Medium Chain Triglyceride in Early Life: Effects on Growth of Adipose Tissue[1]

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Effects of feeding early in life a diet high in either long chain (LCT) or medium chain triglyceride (MCT) were studied on the development of adipose tissue in post-weanling rats. The diets were similar in calorie distribution and identical in nutrients except for type of fat. The caloric distribution of the two diets by percent was LCT (corn oil)/protein/carbohydrate, 70/18/12 and MCT/corn oil/protein/carbohydrate, 66/4/18/12. Male littermates with less than 5% weight difference were pair-fed the two diets randomly at age 18–20 days. One-fourth of the rats were killed at 10, 16, 22 and 28 weeks of age and analyzed for adipose depots and adipose tissue cellularity. Results showed that the LCT-fed rats were significantly heavier, with larger epididymal, retroperitoneal, omental and subcutaneous fat pads than the respective pair-fed MCT rats. Also, LCT-fed rats had larger size and number of adipocytes than MCT-fed littermates. It is concluded that the type of fat in the diet, namely LCT or MCT, when fed early in life can influence the development of adipose tissue. MCT appears less lipogenic than LCT. The mechanism for the diminished adiposity of MCT-fed rats is related to extensive oxidation of MCT and its enhancement of thermogenesis leading to lessened energy efficiency.
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Tailormade medium chain triglycerides (MCT) for human consumption have been investigated and in use for over 25 years. Several reviews have dealt with their absorption, transport, metabolism and clinical uses (1–3). In the United States, the available MCT preparations are triglycerides whose constituent medium chain fatty acids (MCFA) are octanoic acid, 8:0 (65–70%); decanoic acid, 10:0 (20–35%); hexanoic acid, 6:0 (1–2%); and dodecanoic acid, 12:0 (1–2%). In Europe, MCT preparations tend to contain lower proportions of 8:0 and higher proportions of 10:0, at times in equal amounts, with trace quantities of 6:0 and 12:0. Although MCFA do appear in naturally occurring mixed triglycerides, pure edible MCT are derived semisynthetically from coconut oil or milk fat. For example, the fatty acids of coconut oil are hydrolyzed and molecularly distilled to yield three fractions: MCFA, relatively pure 12:0 and long chain fatty acids. MCFA are purified prior to reesterification with glycerol to form MCT (4). The final product (MCT) is a clear, light yellow oil with a melting point of −5 C, made up entirely of saturated MCFA ranging in length from 6:0 to 12:0.

Recently, studies of digestion, absorption and transport of MCT have been reviewed (3). It is clear that MCT is hydrolyzed efficiently in the lumen of the small intestine under conditions adverse to the hydrolysis of LCT (Fig. 1). Unlike LCT, in the absence of pancreatic lipase MCT is absorbed into the mucosa of the intestine, where it is hydrolyzed by a mucosal lipase into MCFA, which traverse the capillaries and are transported via the portal vein into the liver. In the liver, MCFA are extensively oxidized and, for this reason, are not incorporated into lipid ester moieties of lipoproteins. Also, less than 3% of ingested MCT are transported as chylomicrons. In view of the efficiency of their absorption and the uniqueness of their mode of transport, MCT have been used in the treatment of a variety of malabsorption syndromes, including long chain fat malabsorption in premature infants (5–7).

Infants and adults fed MCT as a major proportion of calories do not incorporate MCFA into adipose tissue. Feeding MCT to premature infants as 80% of dietary fat in formula diets deriving 50% of calories from fat has been shown to improve fat absorption and nitrogen retention (5,6). The growth curve of the MCT-fed infants did not differ significantly from that of the LCT-fed infants. However, the MCT-fed infants appeared more lean and

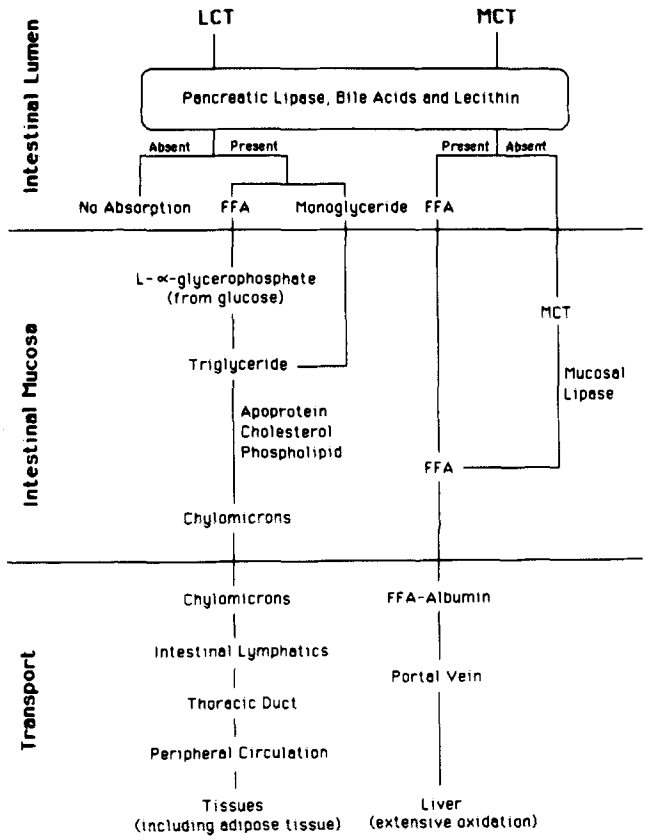

FIG. 1. Digestion, absorption and transport of long chain (LCT) and medium chain triglycerides (MCT). FFA, free fatty acid.

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less fat clinically than the LCT-fed infants. Unfortunately, studies of body composition were not done on these infants. However, it was possible to study the effect of MCT on fat deposition in the rat (8). Three groups of rats were fed either a low fat diet, a diet containing 55% (by energy) MCT and 5% corn oil, or a diet containing 60% LCT in the form of corn oil. The results showed that, unlike LCT, MCT had a reductive effect on fat depots and, like LCT, had a depressive effect on lipogenesis. In contrast, the low fat, high carbohydrate diet had an enhancing effect on lipogenesis. In another study (9) in which food intake was precisely controlled by gastrostomy, body composition was measured in rats overfed with MCT- or LCT-containing diets that provided 50% calories in excess of normal consumption. The MCT-fed rats showed significantly smaller fat depots and adipocyte size than the LCT-fed controls. In a similar study, the mechanism of the reductive effect of MCT on fat depots was related in part to increased metabolic rate and enhanced thermogenesis (10).

The mass of adipose tissue in man and animals is dependent upon the number and size of its adipocytes. It is therefore conceivable to modify the ultimate dimension of the adipose depot in the body by factors that affect adipocyte replication and/or adipocyte size. In man and rat, the number of adipocytes has been reported as fixed at a certain age (11-13). During the postnatal period of growth, adipocyte proliferation continues until a maximum is attained, whereas further growth of adipose tissue occurs primarily as a result of adipocyte enlargement and not cell division (11). However, it has been reported that the adipocyte number can be increased in adult rats by feeding a diet high in long chain fat (14). Similarly, in the Osborne-Mendel rat, a high fat diet markedly increased the adipose tissue mass without significantly altering water, protein or ash content (15). The average human newborn infant has 500 g of fat in 4 billion adipocytes, each containing 0.12 μg of fat (16). In contrast, the normal nonobese adult has 30 billion adipocytes with 0.5 μg of fat per cell, while the obese adult may harbor 1 trillion adipocytes, each containing 0.6 to 1.2 μg of fat (17). Thus, a considerable increase in adipocyte number must occur from infancy to adulthood. Little is known about the regulatory mechanisms that control the growth of adipose tissue. Nutrition appears to be important. Preweaning undernutrition has been shown to result in an ultimate reduction in the cell number of the epididymal fat body of the rat (18). While undernutrition can result in a reduction of adipocyte number (and size), such a manipulation also can produce appreciable reduction in the cell number of the central nervous system (19). It is postulated that MCT feeding in early life will reduce adipose tissue mass with concurrent preservation of adequate nutrition. It is also postulated that chylomicrons resulting from the ingestion of LCT and not MCT are important stimuli for growth of adipose tissue. As LCT digestion and absorption improve postnatally, there is rapid accumulation of fat, associated with enhanced cellularity of the adipose tissue.

MATERIALS AND METHODS

Pregnant Sprague-Dawley rats fed a standard stock diet were allowed to deliver and nurse their pups in our laboratory. At weaning (18-20 days), male littermates from the same dam with weight difference of 5% or less were pair-fed and divided into two groups, to be fed an LCT or MCT diet. MCT was provided by Capital City Products Co. (Columbus, Ohio). The diets were identical in nutrients except for the type of fat and were similar in energy distribution. The LCT diet derived 70% of total energy from corn oil; the MCT derived 66% of total energy from MCT and 4% from corn oil (to supply essential fatty acid). Both diets contained 18% of total energy as protein and 12% as sucrose. Each diet yielded 3.9 kcal/g. The composition of the diets is shown in Table 1. Diet was placed in a cup anchored to the cage and covered with a screen mass and a broad funnel-like lid. In this way spillage was negligible. The animals were pair-fed their respective diets from weaning until they were killed. The animal that ate the least (usually the MCT rat) was allowed to eat ad libitum. The exact amount of food eaten by this rat was then given to the other rat of the pair at the next feeding. The amount of diet consumed was weighed and calculated three times per week. One-fourth of the rats were killed at 10, 12, 22 and 28 weeks of age. Various fat pads were removed as follows: A ventral midline vertical incision was cut toward the head. Two cuts were made laterally from the midline incision about 1 cm above the hip. Each epididymal pad was exposed and cut at the base of the epididymis toward the epididymal vessels. The cut then followed the vessels until the whole epididymal pad was freed. The left epididymal pad was used to determine number and size of the adipocytes. The retroperitoneal fat was removed by cutting its apex

TABLE 1

Composition of Diets Containing Either Long Chain (LCT) or Medium Chain Triglyceride (MCT)

	LCT (wt %)	MCT (wt %)
Casein	17.6	17.6
Sucrose	11.4	11.4
Corn oil	30.4	1.7
MCT		31.1
Alphacel	35.8	33.4
Salt mixture[a]	3.8	3.8
Vitamin mixture[b]	1.0	1.0
Caloric density (kcal/g)	3.9	3.9
Caloric distribution (%)		
Protein	18	18
Fat	70	70
Carbohydrate	12	12

[a]USP XIV salt mixture from ICN Nutritional Biochemicals. Contains (g/100 g mixture): cupric sulfate, 0.007; ferric ammonium citrate, 1.53; manganese sulfate, 0.02; ammonium alum, 0.009; potassium iodide, 0.004; sodium fluoride, 0.051; calcium carbonate, 6.86; calcium citrate, 30.83; calcium biphosphate, 11.28; magnesium carbonate, 3.52; magnesium sulfate, 3.83; potassium chloride, 12.47; potassium phosphate diabasic, 21.88; sodium chloride, 7.71; zinc carbonate, 0.0024.

[b]Vitamin diet fortification mixture from ICN Nutritional Biochemicals. Contains (g/100 g mixture): retinyl acetate (200,000 units/g), 2.95; cholecalciferol (400,000 units/g), 0.16; tocopherol, 3.30; choline chloride, 49.20; menadione, 1.47; p-aminobenzoic acid, 3.18; niacin, 3.18; riboflavin, 0.66; pyridoxine hydrochloride, 0.66; thiamin hydrochloride, 0.66; calcium pantothenate, 1.97; biotin, 13.1 mg; folic acid, 59.0 mg; vitamin B_{12}, 0.9 mg.

at about the midinguinal point and at the medial border of the psoas muscle. The cut then followed the medial border of the psoas muscle until it reached just below the lower pole of the kidney and followed the lateral border of the kidney as far as possible toward the diaphragm. The retroperitoneal fat was freed from the underlying muscle and weighed. The left retroperitoneal fat was used to determine number and size of the adipocytes. The dorsal fat is a rectangular pad. The vertical lines ran through the shoulder from a point 1 cm above the hip to the neck. The caudal horizontal line ran from one vertical line to the other, 1 cm above the hip. The skin was carefully dissected out free of fat. Then this dorsal fat pad was carefully removed free of muscle and brown fat. It was weighed and used to determine cell number and size. The remaining subcutaneous fat was then dissected out and weighed; the omental fat was stripped off easily from the alimentary tract. Thus, the total fat was comprised of all the fat pads: right and left epididymal fat, right and left retroperitoneal fat, dorsal fat and remaining subcutaneous fat.

The cell count was determined by the method of Hirsch and Gallian (20) as follows: A certain amount of adipose tissue (one sample each from the left epididymal and retroperitoneal and two from the dorsal fat) weighing about 80 mg in several small pieces from different sites was weighed on a tared nylon mesh (200 μ) and placed in a polyethylene vial containing 15 ml of 2% osmium tetroxide in 0.05 M collidin buffer, pH 7.4. The tissue was incubated at 37 C for 48 hr. It was thoroughly washed, and the freed adipocytes were counted in a Coulter counter. The total adipocytes per piece of adipose tissue of known weight were thus calculated.

The cell size was determined as follows: One piece each of left epididymal, left retroperitoneal and dorsal fat (about 150 mg) were placed in separate 50-ml ground-glass stoppered tubes, followed by 20 ml of redistilled chloroform and 10 ml of redistilled methanol. The tube was allowed to stand in a cold room for 48 hr. Distilled water was added to the tube, which was shaken gently and allowed to stand for separation of the solution in the tube into two phases. The upper aqueous phase was removed. About 10-15 g of anhydrous sodium sulfate was added to the chloroform phase and shaken gently. Then 5 ml of the lipid phase was pipetted into a tared dish. The chloroform was allowed to evaporate completely, and the remaining lipid in the dish was weighed. Thus, the lipid content of the adipose tissue of known weight was divided by the number of cells, and in this way converted into cell size, expressed as μg of lipid (mainly triglyceride) per cell.

RESULTS

Body weight and weights of both epididymal, both retroperitoneal, total subcutaneous and omental fat pads of the two groups of rats at ages 10, 16, 22 and 28 wk are shown in Table 2. The LCT-fed rats were substantially heavier than their pair-fed MCT counterparts at all ages. The increment in body weight in the LCT over the MCT rats increases with age from 3.7% at age 10 wk to 8.5% at age 16 wk to 15% at ages 22 and 28 wk. All fat pads in the LCT group were significantly heavier than those of the pair-fed MCT rats at all ages. The increases in the weights of the epididymal, retroperitoneal and omental fat pads in the LCT group over those of the MCT group were 30-35% at age 10 wk and approximately 50% at ages 16, 22 and 28 wk. The subcutaneous fat, the largest dissectible fat pad in the rat, was 18-30% heavier in the LCT than in the MCT group at all ages. The total dissectible fat without the omental pad (so computed because the weights for the omental fat pads were not obtained at 22 and 28 wi) was 27-36% heavier in the LCT than in the MCT group at all ages.

The total lipid content of the left epididymal, left retroperitoneal and interscapular (dorsal) subcutaneous fat pads of the LCT and MCT groups at ages 10, 16, 22 and

TABLE 2

Body Weight and Weights of Various Fat Pads of Rats Pair-Fed Diets of Either 70% LCT or 70% MCT at Ages 10, 16, 22 and 28 Wk

	10 Wk		16 Wk		22 Wk		28 Wk	
	LCT	MCT	LCT	MCT	LCT	MCT	LCT	MCT
No. of rats	8	8	8	8	8	8	9	9
Body weight	269 ± 13[a]	259 ± 11	390 ± 15[c]	357 ± 14	472 ± 21[c]	407 ± 18	480 ± 15[d]	418 ± 14
Total epididymal pads	3.17[b] ± 0.43	2.10 ± 0.19	5.95[d] ± 0.46	3.12 ± 0.30	7.24[c] ± 0.89	3.87 ± 0.42	6.72[d] ± 0.48	3.69 ± 0.37
Total retroperitoneal pads	3.79[c] ± 0.55	2.47 ± 0.40	9.82[c] ± 1.05	4.57 ± 0.61	12.44[c] ± 1.92	5.91 ± 0.79	11.64[c] ± 1.77	5.58 ± 0.85
Subcutaneous pad	22.27[a] ± 2.81	16.86 ± 1.44	35.14[c] ± 2.43	24.74 ± 1.81	41.83[b] ± 4.45	30.57 ± 2.60	37.25[c] ± 3.06	30.41 ± 2.78
Omental pad	5.27[c] ± 0.55	3.78 ± 0.36	10.42[d] ± 0.59	5.32 ± 0.42				
Total fat without omental pad	29.24[a] ± 3.73	21.43 ± 1.98	50.50[d] ± 3.73	32.45 ± 2.48	61.51[c] ± 6.99	40.35 ± 3.71	55.61[d] ± 5.04	38.68 ± 3.93

Values represent mean ± SE, g.

[a]Significantly different from MCT rats at the same age: $p < .025$.

[b]Significantly different from MCT rats at the same age: $p < .01$.

[c]Significantly different from MCT rats at the same age: $p < .005$.

[d]Significantly different from MCT rats at the same age: $p < .001$.

28 wk is shown in Table 3. The LCT rats had significantly more lipid per unit weight of the three fat pads than their respective pair-fed MCT rats, except for the dorsal pad at 28 wk.

The fat cell size of the left epididymal, retroperitoneal and interscapular (dorsal) subcutaneous fat pads of the two groups at all ages is shown in Table 4. The LCT rats had significantly larger adipocytes in all depot fat pads than the MCT rats at all ages, except for the epididymal fat pad at age 10 wk and the dorsal fat pad at 28 wk.

The fat cell number of the left epididymal, retroperitoneal and interscapular (dorsal) subcutaneous fat pads of the two groups of rats at all ages is shown in Table 5. For the epididymal fat pad, the LCT rats tended to have a larger adipocyte number than the MCT rats at all ages, but the difference was significant only at 28 wk. For the retroperitoneal fat pad, the LCT rats had significantly more adipocytes at all ages. In contrast, the number of fat cells in the dorsal pad did not differ significantly between the LCT and the MCT groups.

DISCUSSION

The data show that the LCT-fed rats were significantly heavier than the respective pair-fed MCT rats at all ages. Also, the various adipose depots weighed on an average 30% to 50% more in the LCT- than in the MCT-fed rats. It is noteworthy that both groups of animals consumed the same amount of calories, and their diets, except for quality of fat content, were identical in caloric distribution and density. Under the conditions of pair-feeding, rats in the LCT group tended to finish their food, while those in the MCT group usually had food available throughout the 24 hr. Thus, the LCT-fed rats tended to be hungry and therefore more active than the MCT-fed rats. If the activity of both groups had been similar, the LCT-fed rats would have been heavier and their adipose depots larger, and the difference in the parameters between the two groups would have been more pronounced. A possible explanation for the diminished spontaneous food intake of rats fed MCT in comparison with animals

TABLE 3

Total Lipid Content of Left Epididymal, Left Retroperitoneal and Dorsal Fat Pads of Rats Pair-Fed Diets of Either 70% LCT or 70% MCT at Ages 10, 16, 22 and 28 Wk

Age (wk)	No. of rats	Left epididymal pad		Left retroperitoneal pad		Dorsal pad	
		LCT	MCT	LCT	MCT	LCT	MCT
10	8,8	84.4[e] ± 1.4	79.6 ± 0.9	82.2[c] ± 3.1	77.1 ± 2.9	79.1[b] ± 3.2	69.8 ± 1.0
16	8,8	89.5[e] ± 0.8	83.1 ± 1.1	90.0[e] ± 0.6	83.2 ± 0.8	84.1[d] ± 1.2	70.2 ± 1.9
22	8,8	88.2[d] ± 1.3	81.1 ± 0.7	86.4[a] ± 1.6	81.9 ± 0.9	77.5[e] ± 1.6	68.8 ± 1.3
28	9,9	83.9[d] ± 0.4	70.9 ± 0.9	85.2[d] ± 0.9	81.0 ± 1.2	74.2 ± 1.0	71.2 ± 1.6

Values represent mean ± SE, % w/w.

[a]Significantly different from MCT rats at the same age: $p < .05$.

[b]Significantly different from MCT rats at the same age: $p < .025$.

[c]Significantly different from MCT rats at the same age: $p < .01$.

[d]Significantly different from MCT rats at the same age: $p < .005$.

[e]Significantly different from MCT rats at the same age: $p < .001$.

TABLE 4

Fat Cell Size of Left Epididymal, Left Retroperitoneal and Dorsal Fat Pads of Rats Pair-Fed Diets of Either 70% LCT or 70% MCT at Ages 10, 16, 22 and 28 Wk

Age (wk)	No. of rats	Left epididymal pad		Left retroperitoneal pad		Dorsal pad	
		LCT	MCT	LCT	MCT	LCT	MCT
10	8,8	0.285 ± 0.030	0.221 ± 0.022	0.384[a] ± 0.048	0.298 ± 0.033	0.229[a] ± 0.028	0.158 ± 0.010
16	8,8	0.656[b] ± 0.066	0.381 ± 0.081	0.720[c] ± 0.042	0.391 ± 0.056	0.342[d] ± 0.014	0.188 ± 0.017
22	8,8	0.481[d] ± 0.024	0.280 ± 0.026	0.670[c] ± 0.055	0.429 ± 0.036	0.297[b] ± 0.022	0.188 ± 0.014
28	9,9	0.435[c] ± 0.024	0.285 ± 0.033	0.672[d] ± 0.046	0.429 ± 0.041	0.285 ± 0.022	0.231 ± 0.040

Values represent mean ± SE, μg lipid per fat cell.

[a]Significantly different from MCT rats at the same age: $p < .05$.

[b]Significantly different from MCT rats at the same age: $p < .025$.

[c]Significantly different from MCT rats at the same age: $p < .005$.

[d]Significantly different from MCT rats at the same age: $p < .001$.

TABLE 5

Fat Cell Number of Left Epididymal, Left Retroperitoneal and Dorsal Fat Pads of Rats Pair-Fed Diets of Either 70% LCT or 70% MCT at Ages 10, 16, 22 and 28 Wk

Age (wk)	No. of rats	Left epididymal pad		Left retroperitoneal pad		Dorsal pad	
		LCT	MCT	LCT	MCT	LCT	MCT
10	8,8	4.821 ± 0.488	3.892 ± 0.306	4.266[d] ± 0.342	3.204 ± 0.371	8.589 ± 0.703	6.598 ± 0.467
16	8,8	4.372 ± 0.566	4.203 ± 0.618	6.231 ± 0.510	5.244 ± 0.526	9.617 ± 0.676	10.898 ± 1.248
22	8,8	6.306 ± 0.534	5.746 ± 0.308	7.613[b] ± 0.946	5.465 ± 0.421	12.620 ± 1.676	12.081 ± 1.144
28	9,9	6.534[a] ± 0.386	5.366 ± 0.374	7.581[c] ± 0.793	5.264 ± 0.404	10.270 ± 0.609	10.606 ± 1.029

Values represent mean ± SE, × 10^6.

[a]Significantly different from MCT rats at the same age: $p < .05$.

[b]Significantly different from MCT rats at the same age: $p < .025$.

[c]Significantly different from MCT rats at the same age: $p < .01$.

[d]Significantly different from MCT rats at the same age: $p < .001$.

fed LCT ad libitum is the development of hyperketonemia during MCT feeding (2,21,22). For this reason, the present study used pair-feeding to ensure similarity in food intake in both groups of animals. Rats fed ad libitum a diet containing 20% lard were reported to be heavier than animals fed a diet containing 20% MCT and had larger epididymal fat pads. The energy requirement for weight maintenance was higher in rats fed MCT than in those fed lard (23). Pigs fed a 10% MCT diet exhibited weight gain and feed efficiency at a rate considerably lower than that of animals fed a 10% LCT diet (24). Premature infants fed a formula containing 40% of total calories from MCT gained less weight than infants fed an identical formula containing 50% of total energy from LCT (corn oil), despite the fact that the groups had the same calorie intake and that the MCT-fed infants absorbed more fat, protein, calcium and magnesium than the LCT-fed group (5,6).

This study supports the hypothesis that MCT feeding early in life results in diminished fat deposition and adipose tissue cellularity. The mechanism whereby MCT feeding results in diminished adiposity compared to LCT feeding may be related to vast differences in the modes of digestion, absorption and metabolism of the two triglycerides. In contrast to LCT, which appear in the systemic circulation as chylomicrons, the MCT-derived fatty acids are transported via the portal vein (25) to the liver, where they are extensively oxidized into CO_2 and other water-soluble metabolites, such as ketones, and are not incorporated into hepatic lipids to any appreciable extent (1,2). In the rat, the cumulative oxidation of labeled MCT over a period of 48 hr was about twice that of LCT (26). In both man and animals, the oxidation of MCT-derived fatty acids is not appreciably affected by the nutritional state. In contrast, feeding drastically reduces the oxidation of LCT-derived fatty acids (27). Thus, it appears that MCT must undergo obligatory oxidation in the liver after absorption and transport. The oxidative aqueous products other than CO_2 reach the systemic circulation and are utilized by various organs, including the adipose tissue. Theoretically the adipose tissue can synthesize long chain fatty acids from MCT-derived oxidative products. However, studies have shown that the adipose tissue in fact contains very little medium chain fatty acid (3). In contrast, the LCT moieties of chylomicrons are hydrolyzed, picked up and readily reesterified into triglycerides by the adipose tissue. Thus, the metabolic steps that lead to formation of triglycerides in adipose tissue are much simpler for LCT than for MCT. In the intact animal and in man, evidence suggests that MCT feeding is associated with enhanced thermogenesis, thus favoring diminished energy efficiency and reduced deposition of fat (10,28).

LCT-fed rats had not only larger fat depots but also higher lipid content per unit weight than MCT-fed rats. The weight increase of various fat depots in the LCT-fed rats was due to adipocytes being bigger and more numerous than in the MCT-fed rats. It is not known whether the increase in adipocyte number in the LCT group is due to actual proliferation or to fattening up of existing pre-adipocytes.

In virtually all mammalian species, the mammary gland synthesizes LCT and varying proportions of MCT (29). In rabbit milk, the predominant fat is MCT, reaching 80% of the milk fat. Smaller proportions of MCT in milk fat occur in the rat (30%), in the mouse (8%) and in man (12%). It appears that the mammary gland begins to synthesize MCT during pregnancy and maintains such synthesis during lactation through a remarkable mechanism involving an enzyme (thioesterase II) that segments long chain fatty acids (LCFA) into MCFA for incorporation into milk triglycerides (30). The fatty acid distribution on the triglyceride appears to favor digestion and absorption of the milk fat by the neonate in that the LCFA tend to occupy the 1 and 2 positions, while the 3 position is occupied by the MCFA (31,32). The net effect of this presentation of milk fat to the neonate is ease of hydrolysis and subsequent elaboration of MCFA, LCFA and long chain monoglycerides for enhanced intestinal absorption. Studies in man suggest a twofold increase in MCFA in breast milk of mothers at premature delivery and that the premature infant, when ingesting its own mother's milk for three months, receives 17% of total fat as MCFA, in contrast to 10% for the full-term infant (33). By increasing the proportion of dietary MCFA from MCT, it may be possible to circumvent the diminished

ability of the premature infant to digest and absorb LCT and concurrently to offer an easily oxidizable fat. The effect of starting MCT feeding in the neonatal period on the development of adipose tissue cellularity awaits further investigation. Our study demonstrates that the type of fat in the diet in the postweaning period can influence the development of adipose tissue cellularity. MCT feeding early in life may offer a potential measure for prevention and control of human obesity.

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