group received an intubation of 90 mg of l-carnitine³ dissolved in 0.5 mL distilled water. The other two animals were supplemented in the same way with a placebo solution of 0.5 mL distilled water. At the end of the 2-mo period, the pigeons were transported in the morning by car in a special pigeon basket with individual compartments for approximately 80 min (85 km) to another laboratory. There, the basket with the pigeons was put in a dark, ventilated room. Following acclimatization, at about 1800 h, the pigeons were placed in individual respiration chambers and respiration measurements were started. Each pigeon was attached to a support stand by means of crosswise bandages in order to enable head and wings to move freely. The support stands were designed as shown in Figure 1. One carnitine-supplemented pigeon and one placebo-supplemented pigeon were prepared for a flight simulation (CS and PS, respectively). The other two pigeons in each trial, one carnitine-supplemented, one placebo-supplemented, were used as controls at rest (CR and PR respectively). For the flight simulation, a self-adhesive gel electrode⁴ was attached to the skin of each of the two breast muscles (*Pectoralis*), and connected to an apparatus for electrical stimulation.⁵

The next morning at 0830 h, a flight simulation experiment was executed in the respiration chambers. The period between the start of the respiration measurements

³Lonza Ltd., CH-4002 Basel, Switzerland.

⁴Versa-Stim®, Conmed Corp., Utica, NY 13501.

⁵Fysiomed, B-2650 Edegem, Belgium.

and the flight simulation test was referred to as Hour 0. The two pigeons with electrodes attached were electrically stimulated for 6 h at a pulsating current of 20 V with a frequency of five pulses per second, each 2 ms per pulse, forcing the birds to flap their wings.

Before and after the respiration chamber test, the pigeons were weighed individually and blood was taken. Blood was sampled from the femoral vein of the left leg and allowed to drip into heparinized plastic tubes. The samples were centrifuged immediately at $4,000 \times g$ for 5 min and plasma was frozen at -20 C until analyzed. During transport, no feed, water, or supplements were provided in order to simulate transport conditions for gaming pigeons. Evidence for suffering was not found, except for one pigeon from the PS group that was not willing to fly during the first 2 d after the flight simulation test. Housing and experimental procedures were subject to Ministry of Agriculture control: license numbers LA1400084.

Indirect Calorimetry

Respiratory quotient (RQ) and heat production (HP) were measured in individual open circuit respiration chambers designed for small animals.⁶ A detailed technical description of the respiration chambers as well as the calculation of the HP according to the formula of Romijn and Lokhorst (1961) is given elsewhere (Buyse *et al.*, 1997): HP (kilocalories per hour) = 3.88 O_2 consumption (liters per hour) + 1.20 CO_2 production (liters per hour). Heat production was expressed on a metabolic body weight basis (kilograms^{0.75}). Respiratory quotient was calculated as the ratio of volume of CO_2 produced:volume of O_2 consumed.

Plasma Analysis

Plasma triiodothyronine (T_3) and thyroxine (T_4) levels were measured by RIA using commercial antisera⁷ and ^{123}I - T_3 and ^{123}I - T_4 .⁸ Parallel protocols have been described for T_3 by Darras *et al.* (1990) and for T_4 by Darras *et al.* (1991). Standard solutions were prepared in hormone-free human serum.

Preliminary Experiments

To check the effect of fixation of the pigeons in the respiration chambers, a preliminary experiment was set up whereby four pigeons were put in the respiration chambers without fixation and compared with two pigeons fixed as described above.

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⁷Byk-Sangtec GmbH, D-63128 Dietzenbach, Germany.

⁸Amersham International, HP7 9LL Buckinghamshire, UK.

⁹SPSS for Windows 6.0®, SPSS Inc., Chicago, IL 60611.

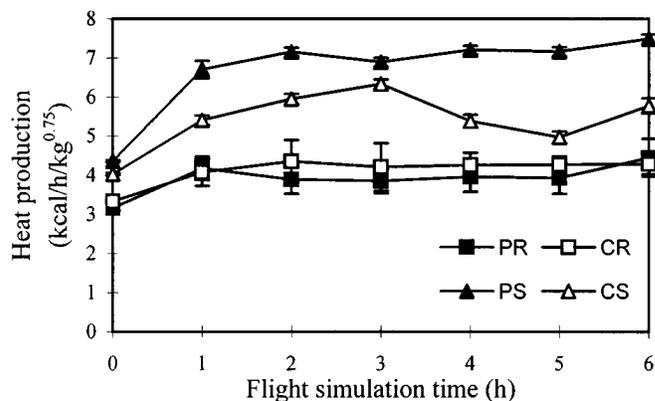


FIGURE 2. Heat production during the flight simulation test. Error bars represent pooled SEM values. The number of observations differs per hour because of removed outliers: the Hours 0 (the acclimatization period), 1, 2, 3, 4, 5, and 6 have $n = 1,513, 154, 149, 165, 177, 183,$ and $156,$ respectively. CS, α -l-carnitine-supplemented, flight-simulated; CR, l-carnitine-supplemented, rested; PS, placebo-supplemented, flight simulated; PR, placebo-supplemented, rested.

Data Handling and Statistical Analysis

Statistical analysis was performed with SPSS for Windows 6.0.⁹ Hourly HP and RQ data were analyzed with the Repeated Measures ANOVA procedure of SPSS with l-carnitine supplementation, flight simulation, and individual as between-subjects factors and the seven consecutive hourly pools from flight simulation onset to termination as within-subjects variables. Body weight loss and thyroid hormone levels were analyzed by General ANOVA procedure of SPSS with l-carnitine supplementation, flight simulation, time (before or after the flight simulation test), and individual as main factors. The factor "individual" was, in fact, a combination of individual and trial. The model is: $Y_{ijkl} = \mu \dots + A_i + B_j + (c/AB)_k + D_l + (AB)_{ij} + (AD)_{il} + (BD)_{jl} + (ABD)_{ijl} + r_{(ijkl)}$, where A = l-carnitine supplementation ($i = 1, 2$); B = flight simulation ($j = 1, 2$), c = individual ($k = 1, 2, 3, 4, 5, 6, 7, 8, 9$); and D = time ($l = 1, 2, 3, 4, 5, 6, 7$ for HP and RQ; $l = 1, 2$ for the other dependent variables). Factors were tested against residual variation. Linear regression analysis was performed to describe the evolution of the hourly averages of RQ in time from Hours 0 to 6. Factor means were tested with the Bonferroni method (Neter *et al.*, 1990).

RESULTS

Heat Production

In the preliminary test, heat production tended to be lower in the pigeons without fixation, so it was concluded that it would be necessary to restrict all pigeons during the further experiments, whether they were subjected to flight simulation or not. The differences were not significant because of the small sample size.

As shown in Figure 2, HP rose significantly due to electrical stimulation of the *Pectoralis*, compared to the

TABLE 2. Body weight and plasma thyroid hormone levels before and after the flight simulation test¹

Variable	Pigeons at rest		Flight-simulated pigeons		Pooled SEM	P value
	Placebo	L-Carnitine	Placebo	L-Carnitine		
Body weight, g ¹						
Before test	490 ^a	515 ^a	500 ^a	534 ^a	6] <0.001
After test	466 ^b	492 ^b	473 ^b	510 ^b	6	
Difference	24 ^a	23 ^a	27 ^a	24 ^a	1	
Plasma triiodothyronine (T ₃), ng/mL ¹						
Before test	1.17 ^a	1.24 ^a	1.10 ^a	1.30 ^a	0.07] 0.001
After test	0.90 ^b	0.97 ^b	0.98 ^b	1.02 ^b	0.05	
Difference	0.27	0.27	0.12	0.28	0.07	
Plasma thyroxine (T ₄), ng/mL ¹						
Before test	11.7	8.0	8.8 ^a	9.3 ^a	0.51] <0.001
After test	9.7	8.2	4.9 ^b	4.1 ^b	0.61	
Difference	2.0 ^a	-0.2 ^a	3.9 ^b	5.2 ^b	0.89	
Plasma T ₃ :T ₄ ¹						
Before test	0.11	0.19	0.13 ^a	0.15 ^a	0.03] 0.035
After test	0.10	0.14	0.26 ^b	0.27 ^b	0.03	
Difference	0.01 ^a	0.05 ^a	-0.13 ^b	-0.12 ^b	0.02	

¹Means of nine pigeons.

groups at rest ($P < 0.001$). Mean HP at rest was not significantly different between the treatment groups and remained at 3.53 ± 0.17 kcal/h/kg^{0.75} (SEM; $n = 1,513$). At rest, l-carnitine supplementation did not cause significant differences, whereas for the pigeons subjected to flight simulation, significantly lower HP values were detected in the CS pigeons than in the PS pigeons ($P = 0.006$).

Respiratory Quotient

During the respiration chamber test, RQ showed a tendency to decrease in the pigeons at rest (Figure 3) but not during the flight simulation ($P < 0.001$): the RQ values of the stimulated groups started at 0.83 and remained at 0.82 during stimulation, whereas the RQ values in the groups at rest decreased from 0.82 to 0.79. No influences of l-carnitine supplementation on RQ were detected.

Body Weight Loss

Average initial body weight before start of the respiration chamber test was 505 ± 5 g (SEM; $n = 36$; Table 2). No carnitine effect was present, but for all pigeons body weight loss occurred during the respiration chamber test.

Thyroid Hormones

For both RIA, the dilution series of pigeon plasma with RIA buffer showed good parallelism with the standard curve (Table 2). By adding increasing amounts to pigeon plasma (loading tests), it was also proved that the measurement of both hormones in the pigeon plasma was accurate. Intra-assay variabilities were 4.5, and 5.4% for T₃ and T₄, respectively.

Plasma T₃ did not significantly vary with any of the treatments but was lower at the end of the respiration

chamber test [0.99 ± 0.29 ng/mL $\bar{x} \pm SE$; $n = 36$] than at the moment before the respiration chamber test [1.30 ± 0.38 ng/mL ($\bar{x} \pm SE$; $n = 36$)] ($P < 0.001$). The relative body weight loss per individual was significantly correlated by 33% with the relative decrease in plasma T₃ ($P = 0.047$). Plasma T₄ did not differ due to carnitine supplementation, but significantly lower levels of T₄ were found in pigeons subjected to flight simulation. The effect of flight simulation on thyroid hormone metabolism was also obvious when T₃:T₄ ratios were considered: in the groups at rest, the T₃:T₄ ratio was slightly decreased after the respiration trial, whereas the opposite was true for the pigeons subjected to flight simulation.

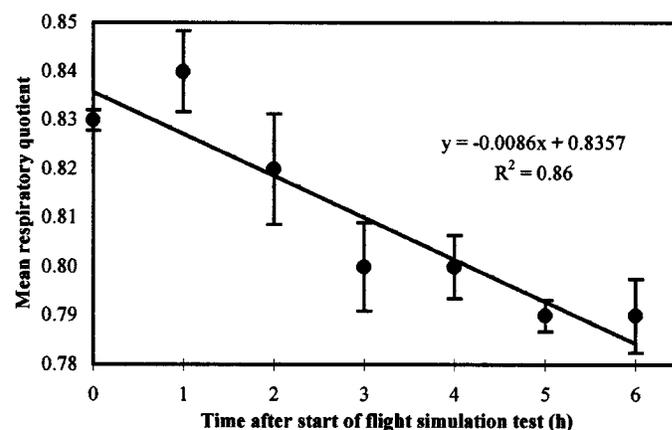


FIGURE 3. Overall hourly means of respiratory quotient during the respiration chamber test. Error bars represent pooled SEM values. The number of observations differs per hour because of removed outliers: the Hours 0 (the acclimatization period), 1, 2, 3, 4, 5, and 6 have $n = 1,513$, 154, 149, 165, 177, 183, and 156 respectively. Other than linear approaches did not show major improvements of estimations. CS, l-carnitine-supplemented, flight-simulated; CR, l-carnitine-supplemented, rested; PS, placebo-supplemented, flight simulated; PR, placebo-supplemented, rested.

DISCUSSION

In a number of publications dealing with different animal species, respiratory parameters other than HP were influenced by l-carnitine supplementation (Ceretelli and Marconi, 1990; Peres, 1993). Concerning HP, very few reports relating to l-carnitine supplementation can be found. Other respiratory parameters, such as maximal oxygen flow, vary as a consequence of different setups or different species or both when looking at the influence of l-carnitine supplementation (Peres, 1993).

In the current study, l-carnitine supplementation clearly prevented the increase in HP during thorough exercise. Pigeons being subjected to flight simulation are on a thin line between aerobic and anaerobic metabolism. The maximal benefit of l-carnitine is early in the fatigue protocol (Brass *et al.*, 1993). As the l-carnitine effect was already present at an early stage of the flight exercise, the present data confirm the effect of l-carnitine at a rather early stage of muscle fatigue.

Although the effect on some of the parameters could have been even more pronounced in untrained pigeons, we preferred to train the pigeons to be sure to measure the supplementary effects of l-carnitine under practical conditions, i.e., with trained pigeons. Former experiments have shown that repetitive exercise of untrained pigeons influenced fatty acid metabolism in a similar way as l-carnitine supplementation (Gevaert *et al.*, 1991). In the literature, we have found evidence for a similar site of action of training and l-carnitine supplementation. Roncero and Goodridge (1992) demonstrated the synergic impact of l-carnitine on the T₃-induced accumulation of mRNA of fatty acid synthase and malic enzyme. Malic enzyme catalyses the conversion of malate to pyruvate. As malate also takes part in the citric acid cycle, where it is the precursor of oxaloacetate, the latter is probably decreased by malic enzyme. Oxaloacetate, in turn, is an inhibitor of succinate dehydrogenase. This enzyme accelerates the citric acid cycle so that a higher capacity for aerobic acyl CoA combustion is created.

Besides l-carnitine supplementation, training also stimulates succinate dehydrogenase, and, in an experiment of Angelini *et al.* (1986), this led to hypertrophy of the type I muscle fibers, by which muscle force can be improved (Dubelaar *et al.*, 1991).

Because our pigeons were trained and there was still an additional effect of L-carnitine on HP, a second mode of action of L-carnitine should be considered. As mentioned above, training as well as l-carnitine supplementation enlarges the capacity for aerobic combustion. This higher capacity can only be utilized if the flow of acetyl groups into the citric acid cycle can be increased. Indeed, exogenous l-carnitine is able to transport acyl groups from acyl CoA in the cytosol to CoA in the mitochondria. After fatty acid oxidation in the mitochondria, acetyl CoA can be combusted (Fritz, 1955). According to Brass *et al.* (1993), this action of

exogenous l-carnitine is not limited by the capacity of carnitine palmitoyltransferase because this enzyme has a rather high Michaelis constant for l-carnitine. Because the electrically forced exertion was similar for all pigeons, the changes in HP can only be explained by changes in energy utilization. On the one hand, improvement of energy efficiency can be stated as a consequence of favored aerobic metabolism. For example, glucose in the aerobic pathway gives rise to 36 ATP molecules, whereas in the anaerobic pathway only 2 ATP molecules can be formed. This difference means that in the anaerobic pathway more energy gets lost as heat than with aerobic metabolism. The anaerobic metabolization of fatty acids is prevented by l-carnitine administration; it lowers the acyl CoA:CoA ratio. A high ratio normally inhibits the pyruvate dehydrogenase enzyme. Due to the l-carnitine enhanced activity of the enzyme, more pyruvate will enter the citric acid cycle instead of being converted to lactate. (Brevetti *et al.*, 1988). This hypothesis is supported in former flight simulation experiments in which plasma lactate levels were lower in the l-carnitine supplemented pigeons (Borghijs and De Wilde, 1992). On the other hand, acyl CoA accumulates in the cytosol when fatty acid metabolism is stimulated, as during feed deprivation and long-term exercise. Exogenous l-carnitine can prevent this accumulation, so that the inhibition of adenylate translocase by acyl CoA diminishes. This enzyme enhances the ADP-dependent oxygen uptake (Bobyleva-Guarriero *et al.*, 1985). In this way, supplementation of l-carnitine promotes aerobic metabolism. The prevention of high acyl CoA concentrations in the cytosol by l-carnitine also prevents cell membrane damage (Brevetti *et al.*, 1988), as indicated by decreased plasma levels of creatine phosphokinase in electrostimulated pigeons when given l-carnitine (Borghijs and De Wilde, 1992). This result proves the muscle protecting ability of l-carnitine during heavy exercise.

Values of HP and RQ of the pigeons at rest were similar to those in studies of Gorssen *et al.* (1993) when compared on a metabolic body weight basis. To start the stimulation procedure, a person had to enter the darkened room in which the respiration chambers were installed. The increase in HP during the 1st h of flight stimulation test in the birds at rest is suggested to be a consequence of this disturbance. Values of RQ at the end were not as low as in the experiments of Gorssen *et al.* (1993) during water deprivation, but a decline in RQ was present in our tests.

One can expect that, due to starvation for more than 1 d, energy from the digested feed gets depleted so that body reserves have to be used. Knowing that for birds the average RQ for carbohydrate, protein, and fat is, respectively, 1.00, 0.67 (instead of 0.83 for mammals), and 0.71 (Kleiber, 1961), the unchanged or slightly decreasing RQ values in our experiment show the shift towards fatty acid combustion during feed deprivation. Racing pigeons switch very easily to fat as an energy

source during exercise (Bordel and Haase, 1993); thus, it can be expected that mainly deposited fat will be utilized for providing energy when birds are starved for at least 1 d. In humans, RQ shifted from 0.96 (l-carnitine supplemented) and 0.97 (placebo supplemented) to 0.79 and 0.78, respectively, during heavy exercise (Cerretelli and Marconi, 1990). Similar findings were obtained in pigeons by Parker and George (1974) during long-term stimulation.

Thus, it is suggested that flight simulation has a lowering effect on RQ. Nevertheless, we saw that flight simulation countered the starvation-enhanced RQ decrease. In the case of mammals, an explanation would be that body protein could also be used at the end of flight simulation but as birds excrete uric acid rather than urea, the combustion of protein gives an RQ value that is theoretically similar to that of fatty acids (0.67 instead of 0.83) (Kleiber, 1961). Therefore, the present result could be due to a depletion of glycogen reserves from muscle and liver during exercise rather than fat mobilization as occurs in starvation.

Plasma T_3 and T_4 were reduced due to starvation and T_4 was also decreased by electrostimulation, causing an increase in the ratio $T_3:T_4$. A completely identical change in the plasma thyroid hormone levels was found in experiments of George *et al.* (1989). This increase in the $T_3:T_4$ ratio can be explained by a higher rate of peripheral conversion of T_4 to T_3 in the flight-stimulated animals. The conversion is to a large extent carbohydrate-dependent, suggesting the mobilization of carbohydrates in the exercising animals. In the present experiment, the lesser decrease in RQ due to flight stimulation, despite the higher HP, agrees with this model.

The only effect on T_3 in the current study was a decrease due to starvation, which was accompanied by a loss of weight. This result was not surprising, as the T_3 -decreasing effect of starvation has been shown in different species, such as the rat (Rondeel *et al.*, 1992), chicken (Kühn *et al.*, 1991), and human (Webber and Macdonald, 1994). In the latter experiments, weight loss and T_3 were correlated, which was also found in our experiment.

The pigeons lost more weight when subjected to flight simulation, which is trivial, but no effect was seen for l-carnitine supplementation. In former experiments with a long period of l-carnitine supplementation, weight loss was not noted in pigeons at rest compared to controls (Janssens and De Wilde, 1995). Thus, l-carnitine does not seem to induce weight loss, as is sometimes incorrectly extrapolated from findings in other species and under other conditions.

Further research can bring insights as to the exact physiological carnitine status during exercise, which can be used as a tool to detect shifts in substrate utilization and the eventual impact of l-carnitine on endocrine regulation of metabolism. Many pigeon fanciers use doping agents to try to stimulate the flying performance

of their animals. These products, most of them belonging to the corticosteroids or the testosterone-like drugs, are often noxious to the animals (Dhondt *et al.*, 1993; Duchatel *et al.*, 1993) and prohibited by Belgian law (Bourgeois, 1995). Thus, l-carnitine can be proposed as an alternative supplement to pigeon nutrition to enhance flight results.

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