

The Effect of Carnitine Supplementation on Apoprotein A and B in Obesity Males During Exercise Training

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Abstract—Aim: this study performed with aim to investigation effect oral L-carnitine L-tartrate ingestion on Apoprotein A and Apoprotein B concentration during a standard ergometry cycling. Method: sixteen adult obese males performed above mentioned ergometry protocol while ingested L-carnitine as experimental group or oral lactose as control group. Our study performed in two trails: firstly, ergometry test without L-carnitine or lactose ingestion. Secondly, ergometry test after L-carnitine or Lactose ingestion. Blood samples were drawn immediately followed up exercise for the purpose of determination Apoprotein A and Apoprotein B, LDL and HDL concentration. Result: the finding of paired T student statistically indicated that L-carnitine ingestion had no influence on Apoprotein A and Apoprotein B ($p < 0.05$). These finding also observed in control group. Discussion: Our study finding indicated that L-carnitine L-tartrate ingestion, 3g for 2h before exercise could not affect the effective variables in fat metabolism. Additional investigation is required to directly identify the effect of these supplementations on the substrate utilization and fat-carbohydrate metabolism and exercise performance.

Keywords—ergometry cycling, L-carnitine L-tartrate, Apoprotein

I. INTRODUCTION

Apolipoprotein B (apoB) concentration and age are independently associated with an increased risk for cardiovascular disease (1). Carnitine is a naturally occurring compound that can be synthesized in mammals from the essential amino acids lysine and methionine or ingested through diet. Primary sources of dietary carnitine are red meat and dairy products; however, commercially produced supplements also are available and have been shown to be safe in humans. Carnitine supplementation enhances fatty acid oxidation during exercise and, hence, spares glycogen (2). The increases in fatty acid oxidation and plasma free fatty acid concentration suggested a glycogen-sparing effect of carnitine supplementation (3). Conflicting results characterized the research focused on L-carnitine supplementation's ability to enhance endurance performance. Matera (2003) stated that L-carnitine supplementation led to

reduction of lactate production during exercise (4). But, Eroğlu (2008) show that L-carnitine intake one hour prior to the exercise has no effect on the metabolic and blood lactate values of badminton players(5). Despite this strong foundation and 20 years of research, no compelling evidence exists that carnitine supplementation can improve physical performance in healthy subjects. Therefore, this study was performed with purpose to determine the effect of chronic carnitine supplementation on aerobic capacity, rest and submaximal heart rate, plasma Lactate and glucose concentration during submaximal ergometry cycling.

II. MATERIAL AND METHOD

Eighty untrained obese male volunteered and were randomly divided into experimental and placebo groups. The subject ingested during 3 wk supplementation periods, with either 3g L-Carnitine L-tartrate ($n=15$) or 3g Lactose ($n=15$) daily in experimental and placebo groups. Before and after of supplementation periods, the all subjects performed, ergometry cycling tests according to Astrand submaximal protocol on cycle for twenty minute. Blood samples were drawn immediately followed up exercise. The samples were assayed for the concentrations Apoprotein A and B according to the established procedures. Rest and submaximal heart rate monitored by polar telemetry. Maximal oxygen consumption calculated by the formula of Astrand protocol. Data are reported as means \pm standard deviation. A two-way repeated measure ANOVA was used to determine significant differences between the two groups. A value of $P < 0.05$ was considered to be significant.

III. RESULT

The finding of our study showed that L-carnitine supplementation had no influence on plasma glucose concentration (figure 1). Also plasma Lactate concentration remained unchanged after chronic L-carnitine supplementation (figure 2). In addition, rest and submaximal heart rate (figure 3), VO_{2max} (figure 4) and LDH activity was equal in pre and posttest ($P < 0.05$). All variables unaffected with the placebo trial ($P < 0.05$).

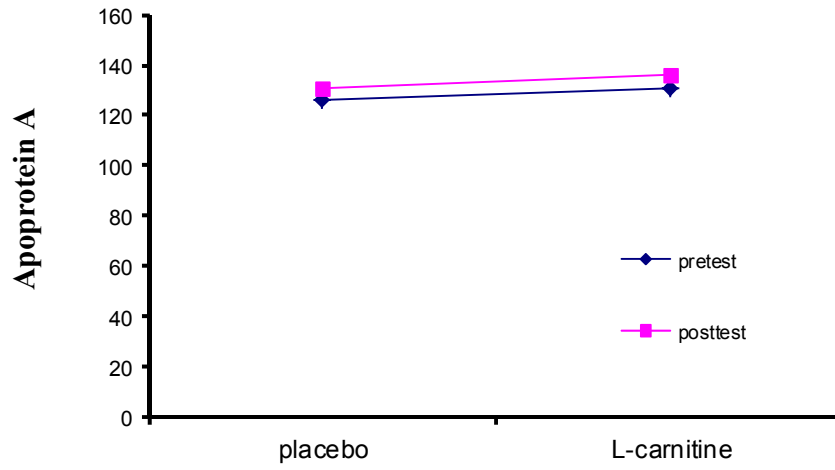


Figure 1. The changes pattern of plasma apoprotein A concentration in pre and posttest of study groups

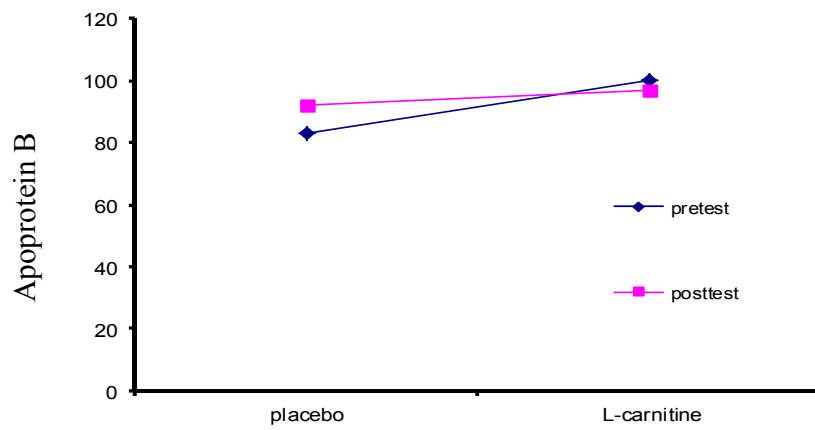


Figure 3. The changes pattern of plasma Apoprotein B Concentration in pre and posttest of study groups

IV. DISCUSSION

L-Carnitine has been reported to have a beneficial effect on several cardiovascular risk parameters, including plasma lipids and lipoprotein (a) (6). Researchers have argued that administration of carnitine may shift the metabolic bias of the liver away from etherification and synthesis of triglycerides toward the formation of acetylcarnitines.

Lipoprotein (a) [Lp(a)] concentration is generally related to coronary artery disease (CAD) and cerebrovascular disease. Some studies strongly suggest that l-carnitine may have a role among lipid-lowering. The results obtained of a study showed that the L-carnitine treatment tends to restore to normal values both the chemical composition of lipoproteins and the apoproteins pattern of rats fed on a diet enriched with cholesterol. It is still a matter of debate whether the administration of L-carnitine improves performance of endurance exercise. Our finding indicated that L-carnitine ingestion, 3g for 2h before exercise could not affect the effective variables in fat metabolism. Additional investigation is required to directly identify the effect of these supplementations on the substrate utilization and fat-carbohydrate metabolism and exercise performance. Oral ingestion of carnitine would result in an increase of the total carnitine concentration in muscle. This increase in muscle carnitine would result in an increased rate of oxidation of intramuscular fatty acids and triacylglycerols during exercise, thereby reducing muscle glycogen breakdown and postponing fatigue (7).

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