The Effects of MCT Oil and Glucose Polymer Ingestion on Endurance Exercise

by

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(Abstract)

Seven experienced male bicyclists performed four endurance test rides at 70% (± 5) VO_2 max on a stationary bicycle at 90 RPM over a four week period. Subjects consumed a high carbohydrate diet (70%) for two days prior to each test ride.

During each test, heart rate (HR), rate of perceived exertion (RPE), VO_2, respiratory exchange ratio (R), serum free fatty acid (FFA) and serum glucose levels were measured. One of the four test treatments was randomly administered, in a single-blind design, at 5, 25, and 40 minutes into each exercise bout. The control trial (CTR) included 50 gelatin capsules containing water, and a lemonade beverage (150 ml each) sweetened with an artificial sweetener (Saccharin). The test mixtures were made up in the same manner as the control with the addition of one of the test substances: 1) MCT oil (M), 2) glucose polymer (P) (Polycose, Ross Laboratories), 3) MCT plus glucose polymer (MP). Depending on the treatment used, MCT oil-containing capsules replaced water-capsules and/or Polycose was dissolved in the lemonade beverage. Total caloric intake of each trial, except control, was 360 calories.

No significant difference was found between mean time to exhaustion for the four treatments. No significant difference was noted between treatments for R, VO_2, and HR responses ($p < 0.05$). Significantly greater RPE values were found over the first 60 minutes of exercise for the Control treatment as compared to the other three treatments ($p < 0.05$). Repeated measures ANOVA showed that significantly higher serum glucose values existed for treatment P as compared to M and also significantly higher serum FFA values existed for treatment M as compared to both P and MCT oil with Polycose (MP) over the first 60 minutes of exercise ($p < 0.05$).

Although the combination of MCT oil and Polycose would theoretically enhance endurance performance due to an increased supply of both FFA and glucose available for muscular metabolism, this dietary treatment was ineffective in prolonging exercise time.

Index terms: MCT oil; Glucose Polymer (Polycose); endurance performance

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Chapter 1

Introduction

Endurance performance is limited by an individuals' tolerance to fatigue. Fatigue has been related to several factors including muscle glycogen deletion and hypoglycemia. Enhancement of athletic performance via nutritional ergogenic aides has been the subject of many studies (Costill, Dalsky, & Fink, 1978). Glucose administered during exercise has been shown to delay fatigue possibly by replenishing depleted muscular endogenous supplies or by delaying hypoglycemia. However, if this same treatment is given prior to exercise, a hypoglycemic reaction at the beginning of exercise has been observed. This results in an impaired endurance performance (Foster, Costill, & Fink, 1979). Results of studies examining the effect of caffeine on performance indicate an increased plasma free fatty acid (FFA) level occurs suggesting an increase in muscular lipid catabolism. This allows for a decrease in carbohydrate utilization (Costill et al, 1978).

In a study by Rennie (Rennie, Winder, & Holloszy, 1976), control-exercised rats had muscle glycogen values of 43.4 micro moles of glucose/gm at rest. After 30 minutes of exercise, the glycogen level was depleted to 11.7 micro mole. Rats treated with a fatty meal had muscle glycogen values of 45.5 at rest and 21.6 micro mole after exercise.

Costill, (Costill, Coyle, Dalsky, Evans, Fink, & Hoopes, 1977) showed that an elevation of FFA following heparin injections lowered the rate of muscle glycogen depletion by 40% as compared to the control.

These comparisons show a muscle glycogen sparing action when FFA are elevated during exercise. This glycogen sparing action results in prolonged endurance exercise performance (Costill et al, 1977). Thus, accumulation and utilization of plasma substrates, other than glucose seem to prolong endurance performance, primarily by sparing muscle and liver glycogen stores.

MCT oil, comprised of Octanoic acid (71%), Decanoic acid (23%) and a mixture of longer and shorter chain length fatty acids (6%), is a lipid that is rapidly absorbed and metabolized. MCT is quickly and directly absorbed into the portal vein as glycerol and fatty acids. It is then transported to the liver where it is quickly metabolized to ketone bodies (KB), CO_2, and acetate (Greenberger & Skillman, 1969). As KB concentration increases, muscular glycolysis is inhibited (Randle, Garland, Hales, & Newsholme, 1963). In addition, KB are metabolized as an energy source by the central nervous system (CNS), and therefore further spares glucose. Thus, MCT oil administered during endurance performance could potentially spare muscle glycogen. This hypothesis has not been tested.

The effect of MCT oil mixed with cereal and milk on metabolism during a 60 minute exercise bout was studied by Ivy (Ivy, Costill, Fink, & Maglischo, 1980). Since this study was of fixed duration (60 minutes), and since MCT was given in a mixture of other foods, the effect of MCT alone on endurance performance was not determined. It is the intent of this study to test the effects of feeding MCT oil alone or in combination with a carbohydrate source during exercise, on metabolism and endurance performance.

Statement of the problem

If glycogen depletion is a major cause of fatigue during exercise, methods which will prolong the availability of muscle glycogen stores may delay fatigue.

Caffeine and heparin administered during exercise, exhibit a glycogen sparing action through their stimulation of plasma FFA and prolong aerobic exercise time. However, the effects of a direct source of FFA and ketone bodies has not been studied during endurance performance. With MCT oil providing a source of FFA's that are quickly absorbed and metabolized to KB, it was the intent of this study to determine if there were any adverse reactions to MCT oil given during exercise and to determine if this lipid source provides an alternative source of energy which could be utilized in preference to glycogen stores to prolong endurance performance.

Research Hypotheses

Null hypothesis: There will be no significant difference in time-to-exhaustion between endurance tests in which a water, glucose polymer, MCT oil, or combined glucose polymer-MCT oil beverage is administered.

Null Hypothesis: Serum glucose and serum FFA levels, heart rate, R value, and VO_2 will not change over time during exercise for any of the treatments.

Null Hypothesis: The four treatments given during exercise will not affect the serum glucose, serum FFA, heart rate, R value, or VO_2 response during exercise

Delimitations and Limitations

To insure the absence of variance between and within subjects' results, the following measures were undertaken:

I. Delimitations

A. All subjects were males, under 35 years old.

B. All subjects were required to have exercised on a bike a minimum of 50 miles per week for six weeks prior to the study, and for the entire duration of the study.

C. All subjects were screened for diabetes mellitus, celiac sprue, and liver diseases, due to possible complications with MCT oil ingestions in these conditions.

D. All subjects were deleted from the study if they 1) smoked, 2) had low tolerance to needle/IV catheter insertion, 3) were not willing to adhere to a prescribed diet for two days prior to each test ride.

II. Limitations

A. Small sample size (N=7).

B. Individual subject compliance to prescribed diet and overnight fast prior to each test ride was voluntary. Some intersubject variance from this prescription may have existed.

C. Subject acceptance and/or tolerance to the beverages administered may affect tolerance to exercise.

D. True exhaustion had to be assumed for each test, therefore results may not reflect absolute exhaustion.

Definitions

Endurance tests: tests performed at 70% VO_2 max on a bicycle ergometer to exhaustion at 90 RPM's.

Fluid replacement beverage: a beverage administered during the course of strenuous exercise to replace elements depleted during endurance exercise, such as body water, glucose, and fatty acids, and electrolytes.

Free fatty acids (FFA): metabolically, the most active of the plasma lipids comprising less than 5% of the total fatty acids present in plasma; they are an easily metabolized lipid energy source used for muscular contraction.

Glycogen, muscle: storage form of carbohydrates within the muscle cell.

Ketone bodies: a collective term for acetoacetic acid and its derivative B-hydroxybutyric acid and acetone; recognized as products of fatty acids and certain amino acid catabolism formed in the liver and used by most tissues, including the brain, as fuels and reduces the need of glucose.

Maximum oxygen uptake (VO_2): a functional measure of ones' physical fitness status; it is the largest amount of O_2 one can consume per minute.

Medium chain triglycerides (MCT): one form of a neutral lipid, triglycerides, which contain fatty acid molecules with a chain length varying from six to twelve carbon atoms, approximately 75% of the fatty acids are C:8, 23% are C:10, 1% are C:12, and 1% are C:6.

Polycose: a powdered-form, glucose polymer product manufactured by Ross Laboratories.

Rate of perceived exertion (RPE): subjective measurements made by each subject, reflecting their own perceived

amount of exertion during the endurance tests, based on Borgs' scale of numbers from 6 to 20 in a quanto-format with descriptive words printed beside every other number, ranging from "very, very light" at "7" to "very, very hard" at "19".

Respiratory quotient (RQ): the ratio of the volume of CO_2 produced to the O2 consumed at the cellular level; it is 1.0 - 0.9 for carbohydrate metabolism, 0.9 - 0.8 for a mixed diet, and 0.8 - 0.7 for fat metabolism. This parameter gives an indication of fuel utilization.

Significance of the study

If endurance performance can be enhanced via use of a readily available lipid energy source, i.e., MCT oil, alone or with a carbohydrate source, athletes may consider the use of this substance to enhance exercise performance.

Summary

Endurance athletes are continuously searching for ways to increase their athletic performance. Some measures employed have included glucose ingestion during exercise, caffeine ingestion, and heparin administration. These were tested to determine their effect on metabolism and ability to spare glycogen during performance.

An increase in endogenous glucose, and an increase in plasma FFA were observed to enhance endurance performance in

these studies. However, a readily available source of FFA and KB, MCT oil has not been studied for its effects on endurance performance. It was the purpose of this study to determine the benefits of administering MCT oil alone and with a glucose polymer during exercise. Specifically, to observe the effects of MCT oil on plasma FFA and glucose and its influence on endurance performance.

Chapter II

REVIEW OF THE LITERATURE

Introduction

Many factors contribute to the scope of this study, therefore, the following areas will be reviewed: 1.) mechanisms of fatigue during endurance exercise, 2.) glucose: its utilization during exercise, 3.) FFA: an energy source during exercise, 4.) MCT oil absorption and metabolism, and, 5.) gastric emptying time: how it is affected by nutrients and exercise.

MECHANISMS OF FATIGUE DURING ENDURANCE EXERCISE

Depletion of muscle glycogen, exercise-induced hypoglycemia, plus water and electrolyte loss in sweat are the major factors that cause fatigue and impaired performance during prolonged exercise (Costill et al, 1977; Fordtran & Saltin, 1967; Hickson, Rennie, Conlee, Winder, & Holloszy, 1977). This can be characterized by a.) a rapid increase of blood lactate levels, b.) concomittant rise in RQ above 1.0, C.) a heart rate approaching max heart rate, d.) a drop in blood sugar levels, and e.) a rise in the blood FFA/glucose ratio (Rodahl, Miller, & Issekutz 1964; Christensen & Hansen, 1939). Other factors that may contribute to fatigue are enzymatic, hormonal, and nerve conduction changes (Costill, Sparks, Gregor, & Turner, 1971).

Carbohydrate is used to supply energy primarily during high intensity exercise. The main source of carbohydrate is the local glycogen stored in the specific muscle tissue. After depletion of these glycogen stores the muscle depends on blood glucose replenished by liver gluconeogenesis. After severe drain on these carbohydrate sources, exercise above a certain intensity is no longer possible (Bjorntorp, 1983). A decrease in glycogen content as determined by needle biopsy, in the working muscles was shown to be well correlated to total carbohydrate utilization during work periods in a study performed by Bergstrom (Bergstrom, Hermansen, Hultman, & Saltin, 1967). The subjects exercised on a stationary bike at 75% VO_2 max to exhaustion. He, as well as Costill (Costill, Bennett, Branam, & Eddy, 1973) concluded that the glycogen content was a determining factor in the capacity to perform long-term heavy exercise.

Costill (Costill et al, 1977) showed that an elevation of plasma FFA decreased the rate of muscle glycogen depletion by 40%. This increased FFA oxidation produced an accumulation of citrate in the heart and skeletal muscles, and therefore limited the rate of glycolysis and pyruvate oxidation. Due to this increased reliance on fat metabolism and therefore a sparing of muscle glycogen, there is an enhanced capacity for endurance exercise (Ivy et al, 1979).

Holloszy (Holloszy & Booth, 1976) and Hickson (Hickson et al, 1977) also indicated that in trained muscles, there

is an increased fat oxidation and increased capacity for glycogen synthesis, both resulting in a slower depletion of glycogen stores. Thus, allowing for an increase in exercise time to exhaustion.

Severe hypoglycemia has been observed during prolonged heavy exercise and may limit performance (Bergstrom & Hultman, 1972). However, its limitation during exercise is on the central nervous system (CNS) and not on the working muscles. Yet, this limitation on the CNS will influence coordinated muscular performance (Asmussen, 1965).

To avoid glycogen depletion resulting in hypoglycemia the glycogen stores should be well filled prior to the day of hard exercise. This can be achieved by giving a high carbohydrate diet for several days before the event (Bergstrom et al, 1972). Also, hypoglycemia may be delayed by increasing the availability of KB's, which are also a good alternative substrate for the CNS (Hickson et al, 1977).

However, high fat diets consumed prior to exercise were not shown to increase endurance performance (Asmussen, '65). This could be due to the higher oxygen cost for exercise when mainly fat is metabolized, ie, a greater O_2 uptake is required of subjects to metabolize the predominant fat substrate. Also KB's formed during fat combustion, which cause undesireable acidosis in endurance exercise, and the depletion of body glycogen stores as a result of this diet, contribute to poor endurance performance (Asmussen, 1965).

Costill (Costill et al, 1971) demonstrated in a bicycle ergometry study that, at 80% max, the glycogen content of the medial quadriceps femoris was nearly exhausted in about one hour of exercise. This type of exercise tends to isolate metabolic demands to a relatively small muscle mass.

Localized muscular stress can also be a factor in endurance exercise. On a bicycle this is evidenced by higher perceptions of exertion due to greater muscle force required to overcome the resistance when slow pedalling speeds, as compared to fast speeds, are maintained (Pandolf & Noble, 1973). Muscular and joint discomfort realized by the rider would contribute to this sensation of stress. Also, Pandolf suggested that the anaerobic metabolism and therefore lactic acid production would contribute to a higher RPE value.

Endurance performance on a bicycle ergometer can be enhanced by using a high pedalling rate. Pandolf (Pandolf & Noble, 1973) suggests using 60-80 RPM, rather than the usual 50-60 RPM, as the higher pedalling speeds enable one to better evaluate the subjects' cardio-respiratory fitness as a limiting factor to performance rather than localized muscular fatigue. At slower pedalling rates, the resistance becomes greater due to more sustained contraction and increased muscular ischemia with high lactate levels. As a result, cyclist naturally prefer a high pedalling speed for cycling competition. However, Gollnick (Gollnick, Piehl, &

Saltin, 1974) showed that varying the pedalling rate had no effect on patterns of glycogen depletion in the muscle fiber.

The muscle fiber type present in exercising muscles will also play an important role in delaying fatigue during prolonged endurance exercise. Red muscle fiber (slow twitch) are able to utilize an increased supply of FFA for energy, inhibiting carbohydrate utilization and therefore sparing muscle glycogen stores. The white muscle fibers have a low capacity to oxidize and utilize FFA for energy and will deplete muscle glycogen stores more quickly. This may explain part of the effect of training on fuel utilization. For example, Karlsson (Karlsson, Nordes, & Saltin, 1974) proposed that the enhanced fat oxidation and the decreased carbohydrate oxidation in trained subjects are brought about by an increased recruitment of red muscle fibers. This is indicated by the decrease in the RQ value observed in the trained subjects.

GLUCOSE UTILIZATION DURING EXERCISE

Carbohydrate ingestion during exercise may offset fatigue, caused by a decreased rate of muscle glycogen utilization during events of long duration (ie, greater than or equal to 60 minutes), (Costill et al, 1973 and Ahlborg & Felig, 1976). This is due to the maintenance of blood glucose by exogenous ingestion during exercise.

Costill has shown that ingestion of glucose 30 to 45 minutes prior to exercise produces hypoglycemia, depresses the mobilization of serum FFA, and thereby reduces the exercise time to exhaustion. Ivy (Ivy et al, 1980) indicated that insulin levels are also elevated dramatically when a glucose feeding is given immediately prior to exercise. It was therefore shown that the glucose feedings prior to exercise increased the rate of carbohydrate oxidation yet impeded the mobilization of FFA. This decreased the exercise time to exhaustion (Ivy et al,1980).

However, if glucose is consumed during exercise, elevated blood glucose levels are maintained. The rate of fatigue was reduced in the latter stages of an exercise bout, enabling an increase in total work production. Gluconeogenesis in the liver from glycerol, lactate, alanine, and KB's may also delay symptoms of hypoglycemia (Hickson et al, 1977; Neely & Morgan,1974). Yet, the time of total work production was not increased. In contrast, Hickson (Hickson et al, 1977) showed that if glucose was given at the time of fatigue during endurance exercise, the subjects could continue the exercise bout for a significant amount of time; the ingested glucose was utilized by the brain (CNS) as adequate FFA were being metabolized by exercising muscles, and therefore exercise time was prolonged.

Costill (Costill et al, 1973) has shown that glucose ingested during exercise does not contribute much (about 5.4%) to carbohydrate oxidation. Wahren (1977) also indicated that glucose administration during strenuous exercise does not significantly decrease muscle glycogen consumption. Yet, another study by Ivy (Ivy et al, 1979) involved the ingestion of about 90 grams of a glucose polymer (Polycose) during the first 90 minutes of endurance exercise on a bicycle ergometer at 80 RPM. It was shown to have no effect on total work production of VO_2, yet it was effective in decreasing the rate of fatigue over the last 30 minutes of cycling, and therefore may have been effective in sparing muscle glycogen.

However, since the hepatic contribution to blood glucose is reduced following the ingestion of glucose during exercise, it is possible that such feedings may prevent liver glycogen depletion during prolonged severe exercise. Current findings suggest that gluconeogenesis from endogenous precursors is inhibited when glucose feedings are given during exercise (Ahlborg and Felig, '76). Therefore, an increase in exercise time to exhaustion was observed.

A possible mechanism for the beneficial effect of carbohydrate feedings during exercise as opposed to effects if given before exercise may be the contrasting effects on plasma insulin. For example, in a study by Ahlborg, (Ahlborg & Felig, 1977) it was shown that glucose ingested

prior to mild exercise (30% max VO_2) resulted in hyperinsulinemia and hyperglycemia. However, Bonen has shown that glucose ingestion during intense exercise failed to increase glucose and insulin concentration(Bonen, Malcohlm, & Kilgour, 1981). This contrasts with Ahlborgs' study where a rise in serum insulin was observed when glucose was ingested during mild exercise which enhanced the exercise-stimulated glucose uptake (Koivisto, Karonen & Nikkila, 1981). The increase in carbohydrate utilization by exercising legs and inhibition of lipolysis after glucose feedings could be attributed to an increase in arterial glucose, elevation in plasma insulin, or both (Ahlborg et al, 1976).

The difference in insulin responses in these two studies appears to be related to differences in norepinephrine levels provoked by different intensities of exercise, ie, intense being 80% VO_2 max, mild being less than or equal to 65% VO_2 max (Bonen et al, 1981). When glucose is ingested during mild exercise, there is a rise in serum insulin, during intense exercise, the norepinephrine response inhibits insulin release from the pancreas (Bonen et al, 1981). Henter and Sukkar (Costill et al, 1973) reported evidence to suggest that increased utilization of glucose during muscular work is not dependent upon insulin.

Costill (Costill et al, 1977) also presented strong evidence in a study that ingested glucose appeared in the

blood within 5 to 7 minutes after ingestion, and that it was the primary carbohydrate source during intense exercise. Since intense exercise (80%) normally relies extensively on carbohydrate metabolism, glucose ingestion prior to exercise, utilizing glycogen-depleted subjects, is qualitatively and quantitatively more important than its ingestion before mild (40% max VO_2) exercise (Bonen et al, 1981).

Bergstrom (Bergstrom et al 1972), described the general breakdown of glycogen during submax exercise as the following : 1.) initial rapid decrease due to stimulation by the initial muscle contraction, adrenaline release, and possibly a local decrease of O_2 pressure; 2.) second phase notes an adaptation of circulation to work load and changes in enzymatic activities which result in a decreased rate of glycogen consumption, and , 3.) final phase, in which there is a relative lack of glycogen and the muscle partially compensates for this by an increased uptake of blood glucose and an increase in lipid metabolism.

The repeated muscle contraction is associated with a marked increase in muscle cell permeability to glucose. Increased transport of glucose into the cell followed by conversion to glucose-6-phosphate, which would inhibit glycogenolysis (Baldwin, Reitman, Terjung, Winder, & Hollosky, 1973). So, it would seem important to maintain blood glucose as well to achieve a high level of glycogen

storage prior to an exhaustive bout of endurance exercise to delay fatigue (Bergstrom, 1972).

In a study by Astrand, (1969), three different types of diets were compared for exercise time to exhaustion on a bicycle ergometer. Subjects performed only 57 minutes while on a high fat/high protein diet, for 114 minutes with a mixed diet, and for 167 minutes on a high-carbohydrate diet, therefore supporting the importance of glycogen utilization in endurance exercise.

Another important factor related to glycogen sparing is the level of training sustained by individual subjects. Karlsson (Karlsson et al, 1974), showed in a study that there is lower glycogen depletion and lower RQ values during exercise performed at the same O_2 uptake level for each subject. This points to a reduced oxidative use of carbohydrate after training, possibly brought on by an increased recruitment of red muscle fibers (Karlsson & Saltin, 1971; 1974). Therefore, after training, there is an increase in oxidative enzymes and an increase in the capacity to oxidize fatty acids. Hollosky, (Hollosky et al, 1976) indicated that there is also an increased capacity for glycogen synthesis in trained muscles.

FFA: ENERGY SOURCE DURING EXERCISE

At a given metabolic rate, the rate of FFA oxidation appears to be determined by 2 factors: 1) the concentration of fatty acids, (ie, substrate availability),

and 2) the capacity of the tissue to oxidize fat, due to adaptation in muscle mitochondria and an increase in their enzymes involved with FFA oxidation (Oscai & Palmer, 1983). Therefore, the availability of FFA to the mitochondria is perhaps the rate-limiting factor for FFA oxidation at any given work rate (Hollosky et al, 1976).

Ivy (Ivy et al, 1980) indicated that an increased insulin level during exercise, causes a preferential oxidation of carbohydrate even when FFA and ketone bodies are present in elevated amounts. Insulin also acts by decreasing the rate of release of FFA.

In endurance exercise, there tends to be a fall in circulating insulin, with a resultant rise in plasma glucagon. This allows for a progressive increase of lipolysis (Ahlborg et al, 1976). These changes are influenced by norepinephrine production. An increase of norepinephrine in the blood and adipose tissue, increases the lipolytic action in adipose tissue, and possibly also limits insulin release from the pancreas.

With the increased metabolic rate and increased norepinephrine production, plus decreased insulin levels, the energy stores of adipose tissue and muscle become available for metabolism, and a shift towards fat utilization will occur (Paul, 1970).

An increased mobilization of FFA during exercise can result from hydrolysis of triglycerides in adipose tissue,

as indicated by a rise in plasma glycerol concentrations (Havel, Naimark, Borchgrevink, 1963). This can be an important source of energy for muscular contraction (Young, Shapira, Forrest, Adachi, Lim, & Pelligra, 1967). Terjung (Terjung, Mackie, Dudley, & Kaciuba-Useilko, 1983) showed that, in fed, exercising animals, there was a direct uptake of TG-derived FFA that could be used for beta-oxidation. These FFA represented a large source of energy substrate in a post-prandial state. It was this source of lipid, that when experimentally controlled, appeared to enhance exercise performance and spare muscle glycogen (Terjung et al, 1983).

During prolonged exercise, mobilization of FFA's from adipose tissue increases gradually. This results in a decreased plasma concentration initially, followed by the gradual increase, and ending with a peak concentration immediately post exercise. (Bjorntorp, 1983). It is the trained subjects, again, who have a greater ability to release FFA from adipose tissue which enhances proportions of energy that can be produced thru fat metabolism (Lewis, 1973). Trained endurance athletes will be able to derive more of their energy from fat and less from carbohydrate than untrained individuals with less formation of lactic acid (Hickson et al, 1977; Hollosky et al, 1976). This adaptation induced in skeletal muscles could be responsible for delaying muscle glycogen depletion and therefore prolonging endurance exercise in trained individuals. In

addition, the trained person will produce less lactate at a given workload than an untrained person. High levels of blood lactate may interfere with release of FFA (Rodahl et al, 1964 and Lewis & Gutin, 1973). Thus, the trained individual is again at an advantage for fat utilization during exercise.

Bonen (Bonen et al, 1981) demonstrated that during intense exercise (about 80% VO_2 max), causing lactic acid production, fat metabolism was decreased even when FFA concentrations remained elevated; carbohydrate metabolism was then preferred. This is in agreement with a study by Paul (1970) in which it was shown that as long as the O_2 supply was adequate, without a rise in lactate, the FFA turnover and oxidation rate increased in a way that the percentage of total energy derived from FFA oxidation would be equal at higher workloads. But, as lactate rose with increasing workloads, the FFA turnover rose insufficiently. Even though the turnover percentage was large, the amount of energy derived from plasma FFA oxidation was considerably less at this time (Paul, 1970). This indicated that the FFA turnover and oxidation rates were dependent not only on plasma FFA levels, but also on the adequacy of the O_2 supply to the working muscle and adipose tissue.

In a study by Costill et al (1977) 7 males exercised on a treadmill at 70% VO_2 max. Plasma FFA were elevated with heparin (2,000 units), decreasing muscle glycogen depletion

by 40%, as compared to controls. Apparently this was due to the inhibitory effect of citrate on phosphofructokinase, therefore limiting the rate of glycolysis.

Diet, prior to or during an endurance event will also affect the rate of FFA mobilization and utilization. Issekutz (Issekutz, Birkhead, & Rodahl, 1963) suggested that it was the actual carbohydrate intake rather than the amount of fat in the diet that was a determining factor of whether fatty acids or glucose would be primarily utilized during exercise.

FFA metabolism will be decreased when glucose is given prior to or during strenuous exercise (80% VO_2), even though the FFA concentration may remain elevated (Bonen et al,1981).

In a study by Christensen (Christensen & Karlsson, 1939), subjects consumed a specified diet for several days prior to a bicycle exercise bout at 1,080 KPM/min. One plan utilized a high fat, low carbohydrate (5%) diet, which resulted in a decreased work RQ. The second plan, consisted of a high carbohydrate (90%), low fat diet, and had opposite effects (Christensen & Karlsson, 1939). The R value is related mainly to: 1.) diet of subjects, 2.) intensity of work, and 3.) duration of work (Asmussen, 1965). Also, when serum FFA levels are increased prior to exercise, as following an overnight fast, there is a marked increase in FFA oxidation (Ivy, 1980).

In a study by Ivy (Ivy et al, 1980) subjects exercised at 70% VO_2 max. It was indicated that FFA metabolism was inhibited if subjects consumed cereal, a carbohydrate source, one hour prior to exercise. Also that lipolysis would be diminished if carbohydrate was taken during exercise.

Other researchers also indicated that decreased FFA concentration after glucose ingestion during exercise may result in decreased FFA utilization. (Koivisto et al, 1981; Havel et al, 1963; Ahlborg et al, 1976; Bellet, Kershbaum, & Finck,1968). Following a glucose feeding during exercise, the carbohydrate inhibits mobilization of FFA's from adipose tissue , and therefore skeletal muscles receive less to burn. This could be attributable to an elevation in arterial glucose or an increase in plasma insulin, or both (Ahlborg et al, 1976; Foster et al, 1979).

MCT OIL: ABSORPTION AND METABOLISM

MCT oil is a form of neutral lipid triglycerides that contains fatty acid molecules of 6 to 12 carbon atoms in length (Greenberger et al, 1969); Holt, 1971). 71% is octanoic acid (C:8), 23% is decanoic acid (C:10), (Ivy et al,1981).

MCT oil requires less pancreatic lipase of bile salt for digestion than does long chain triglycerides (LCT). They are more rapidly and efficiently absorbed from

intestine than are LCT after digestion and intestinal absorption (Guy, 1981; Baba, Bracco, & Hashim, 1982). They may be absorbed intact by the small intestine and are completely hydrolysed in intestinal mucousal cells. Gastric lipase might hydrolyze ingested MCT. Such gastric hydrolysis might contribute to its efficient absorption (Cohen, 1970).

Trioctanoin, in the form of MCT oil, is rapidly hydrolzed by pancratic lipase to octanoic acid. These fatty acids that are derived from MCT are absorbed and transported via the portal vein as the fatty acid directly to the liver; they are not deposited in adipose tissue, (Guy, 1981; Greenberger et al, 1969; Holt, 1967). It has also been shown that the fatty acids of MCT do not appear in the plasma after oral administration.

Hashim (1966) reported that, after oral intake of MCT oil, 90% was present in the FFA form in the portal vein indicating mucousal hydrolysis of MCT.

Once these medium chain fatty acids (MCFA) reach the liver they are rapidly metabolized and oxidized extensively into CO_2, water, acetate, and ketones (Guy, 1981; Baba, 1982; Greenberger et al, 1969). The MCFA not catabolized are converted to long chain fatty acids (LCFA) and esterified to LCT. The rapid conversion of MCFA to CO_2 has been utilized to estimate intestinal absorption indirectly. In normal human subjects following oral administration of

C-14 trioctanoin, 15 to 20% of the dose was recovered in expired air within 50 minutes (Greenberger et al, 1969). Hashim (1966) reported 15% recovery of C-14 MCT as CO_2 within the first 20 to 60 minutes.

Hyperketonemia, which results from hepatic metabolism of MCFA, causes an increase in insulin secretion. Tamir (Tamir, Grant, Fosbrooke, Segall, & Lloyd, 1968) showed that insulin output from the pancreas in rats, can be directly stimulated by octanoic acid. An increased insulin secretion following MCT ingestion may lower glucose levels. Increasing the concentration of FFA's impairs glucose tolerance causing hypoglycemia, due to reduced hepatic glucose release (Greenberger et al, 1969).

In a study by Tantibhedhyangkul, (Tantibhedhyangkul, Hashim, & VanItallie, 1967), 7 normal subjects were given 1 gm/ kg body weight of MCT oil inducing mild hyperketonemia. At the height of MCT-induced hyperketonemia, glucose tolerance was not impaired. However, octanoate-induced hyperketonemia appears to depress glycolysis in the liver resulting in hypoglycemia and a lower rate of FFA synthesis. This may further enhance ketogenesis. This resulting hyperketonemia may promote insulin secretion, and under this condition, an improvement in glucose tolerance to an administered glucose load will be noted. Pi-Sunyer observed a decrease in glucose transport and utilization in heart and skeletal muscle in the presence of K.B.'s (Pi-Sunyer, Hashim, & VanItallie, 1969).

Newsolme, et al, (Newsolme, Randle, & Manchester, 1962) proposed this is due to an inhibition of glucose transport and phosphofructokinase (PFK), by muscular K.B. catabolism. That is, K.B.-mediated depression of glycolysis is similar to the inhibitory effect of FFA oxidation on glycolysis mediated by PFK inhibition (Randle, et al, '63). K.B.'s formed after MCT oil ingestion can also serve as an energy source to the CNS (Owen Morgan, & Kemp, 1967), and therefore this could be another means to spare muscle glycogen.

Linscheer (Linscheer, Chalmers, & Slone, 1967) and Tamir (1968) showed that glucose given along with MCT would increase serum insulin levels more than if glucose was given alone. If MCT is being administered, and a sudden glucose load is given, the rate of glucose disappearance may be improved above normal rates (Greenberger, 1969).

When carbohydrates are given during the ketogenic phase following MCT ingestion, ketogenesis may be inhibited. This suggests that the TCA cycle is not functioning at a maximal rate when challenged with MCT, but can be accelerated by adding substrate, therefore decreasing the ketone formation (Freund & Weinsier, 1966). Yet, Schwabe (Schwabe, Bennett, & Bowman, 1964) has shown that prompt oxidation of MCT was not substantially inhibited by moderate doses of glucose. MCFA are metabolized as quickly as glucose (Ivy, 1980).

Other results following MCT oil ingestion include nausea, vomiting, abdominal pain, and diarrhea. These may

be related to the rapid hydrolysis and resulting high concentration of FFA in the stomach and small intestine (Holt, 1967 and 1971).

Holt (1971) has shown that 40 to 50 grams of MCT can be tolerated in patients throughout the day. However, Ivy (Ivy et al, 1980) gave subjects 50 to 60 gm of MCT which resulted in 100% of subjects with cramping and diarrhea. Then, 30 gm was given, resulting in only about 10% of subjects experiencing any negative effects from MCT ingestion.

Ivy performed a 60 minute test with 10 subjects, at 70% VO_2, on a bicycle ergometer (80 RPM) and treadmill. This study determined the effect of 30 gm of MCT oil mixed with cereal on metabolism during endurance exercise. Ketone body concentration increased with MCT ingestion. Ketone bodies are readily metabolized by skeletal muscle (Little, Goto, & Spitzer, 1970). Since ketone body oxidation is primarily a function of plasma concentration (Harper, Rodwell, & Mayes, 1977) ketones were likely serving as energy substrates in the muscles after MCT ingestion. The blood concentration of K.B. is probably the determining factor for the rate of oxidation in the CNS (Hultman & Nilsson, 1975).

Of interest is Bassenges' observation (in Little et al, 1970) that when ketones were infused into dogs, an increase in RQ was seen along with a drop in the removal of FFA. However, RQ results in Ivys' study (1980) indicated that the carbohydrate utilization was the same in the MCT and cereal

test as well as the cereal test alone. Since RQ was used only to indicate percent carbohydrate and fat oxidized, ketone body formation and oxidation was not included in this measure. Therefore, the conclusion determined by Ivy, (Ivy et al, 1980) that ketone bodies did not contribute to energy needs of exercise may not hold true.

GASTRIC EMPTYING TIME: HOW IT IS AFFECTED BY NUTRIENTS AND EXERCISE

Many studies have shown that volume, osmotic pressure, and chemical composition of a test meal influence gastric emptying (Costill & Saltin, 1974; Hunt & Knox, 1968). The rate at which the stomach empties is strongly influenced by the chemical and physical properties of the chyme entering the duodenum (Costill et al, 1974).

Hunt has suggested that duodenal receptors monitor the osmolarity of gastric emptying and by a feedback loop, influence the rate of gastric emptying (Hunt et al, 1968). A saline solution affected gastric emptying only in terms of volume infused: as nutrient was added, the emptying slowed down. However, identification of either the receptors or pathways, neural or humoral, by which the receptors might exert their control is lacking.

Also, volume, chemical composition ,and osmotic pressure are major determinants for gastric emptying (Davenport, 1969). Osmotic pressure effects the rate of

gastric emptying via the enterogastric reflex; the higher the pressure, the slower the emptying rate. Of the 3 major foodstuffs, fat has the greatest effect on delaying gastric emptying, carbohydrates empty the quickest.

Painful levels of ingragastric pressure inhibit gastric motility, as volumes greater than 600 to 750 ml reduce the rate of gastric emptying (Costill et al, 1974). In particular, it is important to minimize the sugar content of fluids administered during high intensity exercise (Greater than 70% VO_2 max). Otherwise, the inhibitory effect of both exercise and glucose content on gastric emptying may combine to block gastric emptying. Costill (Costill, 1974) stated that, in general, exercise has no effect on emptying until work intensity is greater than 70% max.

In another study by Costill, it was shown that the osmotic effect of glucose only delayed gastric emptying when the concentration exceeded 139mM. A glucose solution of greater than 139 mM emptied from the stomach in a continuous flow (21 ml/min.) soon after ingestion. Addition of even 5 gm/100 ml, glucose in water, caused a marked inhibition of gastric emptying (Costill, 1973).

When a 125 ml "test meal" of MCT oil was administered to healthy, fasted subjects, at least 6 hours was required for complete gastric emptying to occur (Pirk & Skala, 1970). The ingestion of 1 gm/kg body weight of MCT oil increased octanoic acid from 0% to 50% within 30 minutes, and peaked at 57.7% 90 minutes after ingestion (Tamir et al, 1968).

Another study has shown that approximately 15% of an orally or intravenously administered C-14 labeled MCT was recovered as CO_2-14 within the first 20 to 60 minutes (Schwabe et al, 1964; Hashim, 1966; Greenberger et al,1969). McHugh (McHugh & Moran, 1979) indicated that MCT oil meals at 0.5 calories/ml, emptied at the same rate as glucose. Fatty acids of less than 10 carbon atoms are relatively ineffective in slowing gastric emptying time (Hunt, 1968). Three isocaloric meals, of 150 mls at 0.5 calories/ml, were compared in their gastric emptying time. Casein hydrolysate, an MCT oil suspension, and glucose resulted with superimposable regression lines. Isocaloric loads of these 3 nutrients, as stated, emptied from the stomach at the same rate.

Intensity of exercise effects gastric emptying also. Costill (Costill & Saltin, 1974) has shown that exercise (cycling) has little effect on gastric emptying at workloads requiring less than 70% of one's VO_2 max. In addition, prolonged endurance did not cause a change in gastric emptying time.

In summary, endurance exercise performance has been found to improve when serum glucose or FFA levels are increased via glucose ingestion during exercise or caffeine ingestion and heparin injections, respectively. Total exercise time was increased with glucose ingestion during exercise (Ivy et al, 1983). FFA were utilized more

extensively for energy, therefore possibly sparing muscle glycogen, when increased with caffeine and heparin, given prior to exercise (Costill et al, 1971 and Ivy et al, 1979). However, the effects of administering MCT oil alone or with Polycose during endurance exercise has not been determined previously. Therefore, this study was performed to determine the benefits, if any, of administering these treatments during prolonged exercise.

Chapter III

JOURNAL MANUSCRIPT

The Effects of MCT Oil and Glucose Polymer Ingestion on Endurance Exercise

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Introduction

Fatigue during endurance performance is associated with depletion of muscle glycogen stores and hypoglycemia (Costill et al, 1977; Fordtran et al, 1967; Hickson et al, 1977). Glucose, when ingested prior to exercise, has been shown to cause a hypoglycemic reaction during exercise, and therefore reduced time-to-exhaustion. The glucose ingestion increases serum glucose levels, with increased insulin levels, which allow for increased peripheral glucose utilization with impeded mobilization of free fatty acids (FFA). Therefore muscle glycogen depletion is increased while liver glucose output is diminished (Bergstrom & Hultman, 1972).

However, if carbohydrate is given during exercise, the muscle glycogen utilization rate may be lowered during endurance exercise (equal to or greater than 60 minutes), due to maintenance of blood glucose levels by the exogenously supplied glucose (Costill et al, 1973; Ahlborg et al, 1976). Ivy et al. (1983) have shown that time to exhaustion can be significantly increased when carbohydrate (Polycose, 120 gms) is given during exercise. Costill (1977) has shown that with heparin (2,000 units) muscle glycogen can also be spared by raising serum free fatty acid (FFA) levels, and therefore utilization, during exercise, enhancing the capacity for more endurance exercise (Ivy et al, 1979). Caffeine ingestion and heparin injections have

both been used to increase serum FFA levels and have been shown to cause a muscle glycogen sparing effect and therefore enhanced endurance performance (Costill et al, 1978 and 1977).

Trioctinoin, in the form of medium chain triglyceride oil (MCT oil), is a lipid containing fatty acid molecules with a chain length varying from six to twelve carbon atoms. It is a rapidly absorbed and metabolized source of lipid and could be another means to spare muscle glycogen when ingested prior to or during endurance exercise. Following the ingestion of MCT oil, the resulting medium chain fatty acids (MCFA) are quickly transported to the liver for metabolism and oxidation into CO_2, water, acetate, and ketone bodies, resulting in hyperketonemia (Owen, 1967). This results in increased insulin secretions which may lower glucose levels. Yet, in the presence of ketone bodies, glucose transport and utilization has been shown to decrease. These ketone bodies formed after MCT oil ingestion can serve as an energy source to the central nervous system, therefore sparing glucose utilization (Owen et al, 1967). Thus, MCT oil ingestion could theoretically spare muscle glycogen during endurance performance.

MCT oil given alone and with a glucose polymer (Polycose), during endurance exercise has not been studied. Since FFA oxidation is mainly a function of plasma concentration for these substrates, and since the proportion

of lipids given to energy metabolism is greater with increased FFA levels, carbohydrate utilization should be reduced, causing a glycogen sparing action. Since MCT may increase hepatic FFA synthesis and reduce ketone clearance (Greenberger, 1969) it was the intent of this study to determine if beneficial effects may have been attributable to MCT oil alone and in combination with glucose, during endurance exercise, on total exercise time and on serum FFA and glucose.

Methods

Seven experienced male bicyclists were chosen as subjects for this study. Each biked at least 50 miles/week for six weeks prior to the study, and continued this level of exercise during the entire testing period. (Table 1).

All subjects were between the ages of 18 and 31, and were required to give informed consent. Subjects were disqualified from the study if they had 1) diabetes mellitus, 2) cirrhosis or other liver diseases, 3) inflamatory bowel disease (Greenberger et al, 1969; Holt, 1967), 4) ever smoked tobacco products, and 5) were intolerant to intravenous catheter insertion and usage.

Subjects were requested not to consume alcohol or caffeine for 24 hours prior to the endurance rides and not to exercise strenuously for the same period. The initial meeting included measurements of resting EKG, maximum oxygen consumption on a bicycle ergometer, exercising EKG and blood

pressure, and analysis of a previously completed three-day diet record to determine average calorie intake for each subject. A pre-test trial ride was used to familiarize each subject with the experimental procedures and to determine if the designated work rate was appropriate. Each subject was scheduled to participate in one experiment per week on the same day of the week and at the same time of day.

All subjects consumed a 500 calorie meal of fixed nutrient composition (70% carbohydrate, 18% fat, 12% protein) by 8:00 p.m. the evening before each endurance bicycle ride and arrived to the lab after an overnight fast and without breakfast. Upon arrival to the lab, initial dry weight was obtained. An indwelling catheter was inserted in a forearm vein (cephalic or basilic) by a registered nurse. The vein was kept open during exercise by a continuous drip of sterile saline at an infusion rate of 20 - 40 cc/hour.

Subjects consumed one of four capsule and beverage mixtures, as a single-blind design. The control trial included gelatin capsules containing water, and a lemonade beverage (150 ml each) sweetened with saccharin. The test mixtures were made up in the same manner as the control with the addition of one of the test substances: 1) MCT oil (40 gms), 2) glucose polymer (Polycose powder, Ross Laboratories) (94.7 gms), 3) MCT oil plus glucose polymer (20.0 gm MCT oil plus 47.7 gm Polycose). MCT oil replaced water in all the capsules during the MCT oil-containing test

rides while MCT oil capsules replaced half of the water capsules in the MCT-Polycose test rides. Polycose powder was dissolved in the lemonade in the MCT-Polycose and the Polycose only rides.

The first capsule/beverage (150ml) consumption occurred after 5 minutes of the endurance bout for a total caloric intake of 180 calories. The second and third capsule/ beverage (150 ml each) treatment was consumed at 25 minutes and 40 minutes of exercise, respectively, for a caloric intake of 90 calories each. Calories from the combined MCT oil and Polycose solution contributed equally from both nutrients. Therefore, total calories consumed during all exercise bouts, except the control, were 360 calories each.

Immediately prior to each exercise bout, resting EKG, and initial blood samples (5 ml) were obtained. Seat height was adjusted to a determined level specific for each subject throughout the testing period. A fan was placed about 3 feet in front of the subjects during each test ride to minimize thermal stress. A metronome was used to control the cadence at 90 RPMs'.

During the 70% (±5) VO_2 max exercise rides, rate of perceived exertion (RPE: Borg scale), VO_2, respiratory exchange ratio (R), and heart rate (HR) were measured every 10 minutes until exhaustion. Exhaustion was determined as that point when subjects were unable to maintain pedalling speed. Blood samples were obtained at 15 minutes into

exercise, every 30 minutes during exercise, at exhaustion, and after 30 minutes of recovery. Total volume blood collected per trial per subject was approximately 30 - 35 mls.

Blood samples were immediately separated by centrifugation. Serum was frozen for later analysis for glucose and FFA. Serum glucose (mg/dl) was analyzed using the glucose oxidase method (#510 Sigma Glucose Enzymatic Colorimetric Assay Kit). Serum FFA (mM) concentration was measured using a colorimetric procedure (Costill et al, 1980).

Expired O_2 and CO_2 were analyzed during the bicycle tests using the Hewlett Packard 4730A Digital Pneumotach to determine VO_2 and R values. Calibration of the analyzers was performed prior to and periodically during each test ride using O_2 and CO_2 reference gases. Heart rate measurements were taken during exercise using a single lead EKG.

One-way ANOVA was performed on HR, R, VO_2, FFA, and glucose at their specific measured intervals through minute 60 of exercise, and at exhaustion for all four treatments; this was also performed on serum FFA and glucose at time-to-exhaustion and 30 minutes post-exercise. Repeated measures ANOVA was performed on RPE plus all the above dependent variables through 60 minutes of exercise. Duncan Multiple Range test was used to determine which treatments were found

to be significantly different from the others. The level of significance accepted was $p < 0.05$.

Results

There was no significant difference noted between mean time to exhaustion for the four treatments. However, as shown in Figure 1 subjects tended to have the longest exercise time when receiving Polycose (P) and the shortest when consuming MCT oil (M) alone (Appendix O). MCT oil and Polycose combined (MP) and the control (CTR) resulted in a similar intermediate exercise time (Appendix O). Most subjects reported "nausea" to be the main reason for terminating exercise during treatment M. Only a few subjects reported this for treatment MP and none for the P or CTR treatments. Almost all subjects reported that boredom, a time factor in their own schedule, or being "saddle sore", were the main reasons for terminating exercise during treatment P and CTR. This would indicate P provided an energy supply that was better tolerated, by the gastrointestinal system, than the other treatments.

The subjects had similar R, VO_2, and HR responses to the four treatments through minute 60 ($p < 0.05$) (Table 2; Figure 2). Based on the respiratory exchange ratio (R), carbohydrate (CHO) and fat oxidation were essentially the same for all treatments through minute 80. The R value for

P and CTR trials continued to rise up to exhaustion while R for the MP and M treatments showed a slight decline until exhaustion (Figure 2). MCT oil given alone during exercise or in combination with carbohydrate appeared to enhance fat oxidation during latter stages of prolonged exercise while provision of carbohydrate substrate alone as Polycose increased the CHO metabolized for energy.

Oxygen uptake (VO_2) was essentially the same through out exercise, for all four treatments. However, VO_2 from minute 60 to exhaustion during treatment M tended to exhibit a higher response than the other three treatments. This indicated a greater O_2 supply was necessary to oxidize and metabolize the increased levels of FFA resulting from treatment M, which was consistent with the slightly lower R values for M.

The heart rate data (Figure 2) showed there was a normal rise in HR during exercise over time for all treatments as exercise commenced. However, there was no significant difference found between any of the treatments during exercise (Table 2).

As expected, rate of perceived exertion (RPE) (Figure 2), also indicated a progressive rise over time. RPE values were only recorded throughout the first 60 minutes of exercise, and then at random for the remainder of the test ride. Treatment CTR data indicated there was a significantly greater perceived effort through the first 60

minutes of exercise than the other three treatments ($p < 0.05$) (Table 2). The least effort was reported during the MP trial. One subject even reported a drop in perceived exertion during treatment P, from "14" at minute 60 to "12" at minute 90 on the Borg scale indicating less perceived effort during the last 50 minutes of his exercise bout.

ANOVA for repeated measures showed that significantly higher glucose values for treatment P existed over the first 60 minutes of exercise compared to treatment M ($p < 0.05$) (Figure 3) (Appendix R). Treatment P caused a noticeable rise in glucose from time 0 to 15 minutes. A slight drop in serum glucose was noted for both MP and CTR trials at minute 15 followed by a rise up to minute 30, at which time there was a progressive drop on through exhaustion. MCT oil consumption tended to result in the lowest levels of blood glucose (Figure 3). All four treatments consistently displayed a drop in mean serum glucose values during the last 30 minutes of exercise, indicating a gradual depletion in carbohydrate supplies. Serum glucose in the MP trial was found to be significantly higher than CTR and MCT oil alone at 30 minutes post exercise ($p < 0.05$).

ANOVA for repeated measures indicated that serum FFA values for treatment M were significantly higher than both P and MP up through 60 minutes of exercise ($p < 0.05$) (Appendix R). Also, there was a significant time effect on FFA levels during this time period, again, treatment M

allowed for the highest values (Figure 3). The divergence in serum FFA values, as noted in Figure 3, began at minute 30 of exercise. At this time CTR rose from 0.4 mM FFA to an average end point of 0.94 mM at exhaustion. P, providing no exogenous fat, showed a consistently lower concentration of FFA throughout exercise. MCT provided the greatest FFA supply, up to minute 90, which was found to be significantly greater than treatment P at minute 60 only ($p < 0.05$).

The blood analysis was found to be consistent with the R data in suggesting a greater supply and utilization of FFA by subjects during exercise when MCT oil was ingested and an enhanced carbohydrate utilization when Polycose was consumed.

Discussion

Numerous studies have indicated that depletion of endogenous carbohydrate stores is a limiting factor in endurance exercise, and that reducing the rate of glycogen utilization should improve endurance performance (Costill et al, 1977; Hickson et al, 1977; Rennie et al, 1976). Seiple et al (1983) have shown that, when compared to free glucose, a 5% carbohydrate solution with Polycose resulted in 69% more fluid and 33% more carbohydrate being sent to the small intestines after 30 minutes of ingestion delaying endogenous glycogen depletion during exercise. Also, it has been shown that with an increased level of serum free fatty acids, there was an increased rate of muscular lipid metabolism

therefore reducing muscle glycogen utilization in exercising skeletal muscle (Costill et al, 1977; Issekutz et al, 1965).

The use of MCT oil (M) treatment alone, and Polycose (P) treatment alone, in the present study, caused a significant increase in plasma FFA and glucose, respectively. However, mean time to exhaustion was not found to be significantly different between MCT oil-containing trials, Polycose trials, or the control. In fact, treatment M, was found to hinder exercise performance due to the nausea that occurred after ingestion of 40 g of MCT oil. A study by Ivy (1980) indicated that even 30 gms of MCT oil when ingested prior to exercise would cause abdominal cramping and diarrhea. Even the MP trial, although containing only half the amount of MCT oil as treatment M, resulted with 3 of 7 subjects complaining of nausea.

Carbohydrate given during exercise, in the form of Polycose, seemed to result in the longest exercise bouts (not significant). However, no significant difference in time to exhaustion was noted between treatments. A study by Ivy et al. (1979), showed that when 90 gm of Polycose was given during a two hour endurance bike ride, work production increased above what was found during the first 10 minutes of exercise. This was possibly due to the elevated supply of blood glucose made available for utilization at that time. This report suggested that Polycose might allow an

increased exercise time to exhaustion in endurance work. In fact, a later report (Ivy et al, 1983) showed that when Polycose (120 gms) was given during exercise, time to exhaustion was significantly increased by 11.5% as compared to the control group.

The downward trend in R values for the M and MP trials could have been indicative of the increasing FFA utilization as a result of the MCT oil ingestion. In studies by Costill et al. (1971) and Ivy et al. (1979) FFA levels were increased by caffeine ingestion and heparin injections prior to exercise. The R data in both studies also demonstrated an increase in fat oxidation with a reduced rate of carbohydrate oxidation , indicating an enhanced utilization of FFA by exercising muscles. Therefore, since serum FFA levels were elevated by the ingestion of MCT oil in the present study, this could indicate an enhanced sparing of glycogen stores, as FFA contributed progressively more to the energy for work. However,the present study was not able to determine this due to the occurrence of nausea. Polycose seemed to provide an exogenous carbohydrate supply that was relied on more extensively than fat, especially during the latter part of the exercise bouts, as noted by the increasing R value throughout exercise. Alternately, the rise in R values at the end of exercise for both P and CTR might also have been influenced by respiratory compensation secondary to increased lactate production as fatigue was approached (Rodahl et al, 1964).

A similar pattern of average heart rate values was noted over the four treatments indicating there was no significant difference in cardiovascular stress as a result of the nutrient treatments. However, RPE results did indicate a significant difference in effort sensations resulting from the various treatments. The greatest mean RPE values reported were for the CTR trials, followed by the MCT oil trials. The sensation of nausea reported by most subjects after ingesting MCT oil, was possibly the main contributing factor affecting the rate of perceived effort reported. The lowest values for mean perceived effort, noted in both trials containing Polycose (P and MP), again support its beneficial metabolic effect in the final phases of endurance exercise when given during exercise.

The mean serum glucose level generally decreased after 90 minutes of exercise and was even found to reach hypoglycemic levels at exhaustion for one subject during the CTR trials. Again, this could be the primary cause for exhaustion during this treatment (Ivy et al, 1979). The significantly lower glucose values found during the MCT oil treatment, as compared to Polycose ($p < 0.05$), may be related to several factors. MCT oil ingestion has been shown to reduce liver glucose output unrelated to increased insulin secretion or enhanced glucose utilization (Tantbhedhyankul et al, 1967). The hyperketonemia noted after MCT oil ingestion may promote insulin secretion,

resulting in diminished glucose output by the liver (Tantbhedhyankul et al, 1967).

When carbohydrate (Polycose) was given with MCT oil (MP), glucose tolerance was not impaired but possibly enhanced in its uptake (Tantibhedgyankul et al, 1967). This seemed to be indicated by the glucose levels noted in the present study for the MP trials. When given alone Polycose tended to maintain slightly higher serum glucose levels during exercise.

MCT oil was found to provide the greatest mean FFA supply. Since the level of plasma FFA levels directly affects the rate of FFA oxidation (Ivy et al, 1979), it can be assumed that MCT oil provided an increased supply of FFA for energy during this endurance exercise bout. However when carbohydrate was given with the MCT oil (MP trial) this effect on serum FFA levels and thus muscle fuel utilization was possibly reduced. Ivy et al. (1980) also found that when MCT oil was given to subjects with carbohydrate one hour prior to exercise, FFA metabolism was inhibited.

Polycose ingestion alone, during exercise, caused the largest reduction in amount of serum FFA's available for metabolism up through minute 90 of trial P. Ivy (1979) has indicated that when carbohydrate (90 g of glucose polymer) was given during exercise, lipolysis was diminished. This could be due to an increase in arterial glucose and an increase in plasma insulin, or both (Miller, 1984). This

was in contrast to a study by Costill et al. (1973), in which glucose given (31.8 gm) at 30 minutes into exercise, followed by 60 minutes of exercise resulted in a raised FFA level. This indicated that plasma insulin was probably not increased in response to the oral glucose given during exercise. Possibly the greater glucose load (94.7 gm) in the present study elicited a greater insulin secretion response, therefore inhibiting FFA mobilization.

In the present study, after 90 minutes, the plasma FFA level did show a noticeable rise to exhaustion and continued to increase for the P and C treatments in the 30 minutes after exercise (Figure 3). This may have occurred since the effect of the last carbohydrate ingestion (40 minutes into exercise) was diminished by this time. Rodahl et al. (1964) showed that when 84 gm of glucose was given during exercise, there was a temporary decrease, followed by a rapid increase in FFA after the effect of insulin diminished.

In conclusion, in spite of the achieved and desired increase in serum FFA levels during exercise after MCT oil ingestion in comparison to the other treatments this treatment is not recommended in the quantities used in this study for the athlete because of its tendency to cause gastrointestinal distress in some individuals. In agreement with other studies, Polycose tended to enhance exercise performance, though not significantly, particularly delaying

a drop in serum glucose. Although the combination of MCT oil and Polycose would theoretically enhance endurance performance due to an increased supply of both FFA and glucose available for muscular metabolism, no evidence to support this dietary strategy was found in this investigation.

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Table 1. Physical Characteristics of Subjects Prior to Initial Endurance Testing.

Subject	Ht (cm)	Wt (kg)	Age (years)	miles per wk	Max VO_2 (ml/kg)	at 70% VO_2 (kg/kgm)
1	175.5	72	22	200	66.6	2.3
2	172.0	62	31	60	57.7	2.0
3	178.0	84	28	165	56.3	2.3
4	178.0	70	30	50	59.4	2.0
5	178.0	71	22	100	54.0	2.0
6	179.0	67	22	75	69.0	2.2
7	178.0	67	23	175	64.3	2.2

(miles per wk = average biked)

Table 2. Statistical summary of Analysis of Variance with Repeated Measures for FFA, Glucose, HR, R, RPE, and VO2 through minute 60 of exercise.

	FFA			Glucose			HR			R			RPE			VO2	
Source	df	MS	F	df	MS	F	df	MS	F	df	MS	F	df	MS	F	df	MS	F
error	51	.00017		54	276.3		108	63.1		90	.00077		75	33.85		90	6.2	
person	6	.00312	18.4*	6	988.8	3.6*	6	2846.0	45.1*	6	.01385	17.9*	5	4.62	10.2*	6	242.4	38.9*
trt	3	.00062	3.7*	3	764.7	2.8*	3	126.3	2.0	3	.00163	2.1	3	3.17	7.0*	3	8.2	1.3
time	3	.00099	5.9*	3	48.2	0.2	6	24213.6	383.8*	5	.00132	1.7	5	27.22	60.3*	5	11.6	1.9
trt X time	9	.00044	2.6*	9	133.6	0.5	18	15.3	0.2	15	.00165	2.1*	15	0.41	0.9	15	9.7	1.6

(* indicates significantly different at alpha=0.05; df = degrees of freedom; MS = Mean Square; F = F value; FFA = free fatty acid; HR = heart rate; R = respiratory exchange ratio; RPE = rate of perceived exertion.)

Table 3. Mean and S.E. values for heart rate and respiratory exchange ratios during exercise.

Time	0	10	20	30	40	50	60	70	80	90	100	110	120
						Heart rate (bpm)							
C	76+4 (7)	151+4 (7)	153+5 (7)	153+5 (7)	155+5 (7)	154+4 (7)	155+5 (7)	152+6 (6)	154+5 (6)	159+6 (4)	167 (1)	167 (1)	170 (1)
M	79+11 (7)	152+5 (7)	154+5 (7)	154+5 (7)	156+5 (7)	154+5 (7)	152+4 (7)	152+6 (4)	153+4 (5)	150+6 (3)	156+4 (2)	-----	-----
P	70+5 (7)	153+4 (7)	157+4 (7)	157+4 (7)	158+4 (7)	158+5 (7)	159+5 (7)	159+5 (6)	161+5 (6)	158+5 (5)	161+6 (3)	156+7 (2)	167 (1)
MP	73+3 (7)	151+5 (7)	154+5 (7)	156+5 (7)	157+5 (7)	156+6 (7)	157+6 (7)	156+6 (7)	153+6 (5)	152+7 (3)	153+7 (3)	145 (1)	145 (1)
						Respiratory exchange ratio							
C	-----	.87+.01 (7)a	.88+.01 (7)	.89+.02 (7)	.86+.01 (7)	.88+.01 (7)	.86+.01 (7)	.88+.02 (6)	.86+.02 (6)	.92+.02 (4)	.95 (2)	-----	-----
M	-----	.87+.02 (7)b	.86+.02 (7)	.89+.02 (7)	.88+.02 (7)	.86+.02 (7)	.86+.02 (7)	.85+.02 (5)	.84+.02 (5)	.86+.04 (3)	.84+.02 (2)	-----	-----
P	-----	.87+.02 (7)c	.84+.03 (7)a	.88+.02 (7)	.87+.02 (7)	.89+.02 (7)	.89+.02 (7)	.87+.02 (6)	.89+.02 (5)	.93+.02 (4)	.89+.05 (3)	.88+.06 (3)	.95 (1)
MP	-----	.91+.02 (7)abc	.89+.01 (7)a	.88+.01 (7)	.87+.01 (7)	.87+.01 (7)	.88+.01 (7)	.86+.01 (5)	.86+.01 (5)	.86+.02 (3)	.85+.02 (3)	.87+.04 (2)	.83 (1)

Values are means + S.E., values in parenthesis are the number of subjects used to compute the mean.

Values with the same letter are significantly different at $p<.1$.

C, control; M, MCT; P, glucose polymer; MP, MCT + glucose.

Table 4. Mean and S.E. values for serum metabolites and results of one-way ANOVA during exercise.

Time	0	15	30	60	90	120	EXH	POST
				Glucose (mg%)				
C	90.7±3.7 (7)	96.4±7.1 (7)	104±4.3 (7)	99.1±6.5 (7)	98.2±6.0 (5)	65 (1)	99.6±6.4 (7)	91.4±4.9 (7)ab
M	92.6±5.1 (7)	92.6±5.8 (7)a	95.7±2.4 (7)	97.1±5.0 (7)	85.9±3.9 (4)	---	95.4±5.3 (7)a	90.0±3.4 (7)cd
P	101.4±3.0 (7)	103.2±13.6 (7)a	106.4±9.6 (7)	107.3±3.6 (7)	114.8±3.4 (5)	105 (1)	112.7±4.8 (7)a	119.1±4.7 (7)ad
MP	104.6±6.0 (7)	99.7±4.1 (7)	104.3±5.7 (7)	99.1±5.2 (7)	96.8±4.1 (4)	89 (1)	101.6±4.8 (7)	108.1±7.6 (7)bc
				Free fatty acids (mM)				
C	.38±.10 (7)	.34±.07 (7)	.38±.07 (7)	.55±.17 (7)a	.64±.17 (5)	.94 (1)	.83±.16 (7)	1.00±.27 (7)a
M	.39±.03 (7)	.36±.07 (7)	.47±.12 (7)	.69±.12 (7)abd	.95±.33 (3)	---	1.74±.21 (7)	.76±.21 (7)
P	.46±.08 (7)	.36±.08 (7)	.40±.09 (7)	.34±.06 (7)acd	.35±.07 (5)	.50 (1)	.63±.13 (7)	.68±.22 (7)
MP	.38±.08 (7)	.35±.05 (7)	.39±.09 (7)	.41±.10 (7)bc	.38±.08 (4)	.56 (1)	.68±.22 (7)	.53±.11 (7)a

Values are means ± S.E., values in parenthesis are the numbers of subjects used to compute the mean. Values with the same letter are significantly different at $p<.1$. C, control; M, MCT; P, glucose polymer; MP, MCT + glucose polymer; EXH, exhaustion.

FIGURE LEGENDS

FIG. 1. Mean time to exhaustion for treatments MCT oil (M), Polycose (P), MCT oil with Polycose (MP), and Control (CTR). No significant difference was found between treatments ($p < 0.05$). Bar extension lines represent S.D.

FIG. 2. Mean VO_2, R, and HR values for treatments MCT oil (•), glucose polymer (◦), MCT oil and glucose polymer (▲), and Control (▪) during exercise.

Fig. 3. Mean serum glucose and free fatty acid values for treatments MCT oil (•), glucose polymer (◦), MCT oil and glucose polymer (▲), and Control (▪), during, at exhaustion, and 30 minutes post exercise.

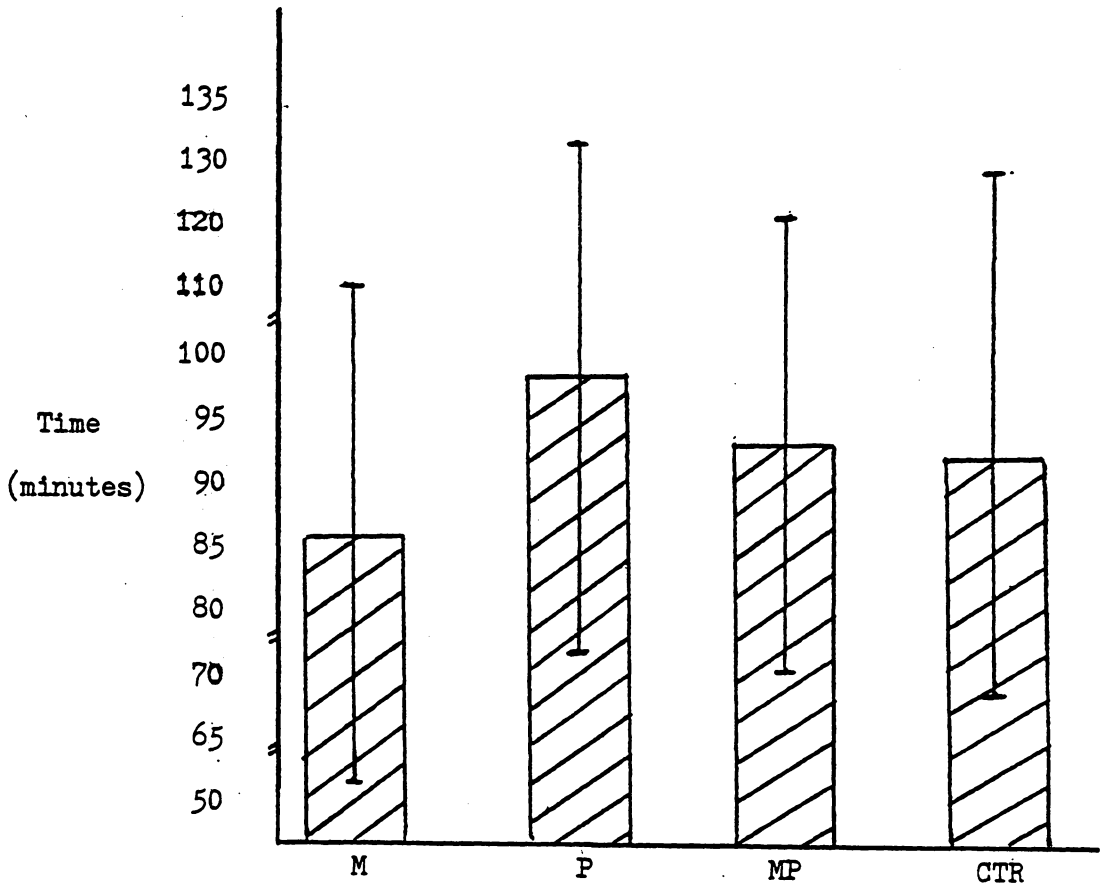

Figure 1.

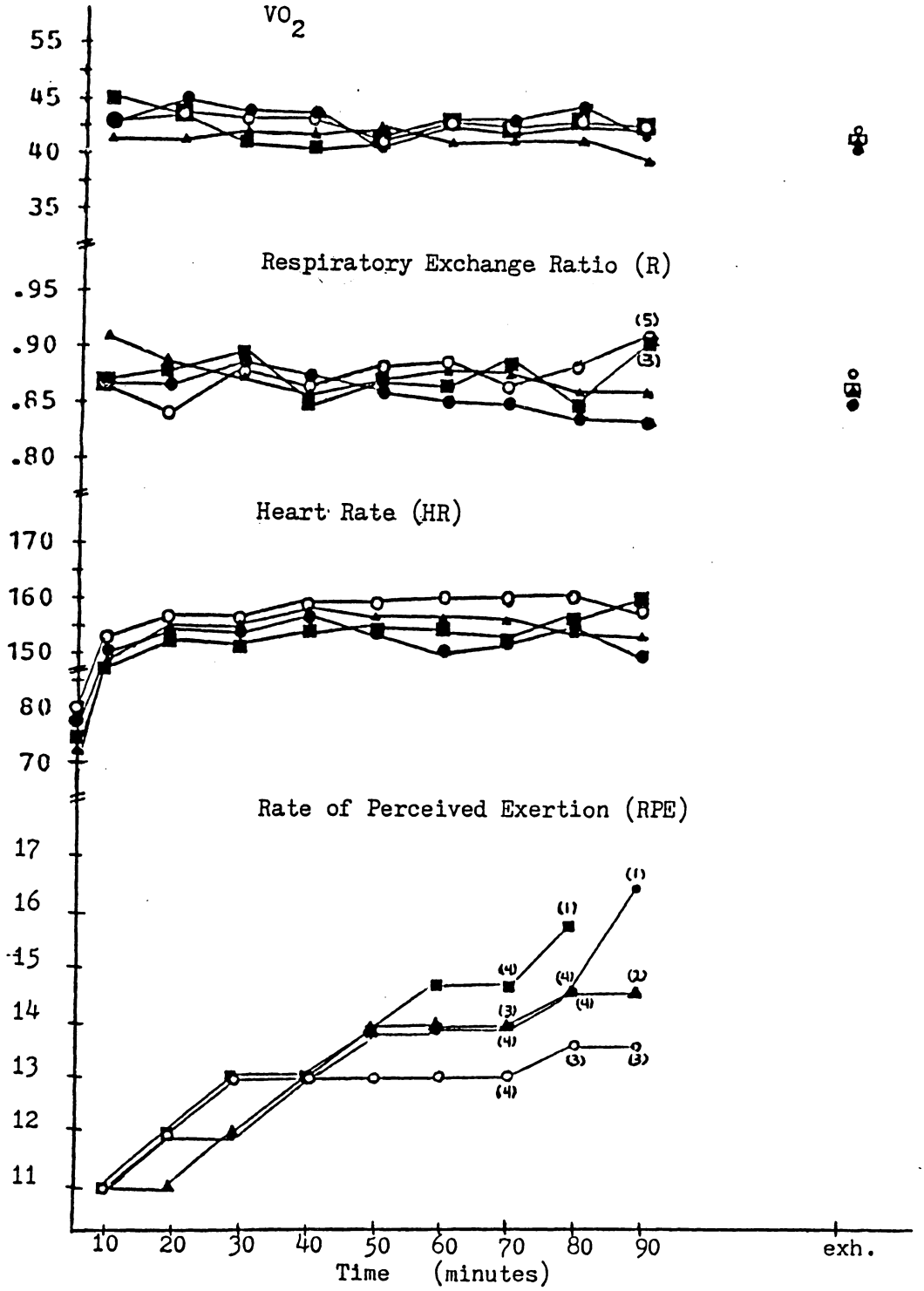
VO2
55
45
40
35
Respiratory Exchange Ratio (R)
.95
.90
.85
.80
Heart Rate (HR)
170
160
150
80
70
Rate of Perceived Exertion (RPE)
17
16
15
14
13
12
11
10
20
30
40
50
60
70
80
90
exh.
Time (minutes)

Figure 2.

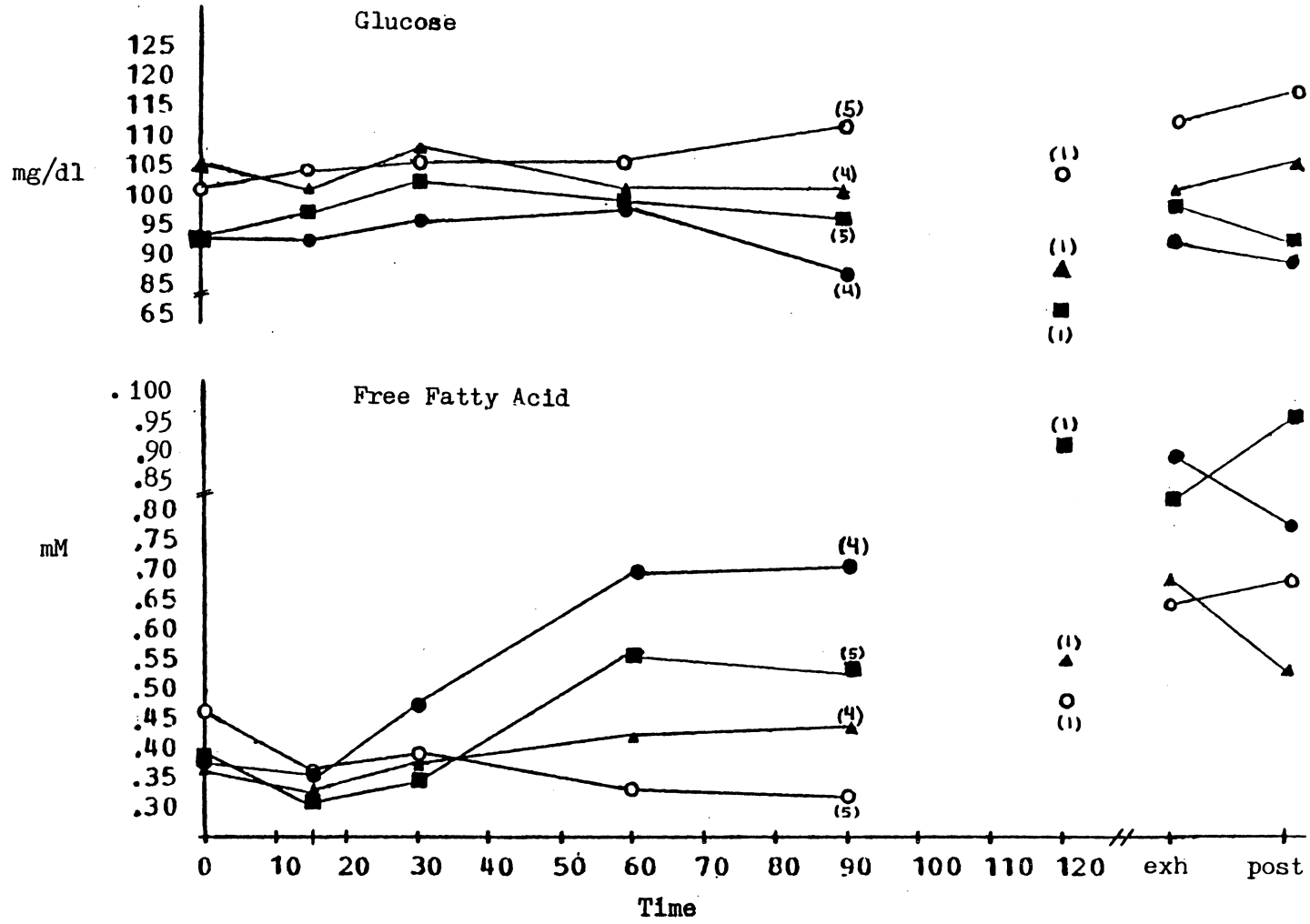
Glucose
mg/dl
125
120
115
110
105
100
95
90
85
65
(5)
(4)
(5)
(4)
(1)
(1)
(1)
Free Fatty Acid
mM
.100
.95
.90
.85
.80
.75
.70
.65
.60
.55
.50
.45
.40
.35
.30
(4)
(5)
(4)
(5)
(1)
(1)
(1)
0
10
20
30
40
50
60
70
80
90
100
110
120
exh
post
Time

Figure 3.

Chapter IV

SUMMARY

Muscle glycogen depletion and hypoglycemia have been shown to be major causes of fatigue during endurance exercise (Costill et al, 1977; Fordtran et al, 1967; Hickson et al, 1977). A hypoglycemic reaction has been shown to result when glucose was ingested prior to exercise, therefore reducing time to exhaustion (Bergstrom & Hultman, 1972). Yet if given during exercise, blood glucose levels have been maintained, sparing muscle glycogen. Ivy et al. (1983) has shown that time to exhaustion can be significantly increased when carbohydrate (Polycose, 120 gms) was given during exercise. Also, raising serum FFA levels, tended to spare muscle glycogen due to increased utilization of FFA for energy, enhancing the capacity for endurance exercise (Bellet et al, 1968; Costill et al, 1978; Ivy et al, 1979).

Supplying a high fat meal prior to endurance exercise has not been shown to improve endurance performance due to resulting acidosis and a low level of body glycogen stores established prior to exercise (Asmussen, 1965). But, glycogen stores can be assumed to be high if a high carbohydrate diet is consumed for several days prior to an event (Bergstrom et al, 1972). Costill et al. (1978 and 1977) has shown that, with caffeine ingestion and heparin

injections prior to exercise, serum FFA levels can be increased and allow for a muscle glycogen sparing effect and therefore enhanced endurance performance.

MCT oil, a rapidly absorbed and metabolized source of lipid could be another means to spare muscle glycogen. The resulting medium chain fatty acids (MCFA) are quickly transported to the liver for metabolism and oxidation into CO_2, water, acetate, and ketone bodies (KB) therefore increasing the concentration of KB. This resulted in increased insulin secretion which may lower glucose levels, although in the presence of ketone bodies, glucose transport and utilization has been shown to decrease (Owen et al, 1967). These ketone bodies formed after MCT oil ingestion can also serve as an energy source to the central nervous system therefore sparing glucose utilization (Owen et al, 1967).

Ivy et al (1980) gave 30 gms MCT oil with cereal prior to a 60 minute exercise bout. Ketone body production was found to be markedly increased above that when just cereal was given, and was therefore probably being greatly relied on for muscle utilization. Results in the MCT with cereal trial given prior to exercise indicated only a slight increase in serum FFA levels above that found in the cereal trial. However, R data indicated that carbohydrate utilization was the same for both the MCT with cereal test

as well as the cereal test alone. Ivy et al (1980) concluded that the ketone bodies did not contribute to energy needs of exercise.

MCT given alone and with a glucose polymer (Polycose) during endurance exercise has not been studied. Since FFA oxidation is mainly a function of their plasma concentration, and since the proportion of lipids given to energy metabolism is greater with increased FFA levels, carbohydrate utilization should be reduced, causing a glycogen sparing action. Since MCT may increase hepatic FFA synthesis and reduce ketone clearance (Greenberger, 1969), it was the intent of this study to determine what beneficial effects there may be of giving MCT oil alone and with Polycose, during endurance exercise, on total exercise time and on blood FFA and glucose.

In this study seven male bicyclists were used as subjects. A graded exercise bicycle test was performed to obtain individual maximum O_2 consumption (VO_2 max). The four endurance test trials were performed at 70% (± 5) max on a stationary bicycle at 90 RPM. Each subject performed their test ride on the same day of the week at same time of day with seven days between each test ride. They consumed a high carbohydrate diet (70%) using an exchange list provided by this researcher for two days prior to each test ride.

During each test, heart rate (HR), rate of perceived exertion (RPE), VO_2, and respiratory exchange ratio (R), were measured at 10 minute increments. Blood samples (about 5 mls each) were drawn from a catheter inserted in either the cephalic or basilic forearm vein by a registered nurse at times 0, 15, and 30 minutes and every 30 minutes during exercise, at exhaustion, and at 30 minutes post-exercise. These were analyzed for serum FFA and glucose levels. One of the four test treatments was randomly administered, in three portions in a single-blind design, within the first 40 minutes of each exercise bout. The control trial included gelatin capsules containing water, and a lemonade beverage (150 ml each) sweetened with an artificial sweetener (Saccharin). The test mixtures were made up as the control with the addition of one of the test substances: 1.) MCT oil (40 gms), 2.) Polycose powder (Ross Laboratories) (94.7 gms), 3.) MCT plus Polycose (20 gms MCT oil plus 47.4 gms Polycose). MCT oil replaced water in all the capsules during the MCT containing test rides while MCT capsules replaced half of the water capsules in the MCT-Polycose test rides. Polycose powder was dissolved in the lemonade in the MCT-Polycose and Polycose only rides.

No significant difference was found between mean time to exhaustion for the four treatments, although, the mean results showed that subjects tended to have the longest exercise time when receiving Polycose (P) and the shortest

when consuming MCT oil (M) alone (with nausea, however). No significant difference was noted between treatments for R, VO_2, and HR responses ($p < 0.05$). A significant difference was found over the first 60 minutes of exercise for RPE between treatment Control and the other 3 treatments ($p < 0.05$). Repeated measures ANOVA showed that significantly higher glucose values for treatment P existed over the first 60 minutes of exercise as compared to MCT oil ($p < 0.05$). Also serum FFA values for treatment M were significantly higher than both treatment P and MP up through 60 minutes of exercise ($p < 0.05$).

Although the combination of MCT oil and Polycose would theoretically enhance endurance performance due to an increased supply of both FFA and glucose available for muscular metabolism, this dietary treatment was ineffective in prolonging exercise time.

Implications

This research may be useful for the practitioner or other researchers with the following implications:

1. MCT oil is a means of increasing serum FFA, therefore providing a possible alternate substrate for energy metabolism.
2. MCT oil did not improve endurance performance in the amount administered in this study due to the gastro-intestinal distress experienced by most of the subjects.

3. Polycose, given during exercise, is a viable source of serum glucose, and may enhance endurance performance in the quantities administered in this study.

Recommendations For Further Research:

The following recommendations are made for further research in this area:

1. MCT oil alone, given in considerably smaller portions, should be studied during endurance performance.
2. The affect of time of administering MCT oil would be of interest. MCT oil given alone prior to exercise with or without Polycose given during exercise should also be examined.
3. Structuring a more comfortable bicycle seat or else accommodating the individual subjects' bicycle seat, may be found to be beneficial in performing a more accurate exhaustive endurance exercise bout.
4. Provision of a TV or video of outdoor scenes may be found useful in keeping subjects positively distracted, again possibly allowing for greater total exercise time.
5. Ascertaining the commitment of each subject to true exhaustion for each test ride may also provide more accurate and useful results. Recruiting subjects

who are highly trained athletes who have the incentive to perform to their maximum ability may facilitate this goal.

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APPENDICIES

Appendix A

Informed Consent

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by th personnel of the Human Performance Laboratory of the Division of Health, Phsical Education and Recreatin of Virginia Polytechnic Institute and State University.

TITLE OF STUDY:

The effect of MCT oil ingestion on exercise metabolism and endurance performance.

PURPOSE:

1.) Identify changes in blood metabolites during exercise after ingestion of MCT (medium chain triglyceride) oil, Polycose (a carbohydrate), the combination of MCT and Polycose, or water.

2.) Determine the effect of these treatments on endurance time to exhaustion at a moderately intense exercise level.

MY PARTICIPATION WILL INCLUDE:

1.) record keeping of my diet intake for 3 days.
2.) performance of a maximal exercise test on a stationary bicycle.
3.) eating a prescribed diet for 2 days before all subsequent laboratory exercise bouts.
4.) completion of 4 endurance bicycle rides at a moderately intense level to voluntary exhaustion performed no less than one week apart.
5.) ingestion of 4 beverages (a different one each exercise bout) during exercise which will include (a) MCT oil, (b) Polycose, (c) MCT + Polycose, and (d) water.
6.) insertion of an indwelling catheter will be performed by a nurse before each exercise bout. Approximately 5 samples of 5 ml (1 teaspoon) each will be withdrawn during exercise and recovery.

THIS STUDY MAY PRODUCE CERTAIN RISKS AND DISCOMFORTS TO INCLUDE:

1.) temporary fatigue and possible muscle soreness during and following the exercise bouts.
2.) temporary discomfort (needle prick) during insertion of catheter.

3.) withdrawal of approximately 100 ml (less than half a cup) of blood over 4 - 5 weeks.
4.) possible mild stomach cramps or diarrhea due to beverage ingestion.

PERSONAL BENEFITS MAY BE EXPECTED INCLUDING:
1.) $40.00 for participation
2.) information regarding your current exercise capacity and nutritional profile.

I understand that any data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or eompensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the local hospital for emergency service.

I have read the above statement and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Appendix A, continued

Scientific inquiry is indispensable to the advancement of knowldege. Your paricipation in this experiment provides the investigator the opportunity to conduct meaninful scientific observations designed to make significant eductational contribution.

If you would like to receive the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

I request a copy of the results of this study.

Date_____Time_____

Participant
Signature_____

Witness_____

Project Director: Dr. Janet Walberg
Telephone: 961-7545

HPER Human Subjects Chair: Dr. Don Sebolt
Telephone: 961-5104

Dr. Charles Waring, Chairman, Institutional Review Board for Research Involving Human Subjects. Telephone: 961-5284.

Appendix B

1000 Calorie Base Diet for 70% Carbohydrate Diet

(used in multiples according to individual subjects' normal caloric intake)

70% carbohydrate = 700 calories
= 175 grams
12% protein = 120 calories
= 30 grams
18% fat = 180 calories
= 20 grams

FOOD	NUMBER OF SERVINGS	PROTEIN	FAT	CARBOHYDRATE	CALORIES
Milk (2%)	1.5	12	7.5	18	202
Fruit	6	-	-	60	240
Meat	0.5	3.5	2.5	-	35
Bread	6	12	-	90	420
Vegetable	1.5	3	-	7.5	54
Fat	2	-	10	-	90

(Appendix B, continued)

Sample of Supplement Exchange List Given to Subjects

MEAT (examples of 1 meat exchange:)
hotdog, 1
peanut butter, 2 tablespoons
egg, 1

BREAD (examples of 1 bread exchange:)
bagel, 1 small
cooked cereal, 1/2 cup

FRUIT (examples of 1 fruit exchange:)
orange juice, 1/2 cup
banana, 1/2 small

VEGETABLE (examples of 1 vege exchange:)
green beans, 1/2 cup
broccoli, 1/2 cup

MILK (examples of 1 milk exchange:)
skim milk, 1 cup
plain yogurt, 1 cup

FAT (examples of 1 fat exchange:)
mayonnaise, 1 teaspoon
margarine, 1 teaspoon

Appendix C

Free Fatty Acid Procedure

Steps:

1. Put .2 ml plasma or saline in large diameter tephlon capped test tubes.

2. Add 3 ml Chloroform-Heptane-Methanol (CHM) into the standard and sample test tubes. CHM is made by mixing 49 ml of chloroform, 49 ml of heptane, and 2 ml methanol. Add 2.985 and 2.4 ml CHM in the .01 and .40 micromoles of palmitic acid tubes, respectively.

3. Add the standard (palmitate) in the .01 and .40 concentration tubes in the amount of .015 ml and .60 ml, respectively.

4. Mix on rotator at 125 RPM for 10 minutes. Do no vortex.

5. Add 1 ml of copper reagent [.5 M Cu(No3)2]. This is made by adding 10 ml 1M triethanolamine, 3.5 ml 1N NaOG and then adjusting to 100 ml with saturated NaCl in water, pH of 8.3.

6. Rotate at 125 RPM for 10 minutes.

7. Centrifuge for 20 minutes at 2.5 RPM.

8. Turn on mass spce to allow for light to warm.

9. Remove 2 ml of the clear upper phase to a clean cuvette.

10. With samples timed 1 minute apart, add .5 ml of color reagent (Na diethyldithiocarbamate) and vortex. Color reagent can be made by adding 22 mg of Na diethldithiocarbamate to 10 ml butanol.

11. Read at OD 436 at 10 minutes after the addition of color reagent.

Appendix D

Serum Glucose Procedure

Steps:

1. Label three or more tubes or cuvets as follows: BLANK, STANDARD, TEST 1, TEST 2. ETC.

2. To BLANK, add: 0.5 ml water. To STANDARD, add: 0.5 ml of a 20-fold dilution of Glucose Standard Solution, Stock No. 635-100 (1 part solution plus 19 parts water), or 0.025 ml of standard plus 0.5 ml water. To each TEST, add: 0.5 ml of a 20-fold dilution of sample (1 part serum or plasma plus 19 parts water), or 0.025 ml of sample plus 0.5 ml water.

3. To each tube, add: 5.0 ml of Combined Enzyme-Color Reagent Solution (Reagent A"). Mix each tube thoroughly.

4. Incubate all tubes at 37 C for 30 ± 5 minutes or at room temperature for 45 minutes.

5. At the end of incubation period, remove all tubes from water bath. Read A of STANDARD and TEST, using BLANK as reference at 425 - 475 nm. Readings should be completed within 30 minutes.

6. Calculate your TEST values as follows: Serum Glucose (mg/100 ml)= (A Test divided by A Standard) X 100. (note: if TEST result is greater than 300 mg/100ml, repeat assay using a 40-fold rather than a 20-fold dilution of sample in Step 2 above. Multiply new result by 2.

Appendix E

VO_2 Max Test Protocol

1. Adjust seat height to individual subjects' preference.

2. Obtain resting heart rate and blood pressure.

3. Adjust headgear to support breathing valve and breathing tube on inspired side of the system; fit subject with breathing valve mouthpiece and nose clip.

4. Set metronome at 90 RPM and allow subject to freewheel for one minute to establish pace.

5. Set initial work load at 150 kgm/min (0.5 kp) at minute 0.
   Increase to 300 kgm/min (1 kp) at minute 2;
   600 kgm/min (2 kp) at minute 4;
   750 kgm/min (2.5) at minute 6;
   900 kgm/min (3) at minute 8;
   1050 kgm/min (3.5) at minute 10;
   1200 kgm/min (4) at minute 12.

6. Measure heart rate at 15 seconds into each minute.

7. Measure blood pressure and rate of perceived exertion at minute 1:30 of each work load.

8. Read VE ATPS, O_2 and CO2 measurements on every minute.

9. Obtain immediate post exercise and minute 2, 4, and 6 postexercise values for blood pressure and heart rate.

10. Claculate data for ralative max VO_2 (ml/kg-min).

Appendix F

VO_2 Max/ Endurance Bike Ride Responses

Subject Name_____ Test_____ Date_____ Time_____

PBar_____ mmHg PH2O_____ TGas_____

Subject Ht_____ Subject Wt_____(before ex.) _____(post)

Rest BP_____ Rest HR_____ Subject bike-seat ht_____

Test Measures

Time (min)	KG	VEATPS	O_2	CO2	HR	BP	RPE	Comments

IPE _____ _____ min. 4 _____ _____

min. 2 _____ _____ min. 6 _____ _____

Appendix G

Individual subjects' heart rate values during exercise.

Subjects	Trt	Time (minutes)												TRT
		0	10	20	30	40	50	60	70	80	90	100	110	120	130
1	M	51	135	140	140	140	140	135	140	142	140	-	-	-	-
1	P	68	135	140	145	140	138	138	140	142	142	-	-	-	-
1	MP	69	140	140	140	140	141	145	140	140	-	-	-	-	-
1	CTR	75	140	140	140	135	140	140	130	135	140	-	-	-	-
2	M	135	160	167	165	167	167	150	-	-	-	-	-	-	-
2	P	97	155	167	167	167	167	170	167	170	167	167	167	167	167
2	MP	66	160	160	167	167	167	167	167	167	167	167	-	-	-
2	CTR	75	160	160	165	165	155	165	167	165	167	167	167	170	170
3	M	87	145	145	150	150	150	150	150	150	160	160	-	-	-
3	P	68	145	145	145	150	150	150	150	150	150	150	145	-	-
3	MP	70	135	145	145	145	142	142	140	145	145	148	-	-	-
3	CTR	70	140	140	140	150	145	145	145	150	-	-	-	-	-
4	M	65	150	145	150	150	145	145	150	150	150	152	-	-	-
4	P	78	160	167	167	165	167	167	167	167	-	-	-	-	-
4	MP	78	140	140	145	150	140	145	145	145	145	145	145	-	-
4	CTR	65	140	145	150	150	155	150	150	150	-	-	-	-	-
5	M	70	150	167	167	167	167	167	167	167	-	-	-	-	-
5	P	66	167	167	155	167	167	167	165	167	165	167	-	-	-
5	MP	88	167	177	175	175	177	185	177	-	-	-	-	-	-
5	CTR	100	167	170	167	170	170	170	-	-	-	-	-	-	-
6	M	50	145	145	140	145	145	150	150	155	-	-	-	-	-
6	P	130	160	165	167	165	167	167	167	167	167	-	-	-	-
6	MP	63	150	150	155	155	155	150	155	167	-	-	-	-	-
6	CTR	70	150	150	145	145	145	145	150	155	160	-	-	-	-
7	M	95	177	167	167	170	167	-	-	-	-	-	-	-	-
7	P	50	150	150	150	155	150	155	-	-	-	-	-	-	-
7	MP	80	165	165	167	167	167	167	170	-	-	-	-	-	-
7	CTR	80	160	167	167	167	165	167	167	167	167	-	-	-	-

(Trt=treatment; M=MCT oil; P=glucose polymer; MP=MCT oil with glucose polymer; CTR=Control.)

Appendix H

Individual subjects' rate of perceived exertion values during exercise.

Subject	TRT	Time (minutes)									
		10	20	30	40	50	60	70	80	90	100
1	M	11	12	12	12	12	12	12	14	17	-
1	P	11	12	12	12	12	12	13	14	15	-
1	MP	11	12	12	13	15	15	-	-	-	-
1	CTR	11	11	11	12	12	-	-	-	-	-
2	M	11	12	13	14	15	15	-	-	-	-
2	P	11	12	12	12	12	12	13	14	15	-
2	MP	10	10	11	11	12	12	13	14	15	-
2	CTR	-	-	-	-	-	-	-	-	-	-
3	M	10	11	12	14	14	14	14	15	-	-
3	P	11	11	12	12	13	14	14	13	12	-
3	MP	11	11	11	11	12	13	-	-	-	-
3	CTR	11	12	12	12	13	14	14	-	-	-
4	M	11	12	12	13	13	14	14	14	-	-
4	P	-	-	-	-	-	-	-	-	-	-
4	MP	10	11	11	12	13	13	13	13	14	16
4	CTR	-	-	-	-	-	-	-	-	-	-
5	M	11	12	12	13	13	13	-	-	-	-
5	P	-	-	-	-	-	-	-	-	-	-
5	MP	11	12	13	13	14	15	-	-	-	-
5	CTR	12	12	13	13	15	17	-	-	-	-
6	M	12	13	13	13	13	14	14	15	-	-
6	P	11	12	13	13	13	13	-	-	-	-
6	MP	13	13	13	13	14	14	15	16	-	-
6	CTR	12	13	14	14	14	15	15	-	-	-
7	M	11	12	12	13	14	14	-	-	-	-
7	P	11	12	13	13	15	16	-	-	-	-
7	MP	-	-	-	-	-	-	-	-	-	-
7	CTR	11	12	13	14	14	15	15	16	-	-

Appendix I

Individual subjects' respiratory exchange ratio values during exercise.

Subject	TRT	Time (minutes)											
		10	20	30	40	50	60	70	80	90	100	110	120	130
1	M	.84	.86	.90	.82	.87	.85	.86	.84	.90	.88	-	-	-
1	P	.87	.87	.88	.88	.89	.89	.89	.89	.91	-	-	-	-
1	MP	.88	.83	.82	.84	.84	.86	.86	.86	-	-	-	-	-
1	CTR	.83	.89	.91	.85	.85	.88	.86	.83	.88	-	-	-	-
2	M	.94	.89	.95	.92	.93	.85	-	-	-	-	-	-	-
2	P	.99	.96	.96	.97	.97	1.00	.91	.95	.99	.98	.97	.95	.96
2	MP	.95	.91	.92	.89	.90	.94	.94	.91	.90	.90	.91	-	-
2	CTR	.88	.88	.92	.85	.87	.88	.93	.89	-	-	-	.87	.87
3	M	.82	.80	.85	.85	.80	.83	.81	.81	.77	.80	-	-	-
3	P	.85	.81	.84	.90	.86	.86	.88	.88	.86	.88	.86	-	-
3	MP	.97	.90	.89	.86	.84	.85	.84	.82	.85	.84	-	-	-
3	CTR	.89	.87	.80	.85	.82	.82	.82	.80	-	-	-	-	-
4	M	.89	.79	.82	.79	.80	.79	.83	.78	.78	.78	-	-	-
4	P	.87	.84	.86	.85	.91	.91	.88	.87	-	-	-	-	-
4	MP	.84	.84	.86	.84	.84	.86	.87	.84	.84	.82	.83	.83	-
4	CTR	.86	.88	.88	.89	.93	.84	.87	.82	-	-	-	-	-
5	M	.85	.93	.93	.93	.85	.88	.90	.87	-	-	-	-	-
5	P	.81	.75	.92	.84	.84	.84	.82	.8	.80	.82	.82	-	-
5	MP	.92	.92	.87	.86	.87	.86	.84	-	-	-	-	-	-
5	CTR	.88	.87	.95	.83	.87	.84	-	-	-	-	-	-	-
6	M	.85	.86	.87	.88	.87	.92	.83	.90	.90	-	-	-	-
6	P	.86	.85	.83	.82	.94	.90	.90	.91	.95	-	-	-	-
6	MP	.92	.88	.90	.91	.87	.89	.91	.87	-	-	-	-	-
6	CTR	.86	.85	.89	.89	.89	.88	.91	.93	.94	-	-	-	-
7	M	.90	.90	.90	.94	.88	-	-	-	-	-	-	-	-
7	P	.84	.78	.86	.85	.84	.83	.84	-	-	-	-	-	-
7	MP	.89	.92	.92	.89	.91	.91	-	-	-	-	-	-	-
7	CTR	.91	.93	.89	.89	.90	.89	.87	.87	.88	-	-	-	-

(Trt=treatment; M=MCT oil; P=glucose polymer; MP=MCT oil with glucose polymer; CTR=Control.)

Appendix J

Individual subjects' relative VO2c values during exercise.

Subject	TRT	Time (minutes)											
		10	20	30	40	50	60	70	80	90	100	110	120	130
1	M	48	50	48	49	43	49	43	47	43	41	-	-	-
1	P	43	42	41	41	40	45	44	44	43	-	-	-	-
1	MP	41	47	47	45	45	44	47	44	-	-	-	-	-
1	CTR	55	47	48	44	45	48	47	48	49	-	-	-	-
2	M	45	47	46	46	41	44	-	-	-	-	-	-	-
2	P	43	43	45	43	39	40	46	47	43	41	44	43	44
2	MP	40	42	42	45	43	42	42	42	42	42	41	-	-
2	CTR	45	49	42	44	44	44	44	45	41	-	-	42	45
3	M	42	45	42	41	41	42	40	40	45	40	-	-	-
3	P	37	40	40	36	38	39	36	37	36	36	36	-	-
3	MP	37	37	38	39	39	38	37	40	39	37	-	-	-
3	CTR	42	42	41	43	39	39	40	40	-	-	-	-	-
4	M	38	43	39	-	-	41	36	36	38	39	-	-	-
4	P	48	44	44	44	43	43	43	46	-	-	-	-	-
4	MP	40	36	41	39	39	33	41	38	38	42	37	39	-
4	CTR	39	41	39	36	43	45	43	49	-	-	-	-	-
5	M	43	45	42	44	41	48	43	45	-	-	-	-	-
5	P	35	37	32	38	38	38	38	37	38	39	-	-	-
5	MP	40	40	40	41	38	40	40	-	-	-	-	-	-
5	CTR	40	36	37	40	42	36	-	-	-	-	-	-	-
6	M	43	45	45	43	43	43	-	45	42	-	-	-	-
6	P	55	-	-	52	51	51	51	51	51	-	-	-	-
6	MP	50	48	47	48	54	51	48	52	-	-	-	-	-
6	CTR	43	44	42	43	41	46	42	41	42	-	-	-	-
7	M	41	41	43	38	39	-	-	-	-	-	-	-	-
7	P	42	42	42	44	44	44	42	-	-	-	-	-	-
7	MP	48	44	46	45	42	43	-	-	-	-	-	-	-
7	CTR	51	49	45	43	43	42	42	39	39	-	-	-	-

(Trt=treatment; M=MCT oil; P=glucose polymer; MP=MCT oil with glucose polymer; CTR=Control.)

Appendix K

MCT OIL TREATMENT: Individual Subjects' Glucose and FFA values during and 30 minutes-post exercise.

Time (minute)	0	15	30	60	90	exh	30 min. post
Glucose (mg/dl)							
Subject							
1	75	80	97	94	85	82	81
2	101	101	92	125	-	125	88
3	101	84	90	90	91	96	95
4	72	82	101	89	75	88	79
5	98	88	101	100	-	89	105
6	106	89	86	86	92	92	88
7	95	124	103	-	-	96	94
$\bar{X}$ =	93	93	96	97	86	95	90
FFA (mM)							
Subject							
1	.038	.018	.020	.040	-	.050	.033
2	.048	.038	.043	-	-	.042	.042
3	.052	.058	.072	.110	.126	.154	.098
4	.025	.066	.102	.095	.130	.174	.156
5	.042	.023	.047	.078	-	.102	.140
6	.032	.024	.031	.026	.030	.030	.039
7	.035	.023	.015	-	-	.092	.024
$\bar{X}$	.039	.036	.047	.070	.072	.092	.076

Appendix L

POLYCOSE TREATMENT: Individual subjects' Glucose and FFA values during and 30 minutes-post exercise.

Time (minutes) 0	15	30	60	90	120	exh	30 min. post
Glucose (mg/dl)							
Subject							
1	106	55	89	106	112	-	102	135
2	108	94	118	94	112	105	103	111
3	101	125	95	95	124	-	129	113
4	87	118	68	116	-	-	111	124
5	107	111	102	113	121	-	118	125
6	95	-	137	118	105	-	128	128
7	106	116	136	109	-	-	98	98
$\bar{X}$ =	101	103	106	107	114	105	113	119
FFA (mM)							
Subject							
1	.020	.017	.018	.020	.023	-	.025	.020
2	.031	.030	.037	.020	.027	.050	.070	.140
3	.032	.022	.027	.025	.028	-	.063	.031
4	.047	.038	.036	.033	-	-	.042	.035
5	.065	.035	.055	.047	.060	-	.124	.164
6	.045	.031	.025	.025	.035	-	.035	.033
7	.084	.079	.085	.065	-	-	.080	.052
$\bar{X}$	.046	.036	.040	.034	.035	.050	.063	.068

Appendix M

MCT OIL & POLYCOSE: TREATMENT — Individual subjects' Glucose and FFA values during and 30 minutes-post exercise.

Time (minutes)	0	15	30	60	90	120	exh	30 min. post
Glucose (mg/dl)								
Subject								
1	85	82	91	99	-	-	97	85
2	87	99	130	107	105	-	101	88
3	112	95	115	90	93	-	100	90
4	127	112	122	84	102	89	89	117
5	128	101	99	126	-	-	116	135
6	102	114	99	95	87	-	87	118
7	91	95	95	93	-	-	121	124
$\bar{X}$ =	105	100	107	99	100	-	102	108
FFA (mM)								
Subject								
1	.023	.030	.045	.040	-	-	.070	.067
2	.037	.034	.030	.021	.023	-	.028	.030
3	.035	.025	.030	.059	.057	-	.079	.079
4	.045	.045	.034	.027	.047	.056	.056	.028
5	.076	.062	.088	.090	-	-	.190	.092
6	.043	.028	.027	.031	.025	-	.025	.060
7	.010	.023	.018	.020	-	-	.027	.015
$\bar{X}$=	.038	.035	.039	.041	.042		.068	.053

Appendix N

CONTROL TREATMENT: Individual subjects' Glucose and FFA values during and 30 minutes-post exercise.

Time (minutes) 0	15	30	60	90	120	exh	30 min. post
Glucose (mg/dl)							
Subject							
1	77	76	90	94	110	-	110	95
2	93	75	120	76	77	-	64	68
3	98	85	99	83	-	-	105	88
4	90	109	117	112	97	65	97	90
5	110	98	94	122	-	-	114	112
6	95	125	108	93	110	-	110	96
7	91	107	100	114	97	-	97	91
$\bar{X}$ =	91	96	104	99	99	-	100	91
FFA (mM)							
Subject							
1	.015	.015	.023	.022	.040	-	.040	.033
2	.050	.038	.042	.058	.094	.094	.105	.224
3	.048	.038	.067	.092	-	-	.134	.114
4	.025	.047	.050	.034	.114	-	.114	.053
5	.090	.062	.049	.138	-	-	.114	.158
6	.020	.019	.017	.018	.026	-	.026	.032
7	.024	.017	.016	.023	.047	-	.047	.089
$\bar{X}$=	.039	.034	.038	.055	.052		.083	.100

Appendix O

Individual subjects' time values for Total Exercise Time

Subject	MCT oil	Polycose	MCT oil & Polycose	Control
1	100	97	85	90
2	60	131	110	130
3	102	110	100	80
4	110	80	120	89
5	80	110	74	67
6	90	90	88	91
7	54	71	70	90

Appendix P

Statistical summary of Duncan Tables showing differences between means for FFA, glucose, HR, R, RPE, and VO_2 through minute 60 of exercise ($p < =0.05$).

FFA		Glucose		HR		R		RPE		VO_2	
DT	TRT	DT	TRT	DT	TRT	DT	TRT	DT	TRT	DT	TRT
A	M	A	P	A	P	A	MP	A	CTR	A	M
BA	CTR	BA	MP	A	MP	BA	CTR	B	M	A	CTR
B	P	BA	CTR	A	M	BA	P	CB	P	A	P
B	MP	B	M	A	CTR	B	M	C	MP	A	MP
*		*						*			

(Treatments are listeds in descending order under each dependent variable.)

* indicates dependent variable is significantly different at $p < 0.05$; FFA=free fatty acids; HR=heart rate; R=respiratory exchange ratio; R=rate of perceived exertion; DT=Duncan Table results; Trt=treatment; M=MCT oil; P=glucose polymer MP=MCT oil with glucose polymer; CTR=Control.

Appendix Q

Statistical summary of Duncan Tables showing differences between means for FFA, glucose, HR, R, RPE, and VO_2 through minute 60 of exercise ($p < 0.1$).

FFA		Glucose		HR		R		RPE		VO_2	
DT	TRT	DT	TRT	DT	TRT	DT	TRT	DT	TRT	DT	TRT
A	M	A	P	A	P	A	MP	A	CTR	A	M
B	CTR	A	MP	BA	MP	BA	CTR	B	M	BA	CTR
B	P	BA	CTR	B	M	BA	P	CB	P	BA	P
B	MP	B	M	B	CTR	B	M	C	MP	B	MP
+		+				+		+			

(Treatments are listeds in descending order under each dependent variable.)

* indicates dependent variable is significantly different at $p < 0.1$; FFA=free fatty acids; HR=heart rate; R=respiratory exchange ratio; R=rate of perceived exertion; DT=Duncan Table results; Trt=treatment; M=MCT oil; P=glucose polymer MP=MCT oil with glucose polymer; CTR=Control.

Appendix R

Based on the results of this study, the researcher:

1. Retained the null hypothesis that there would be no significant difference in time to exhaustion between endurance tests in which a water, glucose polymer, MCT oil, or combined glucose polymer/MCT oil beverage was administered.
2. Failed to retain the null hypothesis that serum glucose and serum FFA levels, heart rate (HR), respiratory exchange ratio (R), and VO_2 would not change over time during exercise for any of the treatments.
3. Failed to retain the null hypothesis that the four treatments given during exercise would not affect the serum glucose, serum FFA, VO_2, HR, or R response during exercise.

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