

action for explaining the ergogenic effects of caffeine. Doherty postulates that there are other central and peripheral elements involved. A probable reason for these ambiguities may be related to the training level of the subjects.

The difference in the type of applied tests for measuring power (cycling, running, swimming) and the degree of caffeine consumption (5-6mg/kg and 250 mg) probably have led causing of this contradictory of the results. Therefore, the more precise and exceeding researches are necessary in order to be aware of the effect of caffeine consumption on 400-m freestyle activities.

Another major finding of this investigation was that administration of 1.5 grams of L-carnitine did not affect on Performance time and Blood lactate concentrations.

From the previous data, we find that CAF can induce an increase of FFA in blood and spare glycogen in the liver and muscle (LeBlanc, Jobin *et al.*, 1985), and CAR can increase fat utilization by the transport of long-chain fatty acids into the mitochondrial matrix for β -oxidation (Broquist & Borum, 1982).

The main function of Carnitine is to stimulate fatty acid production in mitochondria and reduce lactate production (Siliprandi *et al.*, 1990).

In a review study (Matera *et al.*, 2003) also demonstrated that reduced lactate production during exercise is another function of L-Carnitine supplementation. In an investigations (Lee, Paik *et al.*, 2003) also reported that 4g per day of Carnitine causes significantly lowers the blood lactate concentration. Increased fatty acid oxidation decreases fatigue upon Carnitine activation, and especially, lactate accumulation caused peripheral fatigue is decreased (Siliprandi *et al.*, 1990). There is higher acetyl-carnitine concentration than acyl-carnitine in skeletal muscle during high intensity exercise (Constantin-Teodosiu, Carlin, Cederblad *et al.*, 1991; Minkler, Brass, *et al.*, 1995). This high level

of acetyl-CoA accumulation suppresses pyruvate dehydrogenase activation and affects acetyl-carnitine and lactate accumulation. Eventually, Carnitine intake has a positive effect on lactate concentration. However, a study (Chun Y, 2008) reported that 2g per day of Carnitine might not have a positive effect on lactate accumulation in Judoists. This implies that there is a close relationship between the amount of Carnitine intake and lactate recovery based upon subjects' characteristics.

In a study_ (Colombani, *et al.*, 1996) has previously been reported acute administration of L-carnitine did not affect the metabolism or improve the physical performance of the endurance-trained athletes during the run and did not alter their recovery.

In a study they (Brass, Hoppel & Hiatt, 1994) found That Carnitine administration had no effect on muscle total carnitine content or the workload-dependent accumulation of acylcarnitines in skeletal muscle. Carnitine had no effect on the respiratory exchange ratio, muscle lactate accumulation, plasma lactate concentration, muscle glycogen utilization, or plasma beta-hydroxybutyrate concentration during exercise. Thus the skeletal muscle carnitine pool is segregated from dramatic changes in the plasma carnitine pool, and short-term administration of carnitine has no significant effect on fuel metabolism during exercise in humans. In a research (Jacobs, Goldstein, *et al.*, 2009) examine the effects of a single dose of GPLC on the performance of repeated high intensity stationary cycle sprints with limited recovery periods in resistance trained male subjects and find that short-term oral supplementation of GPLC can enhance peak power production in resistance trained males with significantly less LAC accumulation.

In the present study, perhaps the acetyl CoA/CoA ratio in the muscle was not influenced, ultimately not disrupting the accumulation of lactate. Further controlled experiments at varying intensities need to be performed to determine the potential benefits of L-carnitine supplementation upon performance.