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Letter to the editors

Carnitine administration as a tool of modify energy metabolism during exercise

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Sirs:

In an article by Veechiet et al. (1990) in this journal, it was shown that carnitine administration increased both maximal oxygen uptake and power output during exercise at maximal intensity. There was also a decreased oxygen uptake, carbon dioxide production, pulmonary ventilation and a reduction of plasma lactate after ingestion of carnitine compared to placebo during trials with similar exercise intensities. This effect was attributed to the carnitine supplied acting as a transporter of free fatty acid (FFA) and as an acceptor of acetyl groups formed by pyruvate oxidation, i.e. the carnitine worked as an acetyl group buffer. This would, according to the authors, have enhanced pyruvate utilization by lowering the acetyl-coenzyme A/coenzyme A quotient and thereby releasing the inhibition of pyruvate dehydrogenase.

However, the dose of L-carnitine was 2 g, taken orally 90 min before the start of the cycling exercise. From earlier studies of resorption of carnitine, it is known that maximal plasma carnitine concentrations after a 2-g dose were seen in individuals after 3 to 9 h (Harper et al. 1988), and in another study the peak was reached after 3.5 h (Bach et al. 1983). The mean increase in plasma concentration was 12 $\mu mol \cdot kg^{-1}$ after 2 h, range 7–20, representing approximately 50% of the peak value (Harper et al. 1988). Furthermore, the oral bio-availability of carnitine was low, a mean value of 13%, range 8%–21%, was found during the first 12 h after a single 2-g (12.4 mmol) L-carnitine dose. This would mean that the maximal available amount in the body of the given dose (12.4 mmol carnitine) would be 1.6 mmol. A more probable amount is less than 50%, or 0.8 mmol.

As the effects of the carnitine supplementation described were related to metabolic processes in muscle tissue, the amount of the carnitine confined to the muscle tissue must have been of critical importance. The muscle mass is 40% of the body mass in normal man and would correspond to 28 kg in a 70-kg subject. The carnitine dose given would, if rapidly and equally distributed into the muscle tissue, result in a maximal increase of 30–60 $\mu mol \cdot kg^{-1}$. The median content of carnitine in human vastus lateralis muscle has been found to be 17.9 $mmol \cdot kg^{-1}$ dry mass, range 6.5–24.1, i.e. 4300 $\mu mol \cdot kg^{-1}$ wet mass, range 1500–5700 $\mu mol \cdot kg^{-1}$ (Cederblad et al. 1974).

At rest the acetylcarnitine in muscle corresponded to about 17% of the total carnitine store but increased to 40%–90% of the store during heavy exercise. This corresponded to an acetyl group uptake by carnitine of about 2000 $\mu mol \cdot kg^{-1}$ wet muscle mass (Harris et al. 1987). The carnitine dose given by Veechiet et al. (1990) would have increased the pre-exercise muscle carnitine content by about 1–2%, an increase which could hardly change the capacity for FFA transport or acetyl group uptake.

Thus, in our opinion, it is difficult to explain the results presented in the paper by Veechiet et al. (1990) as a biochemical alteration of muscle metabolism attributable to the supplementation of carnitine.

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