

# The fatty acids of human depot fat

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**SUMMARY** The depot fat fatty acid composition has been analyzed in apparently healthy White, Cape Coloured, and Bantu males and females in the third and fifth decades of age and in White males in the fifth decade with proved ischemic heart disease. Comparisons have been made between the ages, between the sexes, and between the races.

There was no effect of age on depot fat composition, despite different trends with age between the groups in serum cholesterol concentration and the trend for the degree of body fatness to increase with age in all groups. Females showed higher ratios of monounsaturated to saturated fatty acids than did males.

Interracial differences were confined to myristate, which showed a progressive fall from White through Cape Coloured to Bantu that was strikingly correlated with the total fat intake in these groups, and linoleate, which was significantly higher in the Cape Coloured than in either of the other groups. We could find no dietary factor to account for the latter finding.

**M**AN, LIKE many other mammals and certain amphibians, has some ability to alter the fatty acid composition of his depot fat. He is also able to synthesize fat from non-fat sources. The fatty acids he is able to synthesize and the changes he is able to produce in the deposited fat would appear to be limited to certain fatty acids (1, 2). The degree of alteration and type of change brought about by these processes are probably similar in all normal people. The fatty acid composition of the dietary fat would therefore appear to be the factor of greatest potential importance in determining differences in the fatty acid composition of depot fat among different people. That changes in the fatty acid composition of human depot fat can be brought about by dietary manipulations, albeit slowly, has been demonstrated (3).

Cape Town with its three major racial groups—White, Cape Coloured, and Bantu—subsisting on diets distinctly different, especially with regard to their relative fat intakes, provides material of particular

interest. The dietary habits of these three groups represent between them qualitative extremes of human nutrition. The subjects available here can therefore provide data on the depot fat fatty acids, both qualitatively and in their relative proportion, that would be representative of a large part of mankind. They could in addition provide a means of examining to what extent and in what manner diets that are naturally different affect the composition of depot fat.

Other than the effect of diet on fat metabolism the variables of age and sex have also been shown to influence lipid values. This has been established with respect to both serum lipids (4–7) and total body fat (8–12). Reported studies to date on the fatty acid composition of human depot fat (2, 3, 13–16) have paid scant attention to these factors.

In this communication data are presented on the fatty acid composition of human depot fat based on the analyses of samples from apparently healthy subjects drawn from White, Cape Coloured, and Bantu males and females in the third and fifth decades of age and in 12 White males in the fifth decade with clinical and electrocardiographic (ECG) evidence of ischemic heart disease (IHD). The dietary habits, serum cholesterol concentration, and degree of body fatness in the subjects sampled were determined and found to reflect those trends according to race, sex, and age where these have been previously established in these groups (17, 18).

## MATERIAL AND METHODS

### *Selection of Subjects*

The subjects were drawn from patients admitted to Grootte Schuur Hospital for the treatment of minor surgical conditions like hemorrhoids, hernia, varicose veins, etc., and in whom there was no evidence of a metabolic or endocrine disorder nor any history of a significant change in dietary habits or body weight in the preceding 6 months. Particular care was taken to exclude from

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TABLE 1 AGE OF SUBJECTS SELECTED FROM EACH SEX AND RACE

Group	Sex	n	Age		
			Mean	SD	Range
White	M	12	25.2	2.3	22-49
	M	10	44.4	3.3	40-49
	F	10	24.8	2.6	23-29
	F	14	45.4	2.6	40-49
Cape Coloured	M	13	24.2	2.0	20-28
	M	11	45.0	2.5	41-48
	F	10	24.4	3.0	20-29
	F	12	43.7	2.9	40-49
Bantu	M	11	28.1	3.7	21-31
	M	10	44.6	4.4	40-50
	F	14	25.6	3.4	21-30
	F	10	45.3	4.5	40-50
IHD*	M	12	45.3	1.7	43-49

\* White subjects with ischemic heart disease.

the apparently healthy group subjects with any manifestations of atherosclerosis or IHD elicited by means of interrogation and electrocardiographic studies. The females sampled were all premenopausal. With few exceptions, samples were obtained from each race, sex, and age group at or about the same time and analyzed under the same conditions. In the IHD group ten subjects had unequivocal ECG evidence of myocardial infarction and two had ischemic changes only. The number of subjects drawn from each race and sex and at each age level is shown in Table 1.

#### Dietary Analysis

A dietary history was obtained by recall from each subject sampled. Average quantities of each specific item of food eaten on each day of a typical week were taken as representative of the subject's diet. Due regard was paid to those dietary items not part of the regular eating pattern. The method of preparation of each dish was established and estimates made of the amount and type of fat added to any foodstuff. The intake of each foodstuff was calculated on the basis of average portions, unless there were obvious deviations. The composition of the diet as regards protein, fat, carbohydrate, and calories was calculated from food tables (19, 20). The fatty acid composition of the dietary fat consumed by each subject was also determined from standard food tables (21). It is recognized that estimation of dietary intake obtained

by recall gives, at best, an approximation of the actual intake. The fatty acid composition of the dietary fat was determined from food tables compiled in the United States and may not accurately reflect that of local fats.

#### Relative Obesity

The relative degree of body fatness in the subjects sampled was determined by skinfold measurements. These measurements were made by means of a skinfold caliper<sup>1</sup> of a type giving a constant tension regardless of the extent to which the jaws of the caliper are opened. The caliper was standardized to give a tension of 10 g/mm<sup>2</sup> of jaw surface. Measurements were made at the posterior aspect of the right arm, midway between acromion and olecranon, and on the back, immediately below the inferior angle of the right scapula with the arm adducted. The measurements were made by lightly picking up a fold of skin and subcutaneous tissue 1/2 to 1 inch distal to the point of application of the caliper jaws and determining the thickness of the fold to the nearest millimeter. These sites were selected because of the good correlation shown between the thickness of these skinfolds and estimations of total body fat made by specific gravity studies in man (22).

#### Serum Cholesterol Concentration

This was determined on samples of venous blood obtained from fasting subjects within 24 hr of their admission to hospital. It is unlikely that the serum cholesterol values had been materially affected during this time by one or two meals of the hospital diet. Total serum cholesterol was determined by the method of Abell et al. (23) as modified by Anderson and Keys (24).

#### Depot Fat Analysis

Depot fat was sampled from the buttocks by the aspiration technique of Hirsch et al. (3) within 24 hr of admission to hospital. The depot fat aspirate was immediately taken up into 20 volumes of chloroform-methanol 2:1 (v/v) to give a one-phase system and allowed to stand at room temperature in stoppered tubes for some hours. The extracted lipid was then isolated from non-lipid components by the technique of Folch et al. (25) Methyl esters were prepared by methanolysis with 5%

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TABLE 2 MEAN SERUM CHOLESTEROL CONCENTRATION (MG/100 ML) AT EACH AGE LEVEL IN BOTH SEXES IN THE THREE RACES

Race	Males		Females	
	3rd Decade	5th Decade	3rd Decade	5th Decade
White	240.0 ± 44.6	283.1 ± 44.6	211.7 ± 27.9	275.0 ± 65.5
Cape Coloured	211.1 ± 35.3	253.9 ± 36.5	209.3 ± 55.8	241.8 ± 41.7
Bantu	209.9 ± 32.6	196.6 ± 51.6	206.0 ± 24.2	192.2 ± 39.5

H<sub>2</sub>SO<sub>4</sub> in methanol (26) prior to fatty acid analysis by gas-liquid chromatography (GLC) on a Pye Argon Chromatograph. The stationary phase was 20% (w/w) ethylene glycol adipate polymer, prepared according to the method of James (27), on Chromosorb W (80-100 mesh). Analyses were made at temperatures of 185 or 197° and gas flow rates of 40 to 60 ml/min. This technique does not separate the isomers of oleic acid that have been shown on Apiezon columns to be present in depot fat (3, 16). The fatty acids were identified by chromatography of appropriate standards and by comparison with published data (28) on relative retention times on the same stationary phase at the same temperature of operation. The identification of unsaturated components was confirmed by bromination and by hydrogenation of the sample (28). The linearity of response of the instrument was established by comparing the proportions of fatty acids in a mixture of known composition by weight with those found on GLC analysis and more recently confirmed by analysis of a suitable N.I.H. standard.

It was confirmed that the aspiration technique provided a highly representative sample of adipose tissue from the local site. It was further found that there was no change in fatty acid composition related to the depth from the skin surface at the site at which fat was sampled.

The fatty acid composition of fat from the buttock region was also compared with that at two or three other sites in several subjects. Samples obtained from different sites in the same subjects were not as homogeneous in composition as those obtained at varying depth from the skin surface at the same site; the former showed a degree of variation similar to that previously reported (3).

## RESULTS

### SERUM CHOLESTEROL CONCENTRATION

The established trend for serum cholesterol concentration to fall from White, through Cape Coloured to Bantu is most strikingly evident in the older age group of subjects both in the males and in the females. This effect is largely the result of the trend for serum cholesterol concentration to increase with age in the White and Cape Coloured subjects but not in the Bantu (Table 2).

### RELATIVE OBESITY

The trend for the degree of body fatness to increase with age is evident in both males and females, being much more marked in the females (Fig. 1). These same trends were evident in each racial group.

### THE FATTY ACID COMPOSITION OF DEPOT FAT

The fatty acid compositions of the depot fat in all the apparently healthy subjects selected for this study are

TABLE 3 THE FATTY ACID COMPOSITION OF SUBCUTANEOUS ADIPOSE TISSUE IN ALL THE APPARENTLY HEALTHY SUBJECTS

Fatty Acid*	Mean	sd	Range
12:0	0.4	0.2	0.1-1.3
14:0	3.4	1.2	0.8-8.6
14:1	1.0	0.1	0.2-3.2
15:0	0.6	0.3	0.2-1.8
15? :	0.4	0.1	0.1-1.2
16:0	22.7	2.6	13.0-30.1
16:1	8.4	2.2	2.4-15.1
17:0	0.6	0.3	0.2-1.9
17? :	0.8	0.3	0.4-1.9
18:0	4.3	1.6	1.6-9.8
18:1	45.4	4.1	33.6-56.3
18:2	9.7	3.6	3.8-20.7
18:3	0.7	0.4	0.2-1.8
20:0	0.9	0.5	Trace-2.6
20:1	0.9	0.5	Trace-2.2
20:4			0-0.5

No. of subjects, 137 (all three races).

\* Chain length: no. of double bonds.

given in Table 3. This shows the fatty acids found as well as the percentage of each fatty acid expressed as a mean, with one standard deviation and the range. In the case of 20:4 only a range is given because of the technical difficulties of establishing its presence or absence with certainty.

A feature that is most striking from Table 3 is the consistency with which each of the first 15 components was present in every sample, although some were present in only trace amounts. As samples were obtained in approximately equal numbers from groups whose dietary habits represent extremes of human nutrition and the variables of age and sex also were taken into account, Table 3 can be regarded as reflecting depot fat composition that is representative of a large part of mankind.

The six fatty acids 14:0, 16:0, 16:1, 18:0, 18:1, and 18:2 were found to constitute 93.8% ( $\pm 1.6\%$ ) of the total fatty acids in every subject and group. The coefficient of variation of the method in determining the percentage of each of these six components was tested on different GLC columns, operated at different temperatures, and by doing replicate analyses on samples of tissue obtained from the same site, and was found to vary from 2.1% to 5.0%. The minor components could not be determined with that degree of reproducibility.

Despite the over-all homogeneity in fatty acid composition, there were some differences between the racial groups as well as within each racial group. Only the major components will be considered.

### RELATION TO AGE

Comparison of the proportions of each of the major fatty acids did not show any significant difference

between the age groups in general; within either sex; or within any of the racial groups. Variance analysis gave essentially the same results. Age, as such, was therefore not related to any differences in fatty acid composition at the age levels studied, in spite of different trends with age in serum cholesterol concentration in the different racial groups (Table 1) and the trend for the degree of body fatness to increase with age in all groups (Fig. 1).

#### RELATION TO SEX

On comparing the fatty acid composition between the sexes regardless of age and race it can be seen that statistically significant differences exist (Table 4). These differences are confined to the saturated and monoun-

TABLE 4 PERCENTAGES OF EACH OF THE COMMON FATTY ACIDS COMPARED BETWEEN THE SEXES

Fatty Acid	Males (n = 67)	Females (n = 70)	Difference	P
14:0	3.5 ± 1.3	3.2 ± 1.1	0.3	N.S.
16:0	23.4 ± 2.6	22.1 ± 1.3	1.3	<0.001
16:1	7.4 ± 1.4	9.3 ± 2.3	1.9	<0.001
18:0	5.1 ± 1.7	3.5 ± 1.1	1.6	<0.001
18:1	44.5 ± 4.0	46.1 ± 4.1	1.6	<0.05
18:2	9.8 ± 3.5	9.6 ± 3.7	0.2	N.S.

The values (mean ± SD) are based on the pooled data in the three races.

saturated C<sub>16</sub> and C<sub>18</sub> fatty acids; the saturated fatty acids are higher in the males and the monounsaturated fatty acids are higher in the females. From Table 4 it would further appear that there may be reciprocity between 16:0 and 16:1 and between 18:0 and 18:1. This is supported by the finding that the percentages of 16:0 + 16:1 and 18:0 + 18:1 are the same in both sexes. Variance analysis confirmed that these sex differences were significant but in addition indicated that the sex differences were not the same for each race. On subdivision according to race the differences in composition between the sexes show the same trend, with few exceptions, in each racial group and this too is confirmed by variance analysis. Myristate and linoleate do not show any sex difference, either in general (Table 4) or in any racial group (Table 5).

It is of interest that the sex differences should be present in these particular fatty acids in view of the inter-conversions that may take place between them in the body as a whole (1) and in the adipose tissue itself (2).

TABLE 5 PERCENTAGES OF EACH OF THE MAJOR FATTY ACIDS COMPARED BETWEEN THE SEXES IN EACH RACIAL GROUP

Fatty Acid	Race	Males	Females	Difference	P
14:0	W	4.1 ± 1.5	3.9 ± 1.1	0.2	N.S.
	C	3.7 ± 1.3	3.2 ± 1.0	0.5	N.S.
	B	2.6 ± 0.4	2.5 ± 0.7	0.1	N.S.
16:0	W	24.1 ± 1.9	21.8 ± 2.3	2.3	<0.001
	C	23.6 ± 2.8	21.9 ± 1.7	1.7	<0.05
	B	22.3 ± 2.9	22.5 ± 2.5	0.2	N.S.
16:1	W	8.2 ± 1.2	9.6 ± 1.9	1.4	<0.01
	C	6.6 ± 1.6	7.3 ± 1.4	0.7	N.S.
	B	7.5 ± 1.9	10.8 ± 2.2	3.3	<0.001
18:0	W	4.5 ± 1.2	3.3 ± 0.9	1.2	<0.001
	C	5.2 ± 1.8	3.9 ± 0.9	1.3	<0.01
	B	5.5 ± 1.8	3.2 ± 1.2	2.3	<0.001
18:1	W	45.0 ± 2.9	46.4 ± 4.3	1.4	N.S.
	C	42.0 ± 4.0	44.3 ± 3.8	2.3	N.S.
	B	46.9 ± 3.1	47.4 ± 3.6	0.5	N.S.
18:2	W	7.5 ± 1.9	7.6 ± 2.0	0.1	N.S.
	C	12.2 ± 4.1	13.3 ± 3.7	1.1	N.S.
	B	9.6 ± 2.0	8.3 ± 2.5	1.3	N.S.

N. S. = not significant (P>0.05).

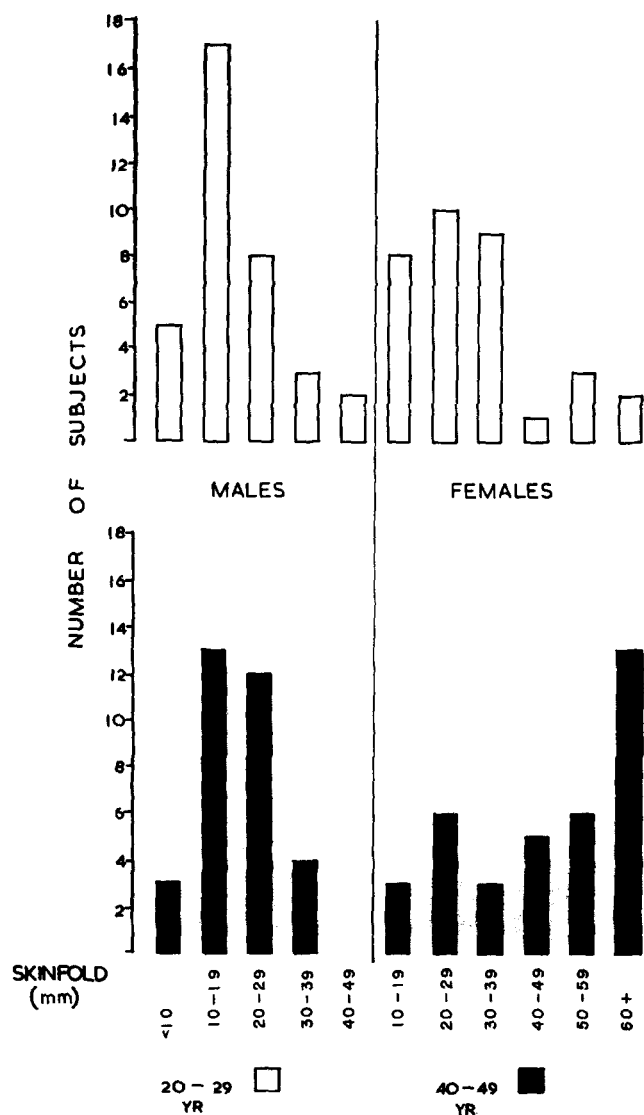


FIG. 1. The relative degree of body fatness, expressed as the combined skinfold thickness, in the males and females at each age level. Note the trend for the degree of body fatness to increase with age, particularly in the females.

TABLE 6 CERTAIN FATTY ACID RATIOS COMPARED BETWEEN THE SEXES IN EACH RACE

Fatty Acid Ratio	Race	Males	Females	Difference	P
16:1 16:0	W	0.34 ± 0.06	0.45 ± 0.11	0.11	<0.001
	C	0.29 ± 0.09	0.33 ± 0.07	0.04	<0.05
	B	0.34 ± 0.08	0.49 ± 0.12	0.15	<0.001
	W + C + B	0.32 ± 0.08	0.43 ± 0.12	0.11	<0.001
18:1 18:0	W	10.80 ± 3.26	15.14 ± 4.06	4.34	<0.001
	C	9.28 ± 4.06	11.97 ± 2.97	2.69	<0.01
	B	9.37 ± 3.06	16.56 ± 6.02	7.19	<0.001
	W + C + B	9.81 ± 3.51	14.63 ± 4.89	4.82	<0.001
16:1 + 18:1 16:0 + 18:0	W	1.88 ± 0.25	2.27 ± 0.37	0.39	<0.001
	C	1.73 ± 0.36	2.02 ± 0.24	0.29	<0.01
	B	1.99 ± 0.32	2.30 ± 0.39	0.31	<0.01
	W + C + B	1.86 ± 0.33	2.21 ± 0.35	0.35	<0.001

In view of these possible interconversions it is suggested that the important difference between the sexes may be not the actual proportions of 16:0, 16:1, 18:0, and 18:1 but the ratio of the monounsaturated to the saturated fatty acids. These ratios (Table 6) are consistently higher in the females in general, at each age level and in each racial group and all are statistically significant, with the exception of the ratio 16:1/16:0 in the older age groups of Cape Coloured and Bantu subjects.

From Table 6 it is evident that there is no specific value for a particular fatty acid ratio that can be regarded as a feature of either sex. Although the sex difference is consistent in each race, some of the values for the males of one race are much the same as those for the females of another. It would therefore appear that some other factor(s) related to race may also influence these values. Variance analysis confirmed that the sex differences were statistically significant for each of these ratios in every race but that while in no instance was the White group different from the Bantu, the Cape Coloured group differed significantly from both White and Bantu in every ratio.

*Dietary Factors*

The role of the diet in determining depot fat composition cannot be ignored, particularly in view of the finding (29) that a fat-free diet abolished the difference between young rats and old rats in the rate of incorporation of acetate into 16:1 and 18:1. From dietary analysis (Table 7) it is obvious that the diet is basically the same in both sexes of each race. Diet would therefore not account for the sex differences in fatty acid composition. Further, despite the marked differences in fat and carbohydrate intake between the White and Bantu groups, in no instance do the Bantu, either males or females, show a difference from the White subjects of the same sex in any of the fatty acid ratios.

Although the protein would appear to be consistently lower in the females than in the males, these differences

are small compared with the interracial differences in fat and carbohydrate intake. There is no evidence that dietary protein favors the deposition in depot fat of any specific fatty acid. Nor does the saturated fatty acid intake correlate with these sex differences. Since the linoleic acid intake is much the same in all the groups and the saturated fatty acid intake the same in both sexes of each race, the total monounsaturated fatty acid intake is probably also similar in them. Dietary factors, in so far as actual intakes can be estimated by the method used, do not account for the sex differences in fatty acid composition reported here. Thus despite differences between the races in these fatty acid ratios, the consistent sex difference in each race suggests a metabolic difference between the sexes.

*Relation to Degree of Obesity*

The trend for females to be more obese than males (Fig. 1) is evident in each racial group and at each age level. The possibility that there may be a relation between the degree of obesity and the above fatty acid ratios merits consideration. The correlation between the combined skinfold thickness and the fatty acid ratio 16:1 + 18:1/16:0 + 18:0, calculated for the females only, was not significant ( $r = +0.148, P > 0.1$ ). (To have included the males in this analysis could have given rise to a spurious association since they are in general

TABLE 7 ESTIMATED DIETARY LEVELS OF VARIOUS CONSTITUENTS IN THE MALES AND FEMALES OF EACH RACIAL GROUP

Dietary Constituent	Males			Females		
	W	C	B	W	C	B
	<i>% of total daily calories</i>					
Protein	18.8	17.8	16.4	14.0	15.8	13.3
Carbohydrate	33.7	42.9	63.1	41.7	42.1	64.6
Fat	47.5	38.5	19.7	44.3	42.0	22.2
Saturated Fatty Acids	23.3	18.3	8.0	21.1	19.8	8.9
Linoleic Acid	3.2	3.1	2.9	3.0	3.4	2.9

leaner and have a lower value for this ratio.) Thus, in females, the degree of obesity bears no relationship to those fatty acid rations.

#### RELATION TO RACE

The only differences of note on comparing the proportion of each fatty acid among the racial groups were in 14:0 and 18:2. Myristate is seen to fall progressively from White through Cape Coloured to Bantu in both the males and females. If the total fat intake is compared with the percentage of 14:0 in both males and females in each race, a striking correlation is evident (Fig. 2).

#### Linoleic Acid

The percentage of 18:2 is consistently higher in the Cape Coloured subjects than in the White and Bantu groups. The level in the Bantu is in general intermediate between the White and Cape Coloured groups. Since this fatty acid apparently cannot be synthesized, its deposition in depot fat would presumably be dependent upon its availability in the diet. Dietary analysis, however, did not show any such relationship (Table 7), so that any relationship between the availability of linoleic acid in the diet and its deposition in depot fat must be complex. A possible explanation may be that the requirement for

linoleate is relatively less in the Cape Coloured subjects than it is in the other groups. Some of the factors said to increase the demand for linoleic acid are dietary fat and cholesterol. It has also been claimed that not total fat but the saturated fat intake is the factor giving rise to an increased demand for linoleic acid (30). Analysis of the total fat intake and the relative saturated fatty acid intake showed no correlation in any instance with the relative proportions of 18:2 in the depot fat in the three groups. The total unsaturated fatty acid intake would therefore also not be expected to correlate.

No data are available on the cholesterol content of the diet in these subjects, but as the serum cholesterol concentration is largely determined both by the nature of the dietary fat and by the dietary cholesterol content (31), it can to some extent be used as an index of the dietary factors that determine the demand for linoleic acid. There is, however, no significant correlation between serum cholesterol concentration and the percentage of 18:2 in the depot fat ( $r = -0.053$ ;  $P > 0.1$ ).

#### Relation to Other Fatty Acids

As can be seen from Table 5 the higher proportion of 18:2 among the Cape Coloured subjects is associated with the lowest proportion of 16:1 and 18:1 in this group. A significant negative correlation was found between 18:2 and both 16:1 and 18:1 (Table 8). No such correlation was found with any of the other major fatty acids, so that this relationship cannot be regarded simply as arithmetical. Although this correlation was not significant in all groups when analyzed separately, there would appear to be little doubt that this relationship between the fatty acids does, in general, exist. Kaunitz et al. (32) have noted a similar relationship between the proportion of 18:2 and 18:1 in rats. From their tables it would appear that 16:1 also shows this relationship to 18:2.

#### SUBJECTS WITH ISCHEMIC HEART DISEASE

The fatty acid composition in this group (Table 9) showed no striking variation from that in the apparently healthy subjects. On comparing the patient group with their most suitable control group—the White subjects in the same age range—the only significant ( $P < 0.02$ ) difference was the higher proportion of 18:2 in the patient group ( $10.1\% \pm 3.4\%$  and  $6.9\% \pm 1.7\%$  in patients and controls respectively). The percentage of 18:2 in the patients did not, however, differ significantly from either the Cape Coloured ( $P < 0.1$ ) or Bantu ( $P < 0.6$ ) groups in the same age range.

#### DISCUSSION

The most striking feature about the fatty acid composition of human depot fat, when considering all the

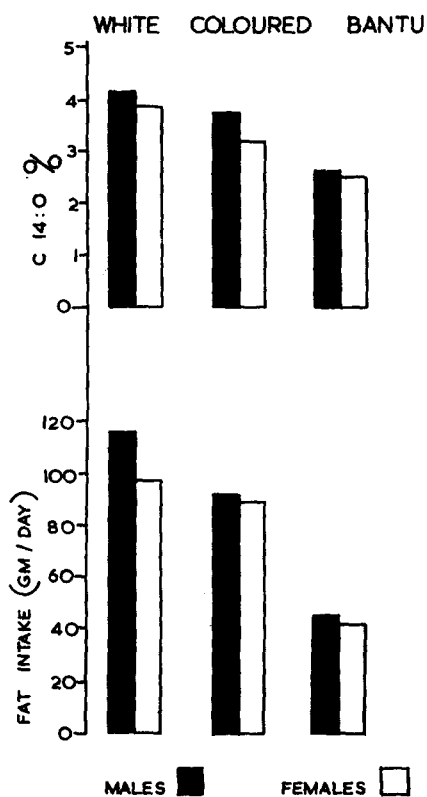


FIG. 2. The fall in the proportion of 14:0 in depot fat from White through Cape Coloured to Bantu, in both males and females, is paralleled by the decrease in total fat intake through these groups.

TABLE 8 CORRELATION OF 18:2 WITH MONOUNSATURATED DEPOT FATTY ACIDS

Correlation with	Males (n = 67)		Females (n = 70)		Both Sexes	
	r*	P	r	P	r	P
16:1	-0.465	<0.001	-0.476	<0.001	-0.439	<0.001
18:1	-0.340	<0.01	-0.376	<0.01	-0.356	<0.001
16:1 + 18:1	-0.441	<0.001	-0.555	<0.001	-0.476	<0.001

\* r = correlation coefficient.

available data, both from here and elsewhere (1, 2, 13-16), is its qualitative and quantitative consistency. Although the range in the percentage of each of the major fatty acid components is considerable, the standard deviation is relatively small when one considers the marked differences in dietary habits among the various groups. This could suggest that diet plays a relatively minor role in determining depot fat composition. However, it is evident that fatty acids common in human depot fat are, in general, paralleled by their relative abundance in nature (33). Human depot fat is therefore not characterized by a particularly distinctive fatty acid composition. The role of dietary fatty acids in determining depot fat composition may therefore be of primary importance.

Age was not found to be related to any difference in fatty acid composition at the age levels studied. Although the depot fat composition in the newborn is quite different from that in the adult (3), an adult type of fatty acid pattern would seem to be established by the age of 1 year (34), and possibly sooner. It thus appears that depot fat, after the first few months of life, remains fairly constant in composition throughout life.

INTERRACIAL DIFFERENCES

Despite the over-all consistency in depot fat composition, the data reveal some differences between racial groups: myristate decreases progressively from White through Cape Coloured to Bantu in both males and females, and linoleate is higher in the Cape Coloured subjects (Table 5).

It is of interest that myristic acid constitutes, on the average, about 3% of the total fatty acids in most natural fats and oils derived from both animal and vegetable sources (33). This figure agrees well with the proportion of 14:0 found in human depot fat. If, as this agreement suggests, the 14:0 deposited is derived primarily from dietary sources, the greater the total fat intake the higher should be the relative proportions of 14:0. There is indeed (Fig. 2) a striking correlation between total fat intake and the percentage of 14:0 in the depot fat. Similar findings in the relative proportion of myristate have been noted between White, Asian, and African groups in East Africa (16). The dietary habits in those groups are

apparently similar to those in our three groups. In Jamaica, where the proportion of 14:0 in common dietary fats (coconut oil) is high, the proportion of depot fat 14:0 is high (15). Endogenously synthesized 14:0 has been found not to accumulate in the depot fat of the rat (29). This further serves to emphasize that the deposition of this fatty acid is dependent upon its availability in the diet.

Linoleic Acid

No dietary factor could be found to account for the difference in the proportion of 18:2 among the racial groups. The relation between the proportion of 18:2 deposited in rat adipose tissue on a constant intake of linoleate and the chain length of the other dietary fats fed (31) may possibly account for the interracial differences reported here. It was, however, not possible to assess this from the available dietary data.

A possible explanation for these findings may be that the available regulatory mechanisms are directed at maintaining the composition of depot fat in an optimal physicochemical state. The fact that animals respond to cold environmental temperatures by an increase in the degree of unsaturation (35), suggests that it is necessary for this tissue to be maintained at a required de-

TABLE 9 FATTY ACID COMPOSITION IN THE SUBJECTS WITH ISCHEMIC HEART DISEASE

Fatty Acid	Mean	SD	Range
12:0	0.3	0.1	0.1-0.5
14:0*	3.5	0.9	2.3-6.1
14:1	0.9	0.2	0.6-1.2
15:0	0.6	0.1	0.3-0.9
15:2	0.3	0.1	0.1-0.5
16:0*	23.8	3.1	19.0-30.0
16:1*	8.2	1.7	6.1-10.5
17:0	0.5	0.3	0.2-1.3
17:2	0.7	0.2	0.5-1.3
18:0*	3.6	0.7	2.9-5.2
18:1*	45.3	3.1	41.5-50.3
18:2*	10.1	3.4	5.2-15.8
18:3	0.5	0.3	0.2-0.9
20:0	0.8	0.4	0.4-1.6
20:1	1.1	0.7	0.5-2.0
20:4			0-Trace

\* These major components constituted 94.5 (±1.3)% of the total fatty acids.

gree of "fluidity." Thus when there is an increase in the proportion of 18:2, for whatever reason, these conditions could be most readily maintained by a proportional decrease in the other unsaturated fatty acids and this could be controlled by the available regulatory mechanisms.

The many months required to produce a significant change in depot fat composition despite massive loading with linoleic acid (3) are of great interest. The deposition of such a fat would give rise to a fatty acid composition with markedly different characteristics from that normally present. It is therefore possible that under those dietary conditions there may be selective mobilization of the unsuitable fatty acid from adipose tissue. Such a form of control may in part account for the rather remarkable over-all homogeneity in human depot fat composition.

It is of interest that despite marked differences in the dietary habits of the racial groups the depot fat composition was so similar among them. It is evident that depot fat regulatory mechanisms are most efficiently able to maintain an "ideal" or optimal composition.

#### SEX DIFFERENCES

The evidence that the turnover of depot fat in both animals and man is greater in the female than it is in the male (36-40) tempts a speculative suggestion for the sex differences reported here. It has been shown in the dog that there is preferential release from the depots of the fatty acids 16:1 and 18:1. This, together with the finding that heart muscle, at least, preferentially extracts oleic acid, has given rise to the suggestion that there is a relatively greater turnover of the monoenoic acids (41). Apparently<sup>2</sup> these monoenoic acids are also preferentially released in man. In the same way the relatively greater proportion of the monounsaturated to the saturated 16 and 18 fatty acids in female adipose tissue could be an expression of the increased turnover in females; the depot fatty acid composition reflecting a static state in dynamic equilibrium. This hypothesis is in keeping with the findings that, together with the increased rate of acetate incorporation into the total lipids of rat adipose tissue, whether due to an effect of age or stimulation by insulin, there was an increase in the percentage incorporation into palmitoleic and oleic acids (29).

The mechanism whereby these sex differences may be produced is also speculative. The *in vitro* fat synthesis from acetate by female rat adipose tissue is greater than in the male. This is associated with an increase in the rate of oxidation of glucose-1-C<sup>14</sup> in the female tissue which is abolished by small doses of stilbestrol given to male rats (42). Estrogens, in promoting glycolysis via the

<sup>2</sup> Krut, L. H., and B. Lewis, data to be published.

hexose monophosphate shunt, could therefore produce these sex differences by the same mechanism that has been postulated as operative in increasing the rate of both total fatty acid and monoene synthesis by insulin (29).

The over-all degree of obesity in the female is not related to the fatty acid ratio 16:1 + 18:1/16:0 + 18:0. It has, however, been found that while these sex differences were present in tissue sampled from the buttock region they were not present in tissue sampled from the trunk (43); the females showing a difference between body sites. This could indicate that there are differences in turnover rate between different body sites in the female. The sex difference in the degree of body fatness is not only that females tend to be more obese but in addition they show a relatively greater deposition over the limbs. The ratio of the arm to subscapular skinfold thickness in the subjects sampled for this study was 0.73 in the males compared with 1.07 in the females and this difference was statistically significant ( $P < 0.001$ ).

The absence of any sex difference in the proportion of 18:2 in any of the racial groups (Table 4), despite differences among the races, suggests that men do not have a greater requirement for this fatty acid, at least at these levels of intake.

#### SUBJECTS WITH ISCHEMIC HEART DISEASE

The findings in this group are not in agreement with the claim of Kingsbury et al. (44) that, when matched for age, subjects with IHD have lower dienoate in their depot fat than do control groups. There is therefore no support for the view that IHD is related to an EFA deficiency, or to an increased demand for EFA. This latter point is particularly stressed since the intake of linoleate in the IHD group was much the same as in the other groups, namely 2.8% of total daily calories (see Table 7). According to Hegsted et al. (15), 18:2 is the fatty acid showing the greatest variation in the depot fat of any group of subjects, and the findings in this study are in agreement with this. Why a small difference in the proportion of 18:2 between one group and another should be interpreted as indicating EFA deficiency is rather obscure.

Fatty acid analysis of serum lipids drawn from healthy White and Bantu males and White males with IHD have, interestingly, shown that the Bantu have significantly lower levels of 18:2 in each lipid class than both the White control group and the patient group.<sup>3</sup>

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<sup>3</sup> Young, G., data to be published.



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