



## METHODS

100 colony-bred male albino rats aged 30 days were studied for a period of 90 days, as this period is long enough to induce significant alterations in the fatty acid composition of adipose tissue (2). The rats were randomly divided into four groups of 25 rats each. Each group received either lard (L), safflower oil (SO), groundnut oil (GO), or coconut oil (CO) as the only source of fat in their diet.

Rats were housed individually in identical plastic cages at an ambient temperature of 28–30 degrees Celsius. The initial body weight of all the rats was recorded. The rats were given free access to food and water and spillage was accounted for. Food intake was measured every third day and body weight was taken once a week on Mondays, at 09:30 Hrs.

To compare the effect of carbon chain length, coconut oil (3) was chosen for its high concentration of medium-chain triglycerides (MCT:C6–C12), while groundnut oil (4) was chosen for its high concentration of long-chain triglycerides (LCT), but it also showed a high monounsaturated fatty acid content. To compare the degree of saturation, lard (5) was chosen for its high concentration of saturated fats, while safflower oil (5) was chosen for its high concentration of polyunsaturated fatty acid.

The four different diets were prepared by adding the selected fats to commercial food pellets (Hindustan-Lever), which were powdered and mixed thoroughly. The added fat was 16% by weight and 33% by energy content, a level very close to that

recommended by the American Heart Association (6). The Hindustan-Lever pellets contained; minimum 21.0% crude protein, 5.0% ether extract, 4.0% crude fibre, 8.0% ash, 1.0% calcium, 0.6% phosphorous, 55.0% nitrogen free extract and 3600 kcal/kg metabolisable energy. In addition to all this, the pellets contain stabilised Vitamins A, D3, E, K, B12, Thiamine, Riboflavin, Pantothenic acid, Niacin, Choline Chloride, Folic acid, all minerals and trace elements. Vitamin C had also been added in adequate quantities.

TABLE I: Fatty acid composition (%) in test diets.

<i>No of C atoms: No of double bonds</i>	<i>Lard</i>	<i>Safflower oil</i>	<i>Coconut oil</i>	<i>Groundnut oil</i>
8:0	–	–	03.2	–
10:0	–	–	04.8	–
12:0	–	–	55.4	–
14:0	02.0	–	19.9	–
16:0	24.7	07.3	08.0	12.4
16:1	03.2	–	–	–
18:0	15.1	02.5	01.8	03.4
18:1	43.9	13.6	05.5	59.0
18:2	06.9	75.7	01.4	21.1
18:3	00.3	00.5	–	01.6
20:0	–	–	–	00.9
20:4	–	–	–	01.6
Others	03.9	00.4	–	–

Based on the food intake and the weight gain, the Calculated food efficiency ratio (CFER) was calculated as the ratio of amount of food consumed to the body weight gained (2). The results are presented as Mean, standard deviation (SD) and standard error of mean. The unpaired Student 't' test was used to look for significant differences in food intake, weight gain and CFER, between the two groups CO and GO (effect of carbon chain length), and between the

two groups L and SO (effect of degree of saturation) separately. Interactions between carbon chain length and degree of saturation were not examined.

## RESULTS

### Food intake

The food intake varied among the four groups of rats depending on the type of fat added. In the groups comparing carbon chain length, there was a significantly higher food intake in GO rats as compared to CO ( $P < 0.001$ ). In the groups comparing degree of saturation, there was a significantly higher food intake in the SO group as compared to L ( $P < 0.001$ ) (Table II, Fig. 1).

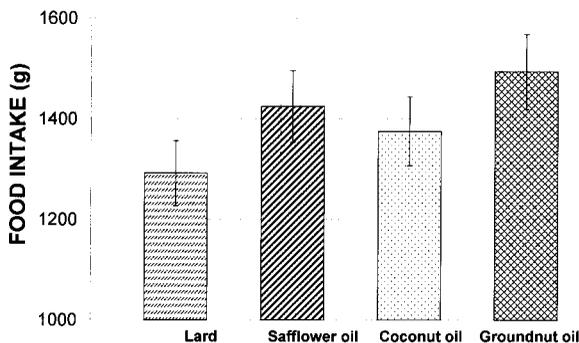


Fig. 1: Effect of type of dietary fat on food intake over a 90-day period in albino rats.

### Weight gain

The average initial weight of the rats in each group was; L =  $44.6 \pm 11.49$  g, SO =  $45.56 \pm 10.55$  g, CO =  $45.36 \pm 9.48$  g, GO =  $48.72 \pm 7.61$  g, and these were not significantly different. At the end of the experiment the, mean weight gain in the four groups ranged from about 228 to 240 g (Table II). However there was no significant difference in the weight gain between the groups compared.

### Calculated food efficiency ratio (CFER)

CFER was similar in rats belonging to groups GO and CO. However CFER was significantly greater in group SO

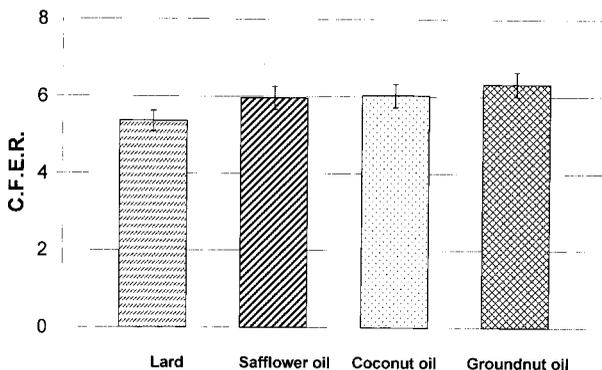


Fig. 2: Effect of type of dietary fat on calculated food efficiency ratio (CFER).

TABLE II: Effect of type of dietary fat on.

	<i>Groups comparing carbon chain length</i>		<i>Groups comparing degree of saturation</i>	
	<i>CO</i>	<i>GO</i>	<i>L</i>	<i>SO</i>
Food intake	1375.84±140.26	1494.20±142.79*	1292.00±122.79	1425.32±153.94*
Body weight gain (g)	228.24±21.5	236.4±30.30	240.88±41.27	239.28±22.3
CFER	6.03±0.62	6.32±0.85	5.36±1.34	5.96±0.77**

\* $P < 0.001$ ; \*\* $P < 0.02$ .

in comparison to that in group L ( $P < 0.02$ , Table II).

Thus in the groups comparing the carbon chain length, the group CO rats ate significantly less ( $P < 0.001$ ), while their weight gain and CFER was similar to that in group GO. On the other hand, in the groups comparing degree of saturation the group L rats gained similar weight as SO rats. This was despite a significantly lower ( $P < 0.01$ ) food intake in the group L rats as compared to SO.

## DISCUSSION

### MCT (coconut oil) v/s LCT (groundnut oil)

The food intake in group GO was significantly greater than in group CO probably because they found GO more palatable. But despite a significant difference in food intake the CFER between the two groups was similar. This can be attributed to the lower net energy value of GO when consumed in moderate to high amounts (approximately 5 Kcal/g), which is due to its lower heat of combustion and its greater thermogenic effect (7). The increased energy expenditure after MCT ingestion is due to their unique absorption, metabolic fate and greater thermogenic effect (7, 8). MCT are transported via hepatic portal circulation as free fatty acids (bound to albumin) directly to liver, where they are preferentially oxidised to acetyl-CoA. This in turn, is either oxidised to carbon dioxide, converted to ketone bodies or used as a substrate for de novo synthesis of long chain fatty acids, i.e. through metabolic pathways that are energetically

costlier and hence result in a substantial part of potential useful energy in MCT being dissipated as heat (7). The greater thermogenic effect of MCT (9–12), especially post-prandial thermogenesis and a lower respiratory quotient, indicates increased fat oxidation leading to less fatty acid deposition. Catecholamines are seen to play a role (13). MCT alters lipolytic sensitivity to catecholamines. The signal that relates MCT to increased sympathetic nervous system (SNS) activity remains to be determined, but potential candidates are insulin and 3-hydroxy butyrate, both of which are shown to increase rapidly after ingestion of MCT in humans and are known to enhance SNS activity (7). In contrast, LCT are absorbed across intestinal lymphatic ducts and transported in chylomicrons through the lymph to reach the systemic circulation (thus bypassing the liver) and are subsequently taken up by peripheral tissues, which does not require much energy (7). This process involves an increase in plasma and hepatic lipids, and an increase in fat deposition in adipose tissue, as well as a marked decrease in activity of lipogenic enzymes in the liver and adipose tissue (14). Besides having a high LCT content, groundnut oil also has a high concentration of monounsaturated fatty acids whose rapid absorption, possible portal-venous transport and subsequent hepatic oxidation (15) might have impeded a greater weight gain for group GO.

### SFA (lard) v/s PUFA (safflower oil)

The decreased food intake in rats fed lard could be due to a greater delay in gastric emptying and earlier stimulation of

the satiety center by gastrointestinal hormones induced by the saturated fats contained in lard. Despite the decreased food intake, the weight gain was comparable to that of group SO, as indicated by the significantly lower CFER. This implies that the amount of food consumed per gram of body weight gained was significantly lower in group L as compared to SO. The dietary PUFA make an earlier and larger contribution to the thermogenic effect of food (16), as their intestinal absorption is enhanced, followed by preferential portal transport and hepatic oxidation saturated fats are committed to chylomicron incorporation and lymphatic transport, thus requiring a greater interval of time before reaching their site of oxidation. Lard (rich in SFA) decreases diet induced thermogenesis (DIT), by a decline in sympathetic activity in brown adipose tissue (BAT) resulting in decreased lipolytic activity thereby promoting body fat accumulation (5). The reduced lipolytic activity may result from lower beta-receptor binding, decreased norepinephrine turnover rates and decreased sympathetic activity in adipose tissue (17). Dietary fatty acid composition affects membrane fatty acid composition in adipose tissue, which alters membrane fluidity. The presence of PUFA in the lipid bilayer contributes to its fluidity, whereas saturated fatty acids are rigidifying molecules because of absence of double bonds (18). The change in membrane fluidity affects the capacity of adrenergic receptor binding and enzyme activity in plasma membranes. Plasma membrane fluidity affects the activity of tyrosine hydroxylase, a key enzyme of norepinephrine synthesis (19).

In contrast, safflower oil (rich in PUFA) increases thermogenic activity in BAT by the stronger SNS stimulation, and a direct influence on BAT (20). Linoleic acid is preferentially channelled towards BAT, leading to an increased uncoupling of respiration through the acute effects of fatty acids or activation of proton conductance pathway (21). The PUFA in safflower oil also modifies membrane functions such as receptor-enzyme coupling and hormone sensitivity (22).

Although dietary fatty acids may increase body fat accumulation by altering DIT, the possibility that some unknown factor other than fatty acids present in animal fats and not present in vegetable oils is responsible for decreasing DIT and increasing body fat accumulation cannot be excluded.

The results obtained in this study agree with those of some workers who have reported that rats fed MCT ate significantly less than those eating LCT (23, 24). However there are other reports (2, 11, 25–27), which indicate that food intake is independent of dietary fatty acid composition. This discrepancy could be explained by differences in feeding protocols. Coconut oil used in this study contained 55% MCT while most of the others used 100% MCT which was synthetically prepared from coconut oil. Also there were differences in the percentage of fat used.

Various researchers have found comparable weight gain in rats fed ad libitum diets differing in fatty acid composition (2, 17, 25, 27). Other workers

found that MCT fed rats gained less weight as compared to LCT fed rats (24), while still others who examined weight gain in rats fed saturated, MCT or LCT, found that MCT fed rats gained less weight as compared to either saturated or LCT fed rats (28). Some researchers have shown that CFER was not affected by dietary manipulation (2). Others have found a lower CFER in rats during transition from low fat diet to MCT as compared to rats fed LCT (11). These MCT rats ate less than the low fat diet rats and also gained less weight. But the decrease in body weight gain was higher resulting in a lower CFER. But the significantly lower

CFER obtained in the present study among the rats fed saturated fats is most likely due to the fact that we used lard, which contains much more saturated fatty acids as compared to beef tallow used by other researchers (2, 3, 18).

In conclusion, when rats are fed an ad libitum diet varying in dietary fats used, the body seems to adjust the food intake to produce a comparable weight gain. It thus appears that the carbon chain length and degree of saturation of fats are not important determinants of weight gain in albino rats fed an ad libitum diet.

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