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# The influence of medium-chain triglycerides supplementation in ultra-endurance exercise performance

Antonio Marcio Domingues Ferreira[1], Paula Edila Botelho Barbosa[1] and Rolando Bacis Ceddia[2]

## ABSTRACT

The ultra-endurance competitions represent a great challenge in the world of sports. The energetic cost of an ultra-endurance event can vary from 5,000 to 18,000 kcal a day. Because of this great demand, many strategies to improve performance have been developed during the last years, like the medium-chain triglycerides (MCT) supplementation in combination with carbohydrates (CBO). The goal of MCT supplementation is to increase the free fatty acids (FFA) utilization as energy source, sparing the body glycogen to the end of the competition. When compared to long-chain triglycerides (LCT), MCT are absorbed faster and transported through the body. Besides that, MCT have a speed of oxidation comparable to CHO, but, since they are lipids, they provide a greater amount of energy when oxidized. Therefore, MCT seem to be the ideal fuel for long-term events. To conclude, the aim of this revision is to elucidate how MCT can influence performance in ultra-endurance exercises.

**Key words:** Ultra-endurance exercise. Medium-chain triglycerides. Performance.

## INTRODUCTION

The interest on and the participation in sports competition have significantly increased over the past years, and there is no question that ultra-endurance or long-duration competition are the most appealing ones[1].

In this type of competition there are the super- and ultra-marathons (from 84 km on), the Ironman Triathlon (3.8 km swimming; 180 km cycling; and 42 km running), contests that last more than 24 hours, such as the Ultraman Triathlon (10 km swimming; 421 km cycling; and 84 km running), cycling competitions that last up to 30 days (*Tour de France*, *Vuelta Ciclista a España*, *Giro d'Italia*, *Race Across America*), and, most recently, the Adventure Races, which include a number of "extreme sports".

The energetic expenditure in "ultra-endurance" competitions may vary from 5,000 kcal (triathlon with 2 km swimming; 90 km cycling; and 21 km running) up to 18,000 kcal (a 24-hour race)[2]. The average energetic expenditure at the *Tour de France* is estimated to be 6,500 kcal/day, reaching 9,000 kcal/day at the mountains. The energetic expenditure in a 5-day, 1,000 km ultra-marathon is of 59,079 kcal in average, with daily expenditure ranging from 8,600 and 13,770 kcal[3]. In a study carried out in Rio de Janeiro with participants of a triathlon competition (3.8 km swimming; 180 km cycling, and 42 km running) the energetic expenditure was estimated in 8,171.1 kcal ± 716.7[4]. To face such high energetic demand, different strategies to improve performance have been developed. Recently, medium-chain triglycerides (MCT) supplementation combined or not with carbohydrates (CHO) has been investigated.

MCT supplementation is intended to optimize the use of free fatty acids (FFA) as source of energy, and spare endogenous glycogen reserves for the final stages of the competition. It is suggested that the ability to sustain exercise may be enhanced with the increase of lipid supply[3,5].

Thus, the purpose of such review is to clarify how MCT may influence performance in long-duration contests.

## METABOLISM AND MCT OXIDATION

MCT are apolar molecules, formed by three saturated fatty acids, with six to 12 atoms of carbon esterified by glycerol. The fatty acids (FA) that form MCT are: caprylic (C8:0; 50-80%), capric (C10:0; 20-50%) acids, and a smaller proportion of caproic (C6:0; 1-2%) and lauric (C12:0; 1-2%) acids. MCT is the main type of fat present in the human diet, and was introduced in the clinical practice about 50

1. Curso de Pós-Graduação em Fisiologia do Exercício e Avaliação Morfo-Funcional. Universidade Gama Filho. Rio de Janeiro, RJ.
2. Department of Biology. York University. Toronto, Canadá.

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**Correspondence to:**
Antonio Marcio Domingues Ferreira
Rua Pontes Corrêa, 147, casa 4 – Andaraí
20510-050 – Rio de Janeiro, RJ
Phone: (21) 2278-4837
E-mail: amdf28@terra.com.br

years ago, for both the treatment for lipid absorption dysfunctional and as source of energy, to replace long-chain triglyceride (LCT) diets[6,7].

The MCT, which are rich in medium-chain fatty acids (MCFA), are hydrolyzed by pancreatic lipase, and are absorbed in the duodenum more rapidly than the long-chain fatty acids (LCFA)[3,7-10].

MCFA are a swift source of energy, as, differently from LCFA, they are not significantly incorporated into lipoproteins (kilomicrons and VLDL), being directly absorbed in the blood stream. The velocity MCFA is absorbed in the intestine is similar to the one of glucose. After passing by the enterocytes, these FA reach the portal flow, and are transported to the liver bound to albumine. The albumine binding to MCFA is weaker than to LCFA. On the other hand, part of MCFA is directly solubilized in the aqueous plasma fraction[3,7-9].

The high intake of MCFA-rich triglycerides (TG) leads to a significant intracellular incorporation of such FA in kilomicrons, as re-synthesized TG. This incorporation seems to depend on the amount of intake and MCFA distribution in the ingested TG. However, the amount of kilomicrons produced when there is a high intake of MCFA-rich TG is approximately 1/5 of the seen in patients with high intake of LCFA-rich TG, suggesting that incorporation of MCFA in kilomicrons is not the most favorable way of absorption[11].

The transport of lipids in the body is typically described in two metabolic ways: exogenous and endogenous. The exogenous way is to transport the ingested lipids from the intestine to the liver. The endogenous way is to transport lipoproteins synthesized in the hepatocytes from the liver to peripheral tissues.

In the post-absorption stage, MCFA plasmatic transport is eased due to albumine binding, and through the portal vein they rapidly reach the liver[12].

### MCT destination in the body

MCT digestive bioavailability is higher than LCT's. Compared to LCT's, MCT hydrolysis, which starts in the stomach, is more swift and thorough, velocity in gastrointestinal transit is higher and absorption takes place in the proximal portion, being more rapid and effective. The chain length FA plays a major role in their behavior in the body. The LCFA leave the intestine as LCT through lymphatic vessels after incorporation in the kilomicrons. A fraction of these kilomicrons undergo intra-vascular hydrolysis, releasing most LCFA to extra-hepatic tissues, whereas the remaining fraction is transported to the liver. The LCFA reach the liver as albumine-bound FA or as TG. On the other hand, the free MCFA, after digestion, go to the liver through the portal vein, weakly bound to albumine. About 80% to 100% of MCFA present in the entire portal flow are captured by the liver, and the remainder follows in the blood stream, becoming available to peripheral tissues[3,7-10].

All FA use both transportation systems in variable proportions. The higher FA's carbonic chain, the more it is found in lymph and less in portal blood. In lymph vessels, they circulate as TG associated to kilomicrons. In the portal blood, FA are bound to albumine. Thus, 8% of the MCFA are associated to kilomicrons three hours after intake of a meal with MCT in healthy individuals. Having such a meal for six days, this amount reaches 15%. In previous studies, the metabolic "destination" of MCFA would be restricted almost exclusively to the liver[13].

A factor that interferes in the distribution of dietary lipids in the tissue is the presence of lipoproteic lipase (LPL). This enzyme is located in the endothelium of vessels that irrigate the muscles, the main site of FA oxidation, and in adipose tissue, the most important site for their storage. The TG capture rate is proportional to LPL activity in the tissue, which is high in the skeletal muscle on fasting, and in the adipose tissue at the post-prandial period. Thus, LPL directs FA towards oxidation (in the muscle) or to be stored as TG (in the adipose tissue). These processes are basically related to LCFA, which are transported associated to kilomicrons or VLDL, and less to MCFA, which are little incorporated in these lipoproteins[13].

### FA distribution between cytoplasm and mitochondria

Due to LCFA hydrophobic properties, binding proteins are necessary to transport them from the membrane to target-organelles. The LCFA easily bind to these proteins, an essential step to reach enzymatic site and be activated as acyl-coA. On the other hand, the water-soluble MCFA weakly bind to these proteins, which explains the weak conversion rate of these FA in acyl-coA[13,14].

An additional carrying mechanism takes place in the inner mitochondrial membrane. Carnitine, acting in the enzyme carnitine palmitoltransferase (CPT), plays a crucial role in LCFA transportation. It was later seen that the entry of MCFA in the mitochondrial matrix is not dependent on carnitine. But such is a partial independence. In the liver, about 10 to 20% of octanoate is transported as acyl-carnitine, whereas in the muscle, MCFA entry in the mitochondria is totally dependent on the transporting activity of carnitine[3,7,10,15].

### MCFA oxidation

MCFA oxidation occurs in all tissues, particularly in the mitochondria. Even though their activation may also take place in the cytoplasm, it occurs basically in the mitochondrial matrix, where a specific acyl-coA is synthesized from

the MCFA. Oxidation rate of MCFA is higher and swifter than LCFA's. The preference for MCFA oxidation is kept even in obesity[13].

One of the most important MCT properties is its ketogenic character, once a significant portion of acetyl-coA abundantly produced during MCFA oxidation is directed towards production of ketonic bodys. A single 45 to 100 g MCT dosage P.O. given to healthy individuals raises plasma concentration of ketonic bodies at 7,000 umol/L over a 1- to 2-hour interval. These values are two- to four-fold higher than those seen in individuals fed with LCT-rich diets. Thus, serum concentrations of ketonic bodies in healthy subjects are of 150 umol/L; after a 48 h-fast, of 25,500; and in uncontrolled diabetics, higher than 10,000[13].

**Overall regulation of MCFA metabolism**

The liver is highly capable to oxidize and synthesize FA, modulated according to body needs or pathophysiological conditions. The regulatory mechanisms take place in two stages, basically.

In the first, the CPT enzyme, located in the outer mitochondrial membrane, which converts acyl-coA in acyl-carnitina, controls the FA mitochondrial oxidation rate. In the second stage, malonyl-coa, a molecule involved in the "FA *de novo* synthesis" (an exclusive LCFA via, negatively regulated when a low-lipid diet is replaced by other enriched with these FA) inhibits, in physiological concentration, acyl-coa, and therefore FA entry and oxidation in the mitochondria. As malonyl-coa inhibits CPT, there is an increase in FA synthesis. As they are relatively independent from carnitine, the MCFA scape from this LCFA-controlling mechanism. Once in the mitochondrial matrix, β-oxidation is an almost exclusive outcome of all FA, regardless of the size of the chain. This explains why MCFA oxidation is little influenced by nutritional or hormonal factors in the body, exactly the opposite that happens with LCFA[13,14,16,17].

Furthermore, MCT tend to slightly raise serum insulin levels, and promote lipogenesis as a result from an increase in insulin/glucagons balance[13].

## MCT AND EXERCISE

The CHO are the energetic substrate for long-duration aerobic activities, but the body organic reserves of glycogen are limited, and may be totally depleted in athletic events of this nature. Thus, it may be advantageous to optimize lipid use (FFA) as source of energy, sparing the glycogen reserve for the final stages of the competition[1,18].

It has been suggested that the ability to sustain exercise may be extended if the supply of lipids is immediately increased prior to the exercise, as the FFA oxidation rate is directly related to their serum concentration levels[3,5,9,15,16,19].

Contrary to this statement, Martin III (1997)[20] mentions that during running or cycling, intra-muscular TGs would be the main source for increase of FA oxidation.

The use of lipids as source of energy during long-duration exercises is very important, as they, reserved in the body as TG in adipose tissue (± 17,500 mmol), skeletal muscle (± 300 mmol), and in plasma (± 0.5 mmol), are the main organic energy storage (± 560 MJ), being up to 60 times higher when compared to glycogen (± 9 MJ)[18,21]. Another important factor is the amount of energy provided by the oxidation of lipids (9 kcal/g), whereas glucose supplies less (4 kcal/g)[22].

Considering such advantages of lipids as source of energy, the use of MCT as supplement has been broadly studied.

As previously mentioned, MCT do not delay gastric emptying, and are more quickly absorbed in the gut than LCT, being transported to the liver by the blood. As the metabolization speed of MCT is similar to the one of glucose, they seem to be the ideal source of energy for long-duration exercises[8,9,18,23].

In order to compare MCT and CHO oxidation rates, Decombaz *et al.* (1983)[24] carried out an investigation in which 12 subjects were submitted to a stimulation of one hour in a cycle-ergometer (60% $\dot{V}O_2$ max), one hour after a standard MCT or CHO meal (± 250 kcal). Oxidation during a period of two hours after the meal was of 30% and 45% (MCT and CHO respectively) of the total ingested. The decrease of glycogen level in the vastus lateralis muscle, assessed in a biopsy, was the same after both meals. The authors thus concluded that a simple MCT or CHO meal before exercising, when glycogen reserves are within normal limits, do not change CHO utilization nor spare organic glycogen during one hour of submaximal exercise.

In order, still, to compare MCT and CHO oxidation rates, Massicote *et al.* (1992)[25] carried out an investigation in which six healthy male subjects completed five two-hour stimulation in cycle-ergometer at 65% of $\dot{V}O_2$ max, with a seven-day interval between them, in the following way: one stimulation-control, with intake of water, two stimulations with intake of 25 g of MCT prior to the exercise, and two stimulations with intake of 57 g of CHO (diluted in one liter of water) during exercise. Over the two hours of exercise, 13.6 ± 3.5 g of MCT and 36.4 ± 8.2 g of CHO were oxidized, which was respectively 54% and 64% of the total ingested. The energetic contribution from MCT and CHO was not significantly different, being, respectively, 7% and 8.5% of the total energetic expenditure.

Jeukendrup *et al.* (1998)[8] also mention that oxidation of exogenous MCT is increased when they are ingested associated to CHO.

## MCT AND PERFORMANCE IMPROVEMENT

As previously mentioned, MCT may be an important source of exogenous energy for endurance exercises. However, studies have shown that MCT alone would not improve performance so much, and some investigations were carried out associating intake of MCT and CHO, with conflicting results.

Jeukendrup *et al.* (1995)[26] submitted eight well-trained cyclists to four 180-minute stimulation at 57% of $\dot{V}O_2$ max, in which each athlete ingested 4 ml/kg of body weight at the beginning of the exercise, and 2 ml/kg during exercise, of the following: 15% of CHO; 149 g of CHO + 29 g of MCT; 214 g of CHO + 29 g of MCT; or 29 g of MCT. At the end of the study, it was observed that a higher amount of MCT was oxidized when ingested in association with CHO, confirming the idea that MCT may be used as a source of energy combined with glucose during exercise, as their metabolic availability was higher during the last hour of the exercise, with oxidation rates reaching 70% of the ingestion rate.

Still with the purpose to check MCT oxidation rate, Jeukendrup *et al.* (1996)[27] carried out a similar study, in which eight elite athletes were submitted to four 90-minute cycle-ergometer sessions (57% $\dot{V}O_2$ max). The athletes ingested two solutions before and during exercise, one with CHO (15%) only, the other, CHO + MCT. Even though total lipid oxidation has markedly increased, MCT oxidation had marginally increased, with a small contribution to the total energetic expenditure, of about 6-8%.

The conflicting outcomes from these two investigations may be explained by the significantly different times of stimulation (90 and 180 minutes). Thus, it seems logical to think the longer stimulation would cause a higher amount of MCT to be oxidized.

As to performance improvement, Van Zyl *et al.* (1996)[28] evaluated six trained cyclists, who performed, at three different occasions with 10 days apart from each other, two-hour stimulation at 60% of $\dot{V}O_2$ max, and immediately after each stimulation, a 40 km time trial session. During each of the stimulation sessions, the athletes ingested three different solutions: with 10% of CHO; or 4.3% of MCT; or 10% of CHO + 4.3% of MCT. At the end of the study, the authors observed that in the stimulation when MCT + CHO was ingested, the time trial session was smaller compared to the other sessions, and that the session in which MCT only was ingested was longer than when CHO only was ingested. In this study, the amount of supplemented MCT during stimulation calls attention, which was equivalent to 86 g. Such an amount goes against the findings of other authors, who mentioned that amounts higher than 30 g of MCT would cause gastrointestinal discomfort and diarrhea[5,8,23].

In a study similar to Van Zyl's, Jeukendrup *et al.* (1998)[29] gave the same amounts of MCT, associated to CHO or not. There were no performance-related positive findings, and when only MCT was ingested, there was a decrease in performance, related to gastrointestinal discomfort reported by the athletes.

Goedecke *et al.* (1999)[30] carried out a study to assess gastric symptoms, energetic metabolism, and performance, with nine cyclists submitted to three 2-hour stimulation followed by a 40 km time trial session, in which solutions of CHO (10%), CHO + MCT (10% + 1.72% or 10% + 3.44%) were given: 400 ml before stimulation, and 100 ml at every 10 minutes during exercise. The subjects did not report any gastrointestinal discomfort, and MCT intake did not affect energetic metabolism or performance. The authors reported that plasma FFA and beta-hydroxybutyrate levels were high after MCT intake.

In order to assess the effect from CHO and CHO + MCT intake in metabolism and performance, Angus *et al.* (2000)[31] evaluated eight athletes who covered 100 km as fast as they could in cycle-ergometer. The solutions taken at each 15 minutes (250 ml) were of CHO 6%, or CHO 6% + MCT 4.2%, or placebo. The results showed that CHO intake during exercise increased performance, but adding MCT did not lead to a performance improvement.

In 1996, Jeukendrup *et al.*[32] carried out a study to assess whether there would be **a** difference in CHO oxidation rate during exercise, when MCT was ingested. Nine trained athletes were assessed in four 180-minute sessions at 57% of $\dot{V}O_2$ max, when they ingested 4 ml/kg of body weight at the beginning of each session, and 2 ml/kg at each 20 minutes during exercise, of the following solutions: 150 g/L of CHO; an association of equal calories of 70% CHO + 30% (29 g) MCT or 150 g of CHO + 20 g of MCT; and a placebo (control) solution. Before and after sessions muscle biopsies were made to assess the amount of glycogen, and breathing samples were collected during exercise to measure exogenous and endogenous CHO oxidation rates. No differences among the sessions were observed regarding exogenous and endogenous oxidation. FFA plasma levels were high during exercise, being similar in all the sessions, whereas plasma ketone levels significantly increased after MCT intake. The authors concluded that the intake of 29 g of MCT associated to CHO during 180 minutes of exercise does not influence the utilization of CHO or glycogen.

Thus, in spite of MCT oxidation rate be increased when they are ingested with CHO, the intake of amounts up to 30 g of MCT does not seem to spare muscle glycogen or enhance performance; as for amounts higher than 30 g, in spite of conflicting data, most authors state that such

amounts would cause gastrointestinal discomfort and diarrhea[5,8,10,21,23].

In all studies, the fact that stimulation does not take more than three hours calls attention. According to Noakes (2001)[33], MCT supplementation seems to be more effective in activities that take five hours or longer, which could explain why most studies did not see performance improvement.

In 2001, Misell *et al.*[34] carried out an investigation to assess effects of chronic MCT intake, in which 12 trained runners used dietary supplements with 56 g of LCT or 60 g of MCT daily, for two weeks. After each supplement stage, the subjects took a treadmill test that included two sessions: a 30 minute one, at 85% of $\dot{V}O_2$ max, immediately followed by another, at 75% of $\dot{V}O_2$ max until exhaustion. The results showed that chronic MCT intake did not improve performance nor significantly changed performance-related metabolism in trained runners.

An interesting aspect related to MCT supplementation was investigated by Kern *et al.* (2000)[35]. The purpose of the study was to check the behavior of serum lipid levels. Trained runners received a low-lipid diet and then had to ingest, twice a day for two weeks, 30 g of MCT or 28 g of LCT. There was a three-week interval between the two stages. At the end of each stage, blood samples were collected, and total cholesterol (TC), HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), and triglycerides (TG) were measured. In spite of TC, HDL-C, LDL-C and TG levels being higher after MCT-intake stage, all serum lipid levels were within normal values, and there was no significant HDL-C difference between the two stages. The authors concluded that MCT intake for two weeks negatively changes the profile of serum lipid in athletes, and recommend further studies to assess the effects of MCT intake over a longer period of time on serum lipids.

## CONCLUSIONS AND RECOMMENDATIONS

MCT supplementation for ultra-endurance exercises does not seem to promote a performance enhancement that would support its use. In spite of the MCT oxidation rates increase when ingested with CHO, this does not seem to spare the glycogen organic reserves or improve performance. Among the studies reviewed for this article, only one found an improvement in performance with MCT intake (associated to CHO), and the amount of supplemented MCT (86 g) was higher than the recommended by the literature (30 g), what could cause gastrointestinal discomfort and diarrhea.

The use of MCT as a supplement requires further studies, that utilize, if possible, a longer stimulation time (from five hours on), and intermediate MCT concentrations, of about 50 to 60 g. It is also recommended that studies assessing chronic MCT use on serum lipid levels and the health of athletes would also be carried out.

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