

The effect of medium-chain triacylglycerols on the blood lipid profile of male endurance runners

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Medium-chain triacylglycerol (MCT) oil is currently marketed for athletes as an ergogenic aid for optimal performance. Research assessing the blood lipid response of humans to MCT consumption is very limited and inconclusive. In this randomized cross-over study, male endurance runners (aged 30.5 ± 5.5 years) were instructed to consume a low-fat diet (approximately 15% of energy) and consume either supplemental MCT oil (30 g twice each day) or long-chain triacylglycerol (LCT) oil (28 g corn oil twice each day) for 14 days. Each dietary trial was separated by at least 3 weeks. At the end of each trial, fasting blood samples were collected and analyzed for serum concentrations of total cholesterol (TC), high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), and triacylglycerol (TG). Concentrations of TC (3.83 ± 0.12 vs. 3.41 ± 0.15 mmol/L, $P = 0.004$), LDL-C (1.76 ± 0.12 vs. 1.51 ± 0.14 mmol/L, $P = 0.033$), and TG (1.26 ± 0.14 vs. 0.98 ± 0.12 mmol/L, $P = 0.006$) were higher following the MCT trial than following the LCT trial, respectively. HDL-C concentration did not differ significantly between trials (MCT 1.48 ± 0.05 mmol/L vs. LCT 1.45 ± 0.04 mmol/L, $P = 0.465$). Although blood lipids remained within desirable ranges established by the National Cholesterol Education Program, these results suggest that consumption of MCT oil for 2 weeks negatively alters the blood lipid profile of athletes. Future studies should determine the effects of longer periods of MCT supplementation on serum lipids of exercisers and other groups of individuals. With little data suggesting that MCT are ergogenic, the adverse effects of MCT on blood lipid concentrations may outweigh any proposed benefits for athletes. (J. Nutr. Biochem. 11:288–292, 2000) © Elsevier Science Inc. 2000. All rights reserved.

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Introduction

Medium-chain triacylglycerols (MCT) are structured lipids, consisting primarily of C8:0 and C10:0 medium-chain fatty acids (MCFA). MCT have been used in the diets of patients with malabsorption disorders since the 1950s due to their ease of digestibility and absorption.¹ MCT are now sold at health food stores, gyms, nutritional supplement stores, and some grocery stores. Most retail products containing MCT are marketed either to dieters as weight loss aids due to their thermogenic properties² or to athletes as ergogenic aids due to theoretical performance-enhancing effects.³ Well-de-

signed human studies suggest that acute consumption of MCT does not provide ergogenic benefits^{4,5}; however, a study of mice demonstrated improved endurance performance after supplementation of MCT for at least 2 weeks at approximately 17% of dietary energy intake.⁶

Fat loading in general has also been studied for its ergogenic potential. Although the performance-enhancing effects of short-term carbohydrate consumption before and during exercise are well established, some research has suggested that long-term adaptation to a diet high in fat may prolong endurance.^{7,8} These findings may promote higher fat consumption in endurance athletes. Determining which fats are most healthy is critical. Recent research suggests that chronic consumption of a diet high in fat, even saturated fat, does not adversely affect the blood lipid profile of athletes.^{9,10} However, this has not been demonstrated in all studies.¹¹ MCT oil is widely available to athletes, yet little is known about the effects of this source of saturated fatty acids on the blood lipid profile.

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Several studies have reported a reduction in serum total cholesterol (TC) concentration following ingestion of MCT compared with corn oil in rats.^{12–15} Conversely, research conducted by Newport et al.¹⁶ indicated that 26 days of MCT supplementation raised blood concentration of TC in pigs. Moreover, Woollett et al.¹⁷ detected no change in serum concentration of TC, low density lipoprotein cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) following 30 days of MCT supplementation in hamsters.

Research on the effects of MCT on blood lipids of humans is limited and equivocal. Beveridge et al.¹⁸ investigated the effects of a variety of saturated fats (i.e., butterfat, coconut oil, MCT oil) on serum TC after 8 days of feeding at 30% of total energy intake. Butterfat and coconut oil raised serum TC concentration; however, MCT oil had no effect. Hegsted et al.¹⁹ also detected no change in serum TC concentration in humans. More recently, Hill et al.²⁰ reported no change in concentrations of TC and HDL-C in the blood following 6 days of MCT administration at 40% of total energy intake. Additionally, Swift et al.²¹ demonstrated that 6 days of MCT feeding (40% of total energy intake) did not alter serum concentration of either TC or LDL-C and lowered HDL-C concentration. Conversely, recent research by Tso et al.²² indicated that 4 weeks of feeding MCT (14% of total energy intake) raised serum TC concentration in subjects possessing the apo E 3/3 ($n = 11$) and apo E 4/3 ($n = 3$) phenotypes. However, that study detected no change in TC in subjects possessing the apo E 2/3 ($n = 3$) phenotype and no effects of MCT feeding on LDL-C or HDL-C in any group.

Elevation of serum triacylglycerol (TG) concentration by MCT feeding has been consistently demonstrated in both animal^{16,23,24} and human^{2,20,21} research models. MCT consumption appears to enhance fatty acid elongation¹⁴ and increase de novo fatty acid synthesis.¹³ These changes may stimulate TG production and very low density lipoprotein (VLDL) secretion and likely account for the elevation in serum TG concentration.¹²

The purpose of this study was to determine the effects of consuming a diet supplemented with MCT versus long-chain triacylglycerol (LCT; corn oil) for 2 weeks on blood lipid concentrations in male runners. Serum TC and LDL-C concentrations were expected to be lower following MCT consumption whereas HDL-C was predicted to be similar between trials. Serum concentration of TG was expected to be higher following the MCT trial than following the LCT trial.

Methods and materials

Subjects

Twelve endurance trained males volunteered for the study. All respondents were asked a series of questions to determine if they met the selection and exclusionary criteria. The selection criteria were as follows: (1) male, (2) aged 18 to 40 years, and (3) currently running 30+ miles weekly. Exclusionary criteria included cigarette smoking, heart disease, metabolic disturbance diseases, chronic use of medications known to alter normal metabolism, and a weight of 20% above or 15% below desirable body weight according to the Metropolitan Life Insurance Tables.

Methods were reviewed and approved by the San Diego State University Committee on Protection of Human Subjects prior to recruitment. A statement of informed consent was signed by all subjects prior to their participation.

Study design

This study was randomized, double-blinded, and cross-over in design. Subjects participated in two dietary intervention trials conducted for 2 weeks each. During these trials, subjects were instructed to maintain their normal training regimens, eat a low-fat diet (approximately 15% of kcal from fat), drink two fat supplement shakes [containing either 28 g corn oil (LCT) or 30 g MCT oil] each day, and keep food records for 3 days during each week of the 2-week trials. MCT [C 6:0 (<6%), C8:0 (67%), C10:0 (23%), and C > 12:0 (<4%)] were provided by Mead Johnson & Company (Evansville, IN USA). A washout period of at least 3 weeks was employed between trials to allow for normalization. During the washout period, subjects were instructed to return to normal eating patterns.

Prior to initiating the dietary trials (1–2 weeks before), subjects were instructed how to reduce their dietary fat intake, consume supplement shakes, and complete food records. Subjects were asked to train and eat similarly during the two dietary trials. At that time weight, height, and body composition were determined. Body composition was measured in triplicate by hydrostatic weighing corrected for residual volume using the oxygen dilution method.²⁵ Maximal oxygen consumption (VO_{2max}) was assessed using a graded treadmill protocol performed to exhaustion.⁷

On the day following each dietary trial, subjects reported to the laboratory in the morning between 7:00 AM and 8:00 AM after a 12-hour overnight fast. Subjects were weighed and sat quietly for 10 min before collection of resting blood samples. They were instructed to abstain from exercise and consumption of alcohol for at least 24 hr prior to blood collection.

Dietary protocol

Subjects consumed two fat supplement shakes each day during the 2-week dietary trials. Each shake consisted of 2.5 g liquid soya lecithin (providing emulsification), 250 mL nonfat milk, one packet (20.4 g) “no sugar added” Carnation® Instant Breakfast (CIB), and either 30 g MCT oil or 28 g LCT as corn oil. Shakes were provided to the subjects fully prepared. The shakes were isocaloric and provided 12.9 g protein, 24.1 g carbohydrate, and 2.8 g fat, in addition to the MCT oil or corn oil. Subjects were instructed to consume low-fat diets in order to achieve a total fat intake of approximately 30% of kcal. Food records were analyzed using Nutritionist IV (First DataBank, San Bruno, CA USA).

Biochemical parameters

Blood samples were drawn by venipuncture from the antecubital region. Blood was collected in a Vacutainer SST tube with gel separator and clot activator. Blood was allowed to clot at room temperature for approximately 30 min and then centrifuged at $1,500 \times g$ for 10 min at 2 to 8°C. Serum was separated and stored at -70°C for future analysis. Serum TG and TC were assessed enzymatically with a kit from Sigma Diagnostics (St. Louis, MO USA). HDL-C was measured using a dextran sulfate precipitating technique followed by enzymatic cholesterol analysis using a kit from Sigma Diagnostics. LDL-C was calculated using the equation of Friedewald et al.²⁶

Statistics

Descriptive data and biochemical data are expressed as means \pm SD and means \pm SEM, respectively. Paired-samples *t*-tests were

Table 1 Subject characteristics ($n = 12$)

Variable	Mean	SD
Age (yr)	30.5	5.2
Body fat (%)	8.9	2.4
Lean body mass (kg)	62.0	4.7
VO _{2max} (mL/kg/min)	72.6	7.6

VO_{2max}—maximal oxygen consumption.

used when comparing body weights, dietary intake, and biochemical data between dietary trials. An alpha level of $P < 0.05$ was selected as the criterion for statistical significance.

Results

Twelve well-trained competitive endurance runners completed the study. All subjects had been endurance trained for at least 2 years. Subjects' characteristics are presented in *Table 1*. Subjects ranged in age from 23 to 40 years and reportedly ran from 30 to 70 miles per week (40.0 ± 14.6 miles/week) at the time of recruitment. Subjects reported no differences in weekly training regimen throughout the study. The weekly running mileage reported and the mean VO_{2max} suggest that the subjects were highly trained.

Dietary intake was similar ($P > 0.05$) between trials. Subjects consumed $2,790 \pm 166$ vs. $2,932 \pm 196$ kcals/d, 116 ± 6 vs. 116 ± 8 g/d of fat, 346 ± 26 vs. 381 ± 34 g/d of carbohydrate, and 104 ± 7 vs. 102 ± 9 g/d of protein for the MCT and LCT trials, respectively. One subject reported failure to consume two shakes during the MCT trial. All others reported consuming all shakes during both dietary trials; however, on two occasions, a subject reported forgetting to consume a shake on one day, but drank it as well as the other shakes on the following day. Body weights were similar ($P > 0.05$) following both the MCT (68.8 ± 4.8 kg) and LCT (68.8 ± 5.2 kg) dietary trails.

Biochemical data are presented in *Figure 1*. Serum concentrations of TC (3.83 ± 0.12 vs. 3.41 ± 0.15 mmol/L, $P = 0.004$), LDL-C (1.76 ± 0.12 vs. 1.51 ± 0.14 mmol/L, $P = 0.033$), and TG (1.26 ± 0.14 vs. 0.98 ± 0.12 mmol/L, $P = 0.006$) were higher following the MCT trial than following the LCT trial, respectively. HDL-C concentration did not differ significantly between trials (MCT 1.48 ± 0.05 mmol/L vs. LCT 1.45 ± 0.04 mmol/L, $P = 0.465$). The TC:HDL-C ratio was higher ($P = 0.025$) following consumption of the MCT (2.37 ± 0.13) supplement shakes than the LCT (2.63 ± 0.15) shakes. A significant difference between the MCT (1.05 ± 0.11) and LCT (1.22 ± 0.10) trials was not detected for the LCL-C:HDL-C ratio, although the difference between trials approached statistical significance ($P = 0.057$).

Discussion

MCT have attracted the attention of researchers for their possible application to the diets of athletes due to their ease of absorption, rapid metabolism, and possible glycogen sparing properties.³ Early animal research suggested that MCT are hypolipidemic^{12–15}; however, more recent human

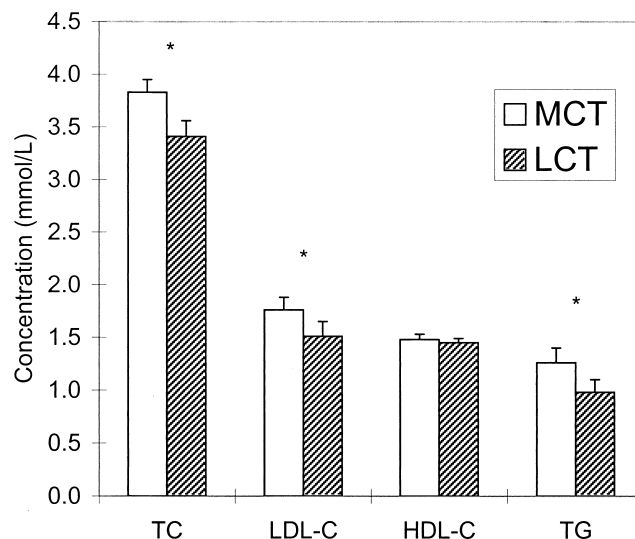


Figure 1 Blood lipids following consumption of diets supplemented with medium-chain triacylglycerol (MCT) oil or corn oil ($n = 12$). Significant differences ($P \leq 0.05$) between trials are indicated by *. LCT, long-chain triacylglycerol; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; TG, triglycerides.

research has provided conflicting reports.^{18–22} This study is the first to examine the effects of 2 weeks of MCT consumption on serum lipid profiles of exercisers. Results of this cross-over, placebo-controlled, randomized double-blind study do not support the hypothesis that MCT consumption would favorably influence the blood lipid profile of trained endurance runners. Serum concentrations of TC and LDL-C were significantly higher following the MCT trial than following LCT trial. Although HDL-C concentration in the serum was similar between the two dietary regimens, the TC:HDL-C ratio was higher following MCT administration and a trend toward a higher LDL-C:HDL-C ratio was detected for the MCT trial. As expected, serum TG concentration was also higher following MCT supplementation. Despite the observed elevations in TC and LDL-C, concentrations of these lipids remained within the desirable limits established by the National Cholesterol Education Program. Although the coronary risk profile based on these lipids remained within desirable ranges, this study suggests that MCT may raise relative risk for cardiovascular disease as assessed by the blood lipid profile in comparison to LCT. Furthermore, although not assessed in this study, it is likely that differences of this magnitude would be of greater consequence for people consuming MCT as a weight loss aid when blood lipids may be less desirable before supplementation.

An explanation for the elevations in serum concentrations of TC and LDL-C has been presented by Newport et al.¹⁶ Those authors suggested that although the liver oxidizes MCFA more rapidly than long-chain fatty acids (LCFA), the uptake of fatty acids by the liver during MCT feeding may exceed the liver's capacity for catabolism, resulting in their elongation to LCFA. To transport these newly synthesized LCFA as lipoproteins from the liver, an increase in the biosynthesis and secretion of cholesterol and

phospholipids must occur.²⁷ Such effects would be consistent with the higher concentrations of TC and LDL-C during the MCT trial than during the LCT trial.

The findings of the present study are consistent with those of Tso et al.,²² which suggest that MCT feeding raises serum TC concentration. In that study a diet that included 14% of total energy intake as MCT was fed to 18 premenopausal women for 4 weeks. MCT consumption raised TC concentration in subjects possessing the apo E 3/3 ($n = 11$) and apo E 4/3 ($n = 3$) phenotypes but had no effect on TC concentration in subjects possessing the apo E 2/3 ($n = 3$) phenotype. No statistically significant effects of MCT on LDL-C or HDL-C were observed. Although the results of MCT feeding appear to be dependent on apo E polymorphism, the authors concluded that MCT are hypercholesterolemic for some individuals.

The hypocholesterolemic effect of MCT feeding observed in many studies¹²⁻¹⁵ contradicts the results of the present study. The lipid-lowering effect detected in those studies may be due to a depression in β -hydroxy β -methyl glutaryl coenzyme A (CoA) reductase activity.¹⁵ Another explanation suggested for the hypolipidemic effect of MCT is that an increase in acetate utilization for fatty acid elongation may divert the acetate from the pathway of cholesterol synthesis.¹⁴ Studies detecting a hypocholesterolemic effect have been conducted on rats. The contradiction in results between the present study and those studies may be due to a difference in research models.

The majority of previous research supports the current study, which suggests that dietary intake of MCT does not alter serum HDL-C concentration. Hill et al.²⁰ reported no significant change in HDL-C following 6 days of MCT feeding. Moreover, a study by Tso et al.²² detected no difference among healthy females in HDL-C following a MCT (C8:0 and C10:0) diet for 4 weeks, regardless of apo E phenotype.

Few studies have reported significant effects of MCT ingestion on HDL-C concentration; however, Swift et al.²¹ observed a 15% decrease in HDL-C following MCT ingestion compared with soybean oil ingestion. The researchers suggested that MCT feeding may lower HDL-C due to a reduced chylomicron TG production. Because HDL-C was not altered by dietary intake in the current study, these results are not supportive.

The higher serum TG concentration following MCT consumption in the present study is consistent with previous animal^{16,23,24} and human studies.^{2,20,21} MCT supplemented diets may increase de novo fatty acid synthesis¹³ and enhance fatty acid elongation¹⁴ activity by the liver. Geelen et al.¹² determined that MCT feeding increases the activities of acetyl-CoA carboxylase, fatty acid synthase, and diacylglycerol acyltransferase. These changes would be expected to increase hepatic TG production and VLDL secretion. This may account for the elevated fasting serum TG concentration. These findings are also supported by previous data showing little or no increase in plasma TG immediately after MCT feeding, suggesting that elevated fasting TG are probably not of chylomicron origin.²

No significant differences were observed in consumption of energy, total fat, protein, or carbohydrate between dietary trials; thus, it is unlikely that the subject's self-selected diets

confounded the results of this study. Subjects were instructed to consume a diet consisting of approximately 15% of energy intake from fat. Actual reported fat intake, excluding fat supplement shakes, was $21.2 \pm 2.0\%$ and $23.1 \pm 1.9\%$ of kcal for MCT and LCT trials, respectively. Therefore, fat consumption from foods was similar between trials and likely did not have a major influence on the results.

Although the increase in TG during the MCT dietary trial was expected, this study is one of few to demonstrate higher serum TC and LDL-C concentrations following MCT supplementation. These findings suggest that consuming MCT for 2 weeks negatively affects the blood lipid profile of endurance athletes. Because TC and LDL-C were relatively low in these subjects, it is possible that the differences between the MCT and LCT trials were more easily detected than would be expected for populations with higher concentrations of these lipids.

Elevations in serum concentrations of TC, LDL-C, and possibly TG have been associated with a higher risk for coronary heart disease.²⁸ Because differences in TC and LDL-C between trials were minimal, although statistically different, research confirming these results is necessary before conclusions regarding heart disease risk can be drawn. Furthermore, the relative effects of MCT feeding in comparison to sources of long-chain saturated fatty acids on blood lipids is not known for this population.

Future research should assess the effects of a longer supplementation period to determine if the negative alterations in the blood lipid profile persist and/or worsen. The effect of MCT feeding on other human populations (i.e., those using MCT as a weight loss aid) also should be examined.

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