Overfeeding with medium-chain triglyceride diet results in diminished deposition of fat

Article *in* American Journal of Clinical Nutrition · February 1983

DOI: 10.1093/ajcn/37.1.1 · Source: PubMed

5 authors, including:

Allan Geliebter
Columbia University/ Touro College
180 PUBLICATIONS **6,499** CITATIONS
SEE PROFILE

Shah Hashim
University of Kuala Lumpur
21 PUBLICATIONS **760** CITATIONS
SEE PROFILE

Original Research Communications—rapid

Overfeeding with medium-chain triglyceride diet results in diminished deposition of fat[1-4]

Allan Geliebter,[5] PhD, Naji Torbay, MD, E Filippo Bracco, MD, Sami A Hashim, MD, and Theodore B Van Itallie, MD

ABSTRACT The study was designed to determine whether overfeeding rats with a diet containing medium-chain triglyceride (MCT) as the major fat source (45% of calories) would impede the expected gain in weight and body fat as compared to rats overfed with isocaloric amounts of diet containing long-chain triglyceride (LCT). For 6 wk rats were fed either MCT diet or LCT diet twice daily via a gastrostomy tube. MCT-fed rats gained 20% less weight ($p < 0.001$) and possessed fat depots weighing 23% less ($p < 0.001$) than LCT-fed rats. Mean adipocyte size was smaller ($p < 0.005$) in MCT- than in LCT-fed rats. Weights of carcass protein and water were similar for both groups as were concentrations of serum insulin and levels of physical activity. The decreased deposition of fat in the MCT-fed rats may have resulted from obligatory oxidation of MCT-derived fatty acids in the liver after being transported there via the portal vein, leaving almost no MCT derivatives for incorporation into body fat. MCT may have potential for dietary prevention of human obesity. *Am J Clin Nutr* 1983; 37: 1–4.

KEY WORDS Medium-chain triglyceride, fat deposition, obesity, overfeeding

Introduction

Medium-chain triglyceride (MCT) is an edible oil that consists of approximately 75% C8 and 25% C10 fatty acids (1). Such fatty acids are naturally occurring components of butter, coconut oil, and other palm kernel oils; coconut oil being the source for commercially prepared MCT (1). MCT has been used in the dietary treatment of various malabsorption syndromes (2) fed either as part of a formula or incorporated into foods (3) by substitution for long-chain triglyceride (LCT).

A few studies in the rat comparing the effects of feeding MCT and LCT diets (4–7) have provided evidence that MCT diets lead to less deposition of body fat than LCT diets. These studies, however, have entailed either ad libitum or restricted feeding, without keeping equal the calories ingested of MCT and LCT diets. In the present study, calorie intakes were precisely controlled by direct intragastric feeding of isocaloric amounts of both diets. Also, unlike previous studies, we overfed MCT and LCT diets to determine whether MCT feeding would impede the expected development of obesity.

[1] From the Department of Medicine, St Luke's-Roosevelt Hospital Center and Institute of Human Nutrition, Columbia University College of Physicians and Surgeons, New York, NY.

[2] Supported in part by USPHS Grants AM-26796, AM-26687, and AM-17624 from the National Institutes of Health and by a grant from Stokely-VanCamp, Inc, Indianapolis, IN.

[3] Presented in part at the 20th annual meeting of the American Society for Clinical Nutrition, Washington, DC, April, 1980.

[4] Address reprint requests to: Dr Allan Geliebter, Obesity Research Center, St Luke's-Roosevelt Hospital Center, Amsterdam Avenue and 114th Street, New York, NY 10025.

[5] Also at Department of Psychology, Touro College, 30 West 44th Street, New York, NY 10036.

Received August 4, 1982.

Accepted for publication August 10, 1982.

Methods

Male Sprague-Dawley rats were anesthetized with chloral hydrate (ip), and one end of a silastic tube was implanted in the stomach. The free end of the tube was passed under the skin and allowed to emerge from the posterior aspect of the neck where the rat could not disturb it. Two weeks after surgery, seven rats, 305 g ± 9.4 ($\bar{x}$ wt ± SEM), began receiving the MCT diet and nine rats, 304 g ± 5.0, began receiving the LCT diet (see **Table 1**).

In both diets 50% of the calories were derived from fat, taking into account the metabolizable energy value of MCT, 8.2 kcal/g, and that of LCT, 9.1 kcal/g. The MCT diet contained 5% corn oil to provide essential fatty acids. To the ingredients listed in Table 1, water was added to the extent of 15% by weight, and each diet was thoroughly mixed in a Waring blendor. Both diets had the same caloric density of 4 kcal/ml.

The rats, which were separately housed, were tube-fed their respective diets in two equally divided portions at 10 AM and 5 PM, for 6 wk. To allow them to adapt to intragastric feeding, the volume injected into the stomach was increased progressively from 8.5 ml twice daily to 15 ml twice daily over the first 3 wk. The 15-ml twice daily regimen was then maintained for the remaining three weeks and provided the rats with 120 kcal/day, or 50% more calories than were consumed ad libitum by rats from the same stock when offered a Purina Chow diet. Intragastric feeding provided the sole source of calories, and water was freely available. The rats were weighed twice weekly. During the last week, three rats from each group were placed on an electronic activity monitor (Lafayette) for 24-h periods to determine the level of spontaneous physical activity.

After 6 wk, the rats were killed with an overdose of pentobarbitol (ip), and blood was drawn by heart puncture. Plasma insulin concentrations were determined by radioimmunoassay (10). Fat was dissected out of the epididymal, perirenal, omental, and dorsal depots and weighed. A sample of fat from the right perirenal depot was taken to measure mean fat cell size and number, using a photomicrographic procedure (11). The carcasses without the dissectible fat were analyzed for fat [Folch method (12)], protein [Kjeldahl method (13)] and water (by drying at 60°C to a constant weight). The data were analyzed statistically by the *t* test for independent groups.

TABLE 1
Composition of the diets

Ingredients	MCT diet		LCT diet	
	% Wt	% Cal	% Wt	% Cal
MCT*	25	45		
LCT (corn oil)†	2.5	5	26	50
Casein	15.2	13.5	15.7	13.4
Sucrose	42	36.6	43	36.6
Cellulose	11.7		12	
Salt mix‡	2.9		3	
Vitamin mix§	0.3		0.3	

* Supplied generously as Captex 300 by Capital City Products, Columbus, OH. The heat of combustion is 8.3 kcal/g and the absorption in the rat is 98.5% (8). Hence, the metabolizable energy value is 8.2 kcal/g.

† Mazola corn oil which has a heat of combustion of 9.3 kcal/g and is absorbed in the rat to the extent of 98% (9). The metabolizable energy value is therefore 9.1 kcal/g.

‡ 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.

§ 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; 2 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 pantochenate, 1.97; biotin, 13.1 mg; folic acid, 59.0 mg; vitamin B_{12}, 0.9 mg.

Results

Body weight changes over the course of the experiment are shown in **Figure 1.** After 4 wk, the rats fed the MCT diet weighed significantly less than the rats fed the LCT diet, and by the end of the experiment the rats fed the MCT diet showed a 20% reduction in weight gain as compared to those fed the LCT diet ($p < 0.001$). **Figure 2** shows that all four fat depots were significantly smaller in rats fed the MCT diet as compared to rats fed the LCT diet. The combined weight of all four depots was 23% less in the rats fed the MCT diet than in those fed the LCT diet ($p < 0.001$).

Table 2 provides a summary of the remain-

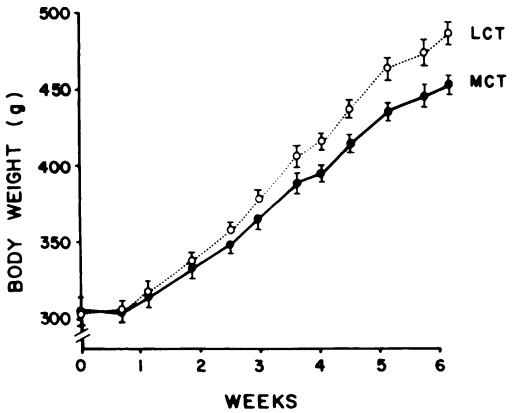

FIG. 1. Mean body weight changes (±SE) of rats overfed with isocaloric MCT and LCT diets. From the 4th wk on, MCT fed rats were significantly lighter in weight ($p < 0.05$) than LCT fed rats.

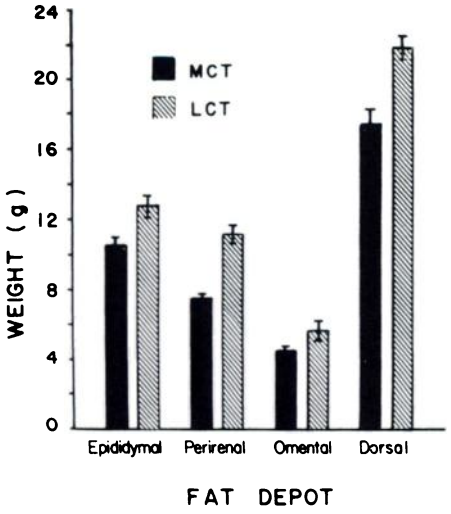

FIG. 2. Mean weight (±SE) of four main fat depots dissected from rats overfed with isocaloric MCT and LCT diets. All depots from MCT fed rats were significantly lighter ($p < 0.05$) than those from LCT fed rats.

TABLE 2
Comparison of data ($\bar{x} \pm$ SEM) for rats fed the MCT diet and rats fed the LCT diet

	MCT	LCT
Mean fat cell size (μg/cell)	0.56* ± 0.04	0.73* ± 0.02
Fat cell number ($\times 10^6$)	5.2 ± 0.40	6.0 ± 0.27
Carcass fat (g)†	116.5 ± 8.4	111.8 ± 5.3
Carcass protein (g)	73.5 ± 2.2	73.0 ± 3.1
Carcass water (g)	192.6 ± 6.9	208.6 ± 6.5
Physical activity movements $\times 10^3$/24 h	47.4 ± 4.4	42.9 ± 7.1
Serum insulin concentrations (μU/ml)	85.0 ± 16.1	117.9 ± 16.7

* Difference is significant, $t = 3.7$, $p < 0.005$. All other differences are not significant.
† Exclusive of the dissectible fat.

ing data. It may be seen that mean fat cell size was significantly reduced in the MCT-fed rats, but that fat cell number was not altered. Carcass analysis after removal of the main fat depots indicated no significant differences in fat, protein, or water. Neither physical activity nor insulin levels differed between the MCT- and LCT-fed rats.

Discussion

The data indicate that overfeeding with the MCT diet resulted in a significant reduction in weight gain that was associated with significantly smaller body fat stores, owing to a relative decrease in adipocyte size. Because levels of physical activity and insulin did not change with MCT feeding, neither of these factors can account for the reduced adiposity. We have previously shown that reductions in physical activity or increases in circulating insulin levels promote increased fat deposition (14).

Given the well-documented efficiency of absorption of MCT which is always greater than LCT (8) differences in fecal energy output also cannot account for our results. Although urinary ketones are higher in animals fed MCT than in those fed LCT, direct measurements of urinary energy losses in rats fed graded amounts of MCT up to 21% of the diet showed this contribution of ketones to urinary energy to be negligible (15).

Thus the decreased fat deposition in the MCT-fed rats appears to be related to the unique absorption and metabolic pathways followed by MCT. Unlike LCT, whose digestive products after absorption and intestinal mucosal resynthesis as triglyceride moieties of chylomicrons are largely transported via the lymph and thus bypass the liver, MCT digestive products after absorption are transported via the portal vein blood as free fatty acid directly to the liver (16). The vast majority of these MCT-derived moieties are then promptly oxidized by the liver, with less than 2% eventually incorporated into depot fat (17). The oxidation of fatty acids from MCT may be less biologically efficient for energy (adenosine triphosphate) conservation than the oxidation of fatty acids from LCT. Therefore, part of the potentially useful energy in MCT is probably dissipated as heat. Indeed, evidence has recently been provided for increased heat production after ingestion of MCT (15). If more energy from MCT than from LCT is converted into heat, less energy would be available for storage as fat.

This reduced efficiency of MCT for conversion into body fat and weight gain may have an application to human obesity. At our hospital, MCT has been fed as an exclusive source of fat (with a small amount of LCT to provide essential fatty acids) to a patient with chyluria for more than 15 yr without producing side effects, and the patient has remained extremely lean (18).

The present findings suggest that an MCT diet deserves investigation as a potential adjunct in the dietary prevention of human obesity.

The authors thank Janet Seylar for skillful technical assistance and Dr. VK Babayan for advice and encouragement.

References

1. Babayan VK. Medium-chain triglycerides—their composition, preparation, and application. J Am Oil Chem Soc 1968;45:23–5.
2. Senior JR, ed. Medium chain triglycerides. Philadelphia, PA: University of Pennsylvania Press, 1968:63–247.
3. Schizas AA, Cremen JA, Larson E, O'Brien R. Medium-chain triglycerides—use in food preparation. J Am Diet Assoc 1967;51:228–32.
4. Kaunitz H, Slanetz CA, Johnson RE, Babayan VK, Barsky G. Relation of saturated, medium- and long-chain triglycerides to growth, appetite, thirst and weight maintenance requirements. J Nutr 1958;64:513–24.
5. Harkins RW, Sarett HP. Nutritional evaluation of medium-chain triglycerides in the rat. J Am Oil Chem Soc 1968;45:26–30.
6. Wiley JH, Leveille GA. Metabolic consequences of dietary medium chain triglycerides in the rat. J Nutr 1973;103:829–35.
7. Lavau MM, Hashim SA. Effect of medium chain triglyceride on lipogenesis and body fat in the rat. J Nutr 1978;108:613–20.
8. Kaunitz H, Slanetz CA, Johnson RE, Babayan VK, Barsky G. Nutritional properties of the triglycerides of saturated fatty acids of medium chain-length. J Am Oil Chem Soc 1958;35:10–3.
9. Hoagland R, Snider GG. Digestibility of some animal and vegetable fats. J Nutr 1943;25:295–302.
10. Herbert V, Lau KS, Gottlieb CW, Bleicher SJ. Coated charcoal immunoassay of insulin. J Clin Endocrinol 1965;25:1375–84.
11. Lavau M, Susini C, Knittle J, Blanchet-Hirst S, Greenwood MRC. A reliable photomicrographic method for determining fat cell size and number: application to dietary obesity. Proc Soc Exp Biol Med 1977;156:251–6.
12. Folch J, Lees M, Sloan-Stanley GH. A simple method for the isolation and purification of total lipids from animal tissues. J Biol Chem 1957; 226:497–509.
13. Ferrari A. Nitrogen determination by a continuous digestion and analysis system. Ann NY Acad Sci 1960;87:792–800.
14. Bracco EF, Torbay N, Geliebter A, Stewart I, Seylar J, Hashim SA. Insulin increases body fat with control of food intake and physical activity. Alim Nutr Metab 1980;1:234(abstr).
15. Travis D, Minenna A, Frier H. Effects of medium chain triglyceride on energy metabolism and body composition in the rat. Fed Proc 1979;38:561(abstr).
16. Bloom B, Chaikoff IL, Reinhardt WO. Intestinal lymph as pathway for transport of absorbed fatty acids of different chain lengths. Am J Physiol 1951;166:451–5.
17. Zurier RB, Campbell RG, Hashim SA, Van Itallie TB. Enrichment of depot fat with odd and even numbered medium chain fatty acids. Am J Physiol 1967;212:291–4.
18. Hashim SA. Medium chain triglycerides-clinical and metabolic aspects. J Am Diet Assoc 1967;51:221–7.