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Part I

Fats in Human Nutrition

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"In view of the importance of atherosclerosis and coronary disease as public health problems in the United States, a broad program of fundamental research in the field of fat metabolism in general, and of the etiology of atherosclerosis in particular, is needed and should be encouraged by foundations, industries, universities, and government.

"*Many problems must be solved in order to obtain the fundamental knowledge necessary for sound conclusions regarding fats in the human diet; they emphasize the urgent need for basic research on every facet of lipid metabolism. To illustrate the type of information needed to establish firmly the optimum fat intake for humans, the role of essential fatty acids in the development of the generative diseases, the safety of hydrogenated fats, and other practical objectives, commercial interests should address their attention to a re-evaluation of the nutritional effects of current processing practices. Since current trends demand that the metabolism of the naturally occurring fatty acids and of the numerous isomers produced during hydrogenation be compared, methods must be developed for preparing pure fatty acids and their isomers in quantities sufficient for human feeding experiments (pilot plant scale). Chemical alterations produced in fats during heating should be defined, and studies should be made of the heated oils produced in food processing and under some cooking conditions. Shortening and margarine producers should re-evaluate the merits of blended fats and anticipate the problems that will be raised by increased demand for such fats. There is every reason to believe that conclusive answers can some day be given to the several important questions involving the role of dietary fat in human health. To this end, research on all of these facets which may throw light on the overall problem should be strongly encouraged and supported.* Human health and science will share alike in the knowledge gained thereby."

Committee on Fats in Human Nutrition
National Research Council
Bulletin #575, 1958.

THE ROLE OF FATS in human nutrition and the responsibility of the fat and oil industry in supplying fats of high nutritional value have been of concern to the National Research Council (1) and the Food and Drug Administration (2). That the Twenty-first Congress of the Communist Party of the U.S.S.R. has had similar problems and thoughts on the matter is exemplified by the following quotation, "There has also been considerable change in the idea of the comparative value of different fats. The widespread idea that the best fats were those of animals (first of all of milk) has become very doubtful. Actually, animal fats as compared to plant fats are poor in essential fatty acids, linoleic and linolenic, which the human organism cannot synthesize, although it needs them. On the other hand, animal fats contain relatively more cholesterol. If such a combination is not the direct reason for sclerotic changes in the walls of the blood vessels, at any rate it predisposes them to these changes. Therefore there is good reason to increase the relative amount of plant fats in foods." (3). It seems apparent that apprehension and the dissemination of a type of misinformation, which has been banned by our own Food and Drug Administration (4), is not the exclusive concern of a capitalistic or a communistic society; it concerns everyone. As the Midwest is a prime producer of both animal and vegetable fats, this worldwide indictment of fats is of particular concern to Illinois. It is impossible to cover every aspect of the role of fat in human nutrition, but I hope my presentation will add light rather than friction to the subject.

Fat has at least three important functions in the human diet; they are a culinary, a physiological, and a nutritional function. The chemist in charge of refinery operations knows how difficult it is to remove odors and flavors from soybean oil. On the other hand, this ability to carry odors and flavors is beneficial in the culinary arts. Fats also add to palatability of meats and tenderness in baked goods. As fats serve as a convenient means of rapid heat transfer, they have found increasing use in commercial frying operations.

Although fats are usually thought of as simple triglycerides, that is, three moles of fatty acids esterified with glycerol, the fatty acids and their derived glycerides have been found in many forms and combinations in animal tissue. The esters of fatty acids include compounds with α-glyceryl phosphoric acid to form phosphatidic acids, with α-glyceryl phosphoryl esters to form lecithins, phosphatidyl ethanolamines and phosphatidyl serines, with sphingosine to form sphingomyelin and cerebrosides, and with inositol N-phosphate to form phosphoinositides. In a typical sphingomyelin, for example, linoleic acid is joined through an amide linkage to the –NH– of sphingosine and phosphocholine joined through an ester linkage to the terminal hydroxyl of sphingosine. Thus this group of fat soluble materials or lipids owe much of their importance in a biological system to the fact that they represent compounds which can bridge the gap from water insoluble to water soluble materials. They are believed to be present in biological membranes and interfaces and take part in physiological functions vital to the life process (5). Analysis of the isolated derived glycerides indicates that many contain the highly unsaturated linoleic acid which does not seem to be synthesized *in vivo* by the higher animals.

The dietary fats, as such, must supply the linoleic acid which is needed for the synthesis of these derivatives and for the synthesis of the triglycerides which are specific to the depot fats of the individual animal. Hilditch devoted his entire career to the analysis of animal and plant fats and pointed out that each living organism contained triglycerides of a specific structure which best fitted its needs (6). The ability to reject and pick certain fatty acids from the dietary fat for the synthesis of specific triglycerides has been illustrated by feeding linseed oil to chickens (7) and turkeys (8). The skin fat of turkeys which had been fed 1% linseed oil contained only 4% linolenic acid (Table I). In comparison, the

TABLE I

Composition of Fat Extracted from Skin of Chicks Fed Linseed Oil

Gr.	Linseed oil	Saturated	Oleic	Linoleic	Linolenic
	%	%	%	%	%
1	0	34	32	31	2
2	6	32	14	27	27
3	12	13	40	20	27
4	25	13	39	21	26

skin fat of chickens which had been fed 6% linseed oil contained 27% linolenic acid. Further increases in dietary linseed oil to 12 and 25% linseed oil did not result in an increased deposition of linolenic acid. Thus a bird is able to metabolize small amounts of linolenic acid; when flooded with large amounts of an unsaturated fatty acid, a breakdown in fat metabolism seems to occur. Similar results have been noted when large amounts of highly unsaturated fats were fed to animals (9). The data presented by Hilditch also indicate that within limits an animal seems to be able to reconstitute dietary fat into its own specific triglycerides. However when these limits are exceeded by flooding the animal with either a hard or a soft fat, the dietary fats do not seem to be metabolized at a rate which is fast enough to prevent alteration in the composition of the depot fats.

A portion of the triglycerides are stored around vital organs and serve as a protective cushion for them. How the consumption of excessive amounts of either saturated or unsaturated fatty acids affects the functional value of the triglyceride stores around the kidney, for example, is not known but should be considered. A substitution of one mole of oleic for stearic acid in an α-position lowers the melting point of tristearin from 73 to 38°C. and of tripalmitin from 66 to 35°C. When one considers that the body temperature of a human being is 37.2°C., the substitution of linoleic or oleic acid for another mole of stearic or palmitic acid may be important to the consistency of the depot fat and its ability to act as a cushioning agent to vital organs. Monostearin-diolein and monopalmitin-diolein have melting points of 23 and 19°C. respectively, both well below body temperature (10).

Fat seems to have another physiological function, namely, its influence on the rate at which the contents of the stomach empty into the intestinal tract. Fat depresses both the motility of the stomach and secretion of gastric juice (11) and delays hunger contraction. Thus fat contributes to the satiety value of a meal. Furthermore other factors such as the total roughage content of the diet may be a factor in lipid absorption and excretion (12).

The actual absorption of fat has been shown to take place in the intestinal tract, although the processes occurring during absorption are largely a matter of conjecture (13). Bollman (14) and others have shown by cannulation of the thoracic duct that most of the products of fat digestion appear in the lymph. Chaikoff and collaborators (15,16) found however that in whatever form fed the longer chain fatty acids appear almost exclusively in the lymph while those with less than 12 carbon atoms appear to a greater extent in the portal vein (17). Since most of the fatty acids of the diet are of the 16 to 18 carbon chain variety, the product of fat digestion will appear largely in the lymph.

The exact mechanism by which absorption occurs is not known. Frazier's hypothesis that small canals of about 0.5 μ diameter in the intestinal cells furnish a simple passageway (18) has been questioned (19). It may be more accurate to think of the intestinal tract as a site at which the digestion of lipids begins rather than as a simple passageway.

Desnuelle (20) has noted that the action of lipases on triglycerides gave rise to a series of reactions from triglycerides to diglycerides to monoglycerides. It has been established that this attack is not random but appears to be directed almost exclusively toward the 1 and 3 position of the glyceride (21,22). These mono- and diglycerides probably play an important first

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step in the absorption of lipids; however lymph lipid is composed of only 2.5% mono- and diglycerides, free sterols, and free fatty acids. The lymph has been shown by Clement and Mead (23) to contain in addition 3.2% sterol esters, 4.3% phospholipids, and 90.0% triglycerides.

Environmental factors in the intestinal tract seem to play an important role in lipid absorption as shown by the following experimental data. Mature rats in which the main intestinal lymphatics had been cannulated with polyethylene tubing were fed one ml. of test sample with the aid of a stomach tube and the intestinal lymph collected every two hours for a total period of 12 hours. The lymph was defibrinated and its optical density measured at 650 mμ. The results were expressed as the optical density multiplied by the weight of the lymph collected every two hours (Figure 1).

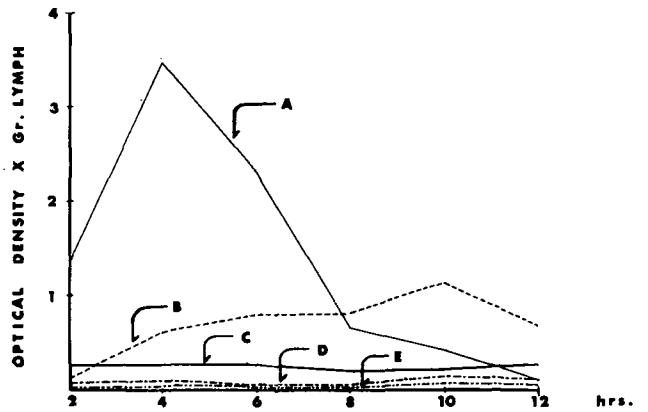

FIG. 1. Effect of the administration of the hydroperoxide of methyl linoleate on intestinal lipid absorption in lymph cannulated rat. A. Methyl linoleate. B. 20% hydroperoxide of methyl linoleate in methyl linoleate. C. None. D. Hydroperoxide of methyl linoleate. E. Methyl linoleate 24 hours after the administration of 1 ml. hydroperoxide of methyl linoleate. (A higher optical density (O. D.) indicates greater lipid absorption than a low O. D. value.)

The lymph obtained from rats two to four hours after they had been fed methyl linoleate had an optical density of 3.48 indicating that rapid absorption of methyl linoleate had taken place. In comparison, the lymph from those fed the hydroperoxide of methyl linoleate or methyl linoleate 24 hours after the administration of the hydroperoxide of methyl linoleate had a value of 0.09 and 0.01 respectively. These values represented less lipid than for those given no supplement whatsoever. When the hydroperoxide of methyl linoleate was diluted with 80% of methyl linoleate and fed, the lymph again contained more lipid than the lymph from the nonsupplemented group, or values of 0.61 and 0.26, respectively (24). Environmental factors such as hydroperoxide can therefore influence lipid absorption and even a simple ester such as methyl linoleate does not find a ready pathway through the intestinal wall.

The rate at which triglycerides are absorbed from the intestinal tract has served as a means of comparing the relative digestibility of hydrogenated vegetable fats with animal fat. Deuel (25) has shown that such comparisons of coefficiencies of digestibility is more dependent on the proportion of saturated triglycerides in the fat than on the origin of the fat. In fact, the triglycerides in commercially available fats or "shortenings" are so carefully blended that a distinction between them based on the coefficiencies of digestibilty is misleading. The mixed fatty acid and triglyceride composition of an animal fat such as blended lard does not differ sufficiently enough from a commercially available hydrogenated shortening to be significant to the coefficiency of digestibility.

Hydrogenated vegetable fats, especially margarines, contain significantly larger amounts of *trans* fatty acids than animal fat (26). If differences in fatty acid content of edible fats will ever have biological significance, it may depend (27) on the *trans* fatty acid content of the fat, although these fatty acids seem to be readily metabolized (28) (Table II). *Trans*

TABLE II

Trans Fatty Acids in the Carcass Fat

Group	*Trans* fatty acids
	%
I. 10% Margarine stock	
Decrease to in:	
0 month	18.0
1 month	6.5
2 months	4.4
II. 5% Margarine + 5% olive oil	
Decrease to in:	
0 month	10.8
1 month	4.9
2 months	2.8

fatty acids were only detected in the tissues of animals which had received a diet containing *trans* fatty acid. At the end of the first month of feeding, the animals contained between 15.5 and 18.8% of *trans* fatty acids in the carcass fat; at the end of the second and third month the *trans* fatty acid content of the carcass increased less than 2 and 1% respectively. Animals which had received 10% of dietary fat in the form of 5% margarine stock and 5% olive oil contained approximately half the amount of *trans* fatty acids found in the carcass fat of animals which had received 10% of margarine stock.

The percentage of *trans* fatty acids in the carcass fat decreased when *trans* fatty acids were removed from the diet. However they did not completely disappear from the tissue even at the end of two months on a diet free of *trans* fatty acid. After one month on the diet free of *trans* fatty acid, the carcass fat of the rats which had received 10% of margarine stock had decreased to 6.5% and after two months to 4.4% of *trans* fatty acids. The carcass fat of the animals which had received margarine stock and olive oil contained approximately 11% of *trans* fatty acids. After one month on a diet free of *trans* fatty acids, this had decreased to 4.9% and after two months to 2.8% of *trans* fatty acids.

The *trans* fatty acids present in human tissue (29) apparently arise solely from dietary fat, and as in rats, they do not normally appear in the tissues unless a source of *trans* fatty acids is included in the diet. Samples of fat from human placental, maternal, fetal, and baby tissue were examined for the presence of *trans* fatty acids. While the maternal tissue contained considerable amounts of *trans* fatty acids, these lipids were not found to any measurable extent in placental, fetal, or baby fat.

The fat absorbed through the blood and lymph is either deposited in the various tissues or used immediately as a source of energy. In the blood, fat appears to be transported in large part in the form of tiny droplets termed "chylomicrons." The fatty acids are also present as components of phospholipids, cholesterol esters, and lipoproteins. Normal human blood

plasma contains from 245 to 470 mg. fatty acids, 120 to 350 mg. phospholipids, and 110 to 310 mg. cholesterol per 100 ml. of plasma (30).

The human body contains approximately 20% of ether extractable or "lipid" material (31). The amount of fat deposited is dependent on the tissue. The adipose tissue, heart, and skin contain the most fat or 71.6, 16.6, and 14.2% respectively, and the lungs and spleen the least fat or 1.3 and 1.2% respectively. Striated muscle contains 6.6%, liver 3.1%, and the kidney 7.2% fat. The adipose tissue fat contains approximately 4% (C_{14}) myristic, 25% (C_{16}) palmitic, 7% (C_{18}) stearic, 6% (C_{16}) palmitoleic, 46%(C_{18}) oleic, and 2% of fatty acids which are shorter than 14 or longer than 18 carbon atoms in length (32). These fatty acids may be classified the "nonessential fatty acids" as they can all be synthesized in the body from nonfat precursors. In addition the body fat contains approximately 9% (C_{18}) linoleic and 1% (C_{20}) arachidonic acid which contain two and four double bonds respectively. These two fatty acids have been classified the "essential fatty acids" as linoleic acid cannot be synthesized in the body and serves as an essential precursor for the synthesis of arachidonic acid.

It has been shown that the normal oxidation of fats is dependent on the presence of one or more of the breakdown products of carbohydrate metabolism. These breakdown products are formed in what is known as the Krebs citric acid cycle, the system in which carbohydrates are metabolized. It is believed that before breakdown, fats are hydrolyzed to glycerol and free fatty acids. The glycerol combines with phosphate and glycero-phosphate, so formed (33), oxidizes to pyruvic acid which is also the key breakdown product of glucose to enter the citric acid cycle. In the presence of the appropriate enzyme system and the energy-rich adenosine triphosphate (ATP) pyruvic acid can act as an acetylating agent and can convert coenzyme A into "acetyl coenzyme A" (34). Coenzyme A or "Co A" is a derivative of the vitamin pantothenic acid and contains in addition phosphate, adenylic acid, and mercaptoethanolamine (NH_2CH_2-CH_2SH). The acetyl group is linked to the coenzyme by means of a thiol ester linkage.

The free fatty acids are normally completely oxidized to carbon dioxide and water through the citric acid cycle (35). They are first esterified with Co A in the presence of ATP, a two-carbon fragment is split off to give acetyl Co A and Co A derivative of the next lower fatty acid. Thus a long-chain fatty acid can be completely degraded to C_2 fragments without the intermediate formation of free short-chain fatty acid. The acetyl Co A combines with oxaloacetate to form citrate (36). The oxaloacetate is subsequently regenerated through the citric acid cycle with the formation of two molecules of carbon dioxide corresponding to the two carbon atoms of the acetate entering the cycle (37).

The acetyl Co A which is not metabolized to carbon dioxide and water can add to the metabolic pool of acetyl Co A and is immediately used to build up new fatty acids and new fat, nonessential amino acids, sterols, and other lipid material. The nonessential unsaturated fatty acids, palmitoleic and oleic acid, are synthesized from the corresponding saturated fatty acids, palmitic and stearic acid, with the aid of tissue dehydrogenases (38). Therefore any one of the nonessential fatty acids can be consumed as dietary fat or synthesized from acetyl Co A (39).

In the absence of linoleic acid, more oleic acid is synthesized in order to keep the total unsaturation as near to "normal" as possible (Table III). For example, animals which had received 5% corn oil contained 29.4% oleic acid in comparison with 37.2% oleic acid for those on a fat deficient diet. The animals which had received corn oil contained almost 4 times more arachidonic acid than those on the fat-free diet, or 8.8 and 2.6% respectively. This difference in arachidonic acid content was also reflected in the offspring from these animals. The young from the animals which had received corn oil contained almost twice as much arachidonic acid as those from the animals on the fat-free diet, or 17.2 and 10.2% respectively.

TABLE III
The Mixed Fatty Acid Composition of Mother and Young in Terms of Percentages

	Mother		Young	
	Fat deficient	Corn oil	Fat deficient	Corn oil
Percentage yield fat	12.2	14.7	1.2	1.3
Iodine value	75.0	84.8	110.6	116.0
Percentage				
Linoleic acid	18.0	16.2	2.7	7.6
Linolenic acid	0.0	0.0	2.4	0.0
Arachidonic acid	2.6	8.8	10.2	17.2
Oleic acid	37.2	29.4	72.2	49.6
Saturated acid	42.1	45.5	12.4	25.4

The arachidonic acid seemed to be concentrated in the phospholipid fraction in both the mature animals and young. The most striking differences were noted in the phospholipid fraction of the fat extracted from the young. The young from the animals which had received corn oil contained from 5 to 10 times more arachidonic acid than those from the animals on the fat-free diet. It was interesting to note that trienoic acid was not present in the phospholipid fraction of the animals which had received a dietary source of preformed fat.

The energy requirements of the body are normally met by metabolizing fat and carbohydrate together. If for any reason carbohydrate metabolism is subnormal, correspondingly more fat has to be metabolized. Under these conditions there is a deficiency of carbohydrate metabolites and a tendency for acetyl Co A to accumulate for lack of oxaloacetate with which to combine to yield citrate (40). Two carbon units then tend to accumulate and pairs to combine to yield acetoacetate, from which β-hydroxybutyric acid and acetone, the other two members of the "ketone bodies," can form. Thus ketone bodies accumulate when the diet contains disproportionately large amounts of fat, when the glycogen reserves of the liver have been exhausted as in starvation, or when the liver's power to store and metabolize glycogen is seriously impaired as in diabetes.

The lipid content of human tissue decreases with an increase in age (Table IV). For example, Briscoe *et al.* (41) have shown that lung tissue which contains 10.1% lipid on a dry weight basis at 25 years of age contains only 4.3% at 85 years of age. It would therefore seem that geriatric foods should be designed to contain more fat, rather than less fat, if it is desirable or possible to reverse this trend in aging. The tissues also vary in the kinds of fatty acids deposited in them. Holman has shown that the most vital organs such as the heart contain a higher percentage of linoleic

TABLE IV

Average Concentration of Lipid in Human Lung per Decade of Adult Life

Age	Lipid[a]
yr.	%
25	10.1
35	9.2
45	8.3
55	7.4
65	6.5
75	5.5
85	4.3

[a] Dry weight.

and arachidonic acid than the depot fats (42). As linoleic acid must have a dietary source, it might be well to increase rather than decrease the percentage of this essential fatty acid in foods designed for the aged.

It has been shown that linoleic acid is converted to arachidonic acid *in vivo* provided the tissue contained an adequate amount of vitamin B_6 (43). By means of tracer techniques, Mead and his co-workers have investigated the mode of *in vivo* conversion of arachidonate from linoleate or γ-linoleate and other highly unsaturated fatty acids. When linoleate-1-C^{14} was fed to rats, the arachidonate fraction isolated from the tissue contained radioactivity, and 74.7 and 24.5% of the total activity was found to be located at the carboxyl carbon and the 3-position respectively. Only a trace of activity was detected at the 2-position, and no activity was found in the 4-position or in the rest of the chain (44). This indicated that linoleic acid was a precursor of arachidonic acid. The findings also indicated that a part of the radioactive linoleate underwent β-oxidation *in vivo*, and some of the two-carbon fragments thus freed were then condensed at the carboxyl of the linoleate. Condensation of linoleate and a two-carbon fragment to form an arachidonate skeleton has also been confirmed by using radioactive acetate and unlabelled linoleate (45). Similar tests proved the absence of formation of arachidonate from linoleate (46).

A survey by the United States Department of Agriculture of food disappearance in the United States indicates that we are now consuming more meat, milk, and eggs and less potatoes and cereals than Americans did forty years ago. This shift in consumption has seemingly represented an advancement in nutrition as it increased the protein and vitamin intake. However as such foods contain a larger amount of fat than bread or potatoes, the total fat intake also increased. To circumvent the weight problem brought on by the intake of extra calories from this increased fat consumption, a flood of "diets" and sometimes recommendations which are contrary to sound nutrition have been advocated.

The Committee on Foods of the National Research Council has considered carefully the caloric requirement for the average American. The Council has recommended that 3,000 calories should be consumed as individual foods to provide a protein intake of 70 gm. per day. If foods are chosen to include milk, meat, and eggs as protein sources and sufficient bread, fruits, and vegetables to provide for an adequate caloric and vitamin C intake, no expensive reducing aids or supplementation with vitamin pills is recommended or necessary (47).

The recommended intake of dietary fat is lower than the actual intake. The proportion of fat in the American diet may be too high for optimum health (Table V). Whether the higher than recommended intake of fat is important to human health is still unknown. In fact, an experiment in which human subjects are used to test this hypothesis is difficult to execute. It is possible however to use rats or chickens in an experiment in which both the level of fat and protein is varied and to note how much variations affect weight and physiological factors such as the serum cholesterol level (48).

Dietary protein was shown to depress the atherogenic effect of dietary cholesterol and fat in chickens. The serum cholesterol, serum lipids, and β-lipoprotein levels of birds fed a low protein diet were found to be higher than those fed a high protein diet. Furthermore, it was noted that the energy value of a diet as calculated from the total caloric intake of carbohydrate, fat, and protein, had a relationship to serum cholesterol levels. The energy to protein or the E/P ratio of a food item can for convenience be calculated by dividing the total available calories in 100 g. of the food item or diet by its protein content (Table VI).

TABLE V

Daily Caloric Consumption as Recommended by the National Research Council Compared with Estimated Consumption Data of USDA

	NRC	USDA
	%	%
Protein	10–15	15
Carbohydrate	55–70	42
Fat	20–30	43

An analysis of variance showed that at low or moderate levels of corn oil, statistically significant lower serum and carcass cholesterol levels were noted on a high as compared to a low protein diet. When the energy supplied by dietary corn oil was increased from 1.3 to 57.1%, the serum cholesterol value decreased from 200 to 136 mg. % at a low protein level (E/P ratio 22.6) and from 166 to 129 mg. % at a high protein level (E/P ratio 11.5). Thus an excessive amount of dietary corn oil decreased serum cholesterol levels. However the level of dietary protein influenced the cholesterol-depressing effect of corn oil, as lower serum cholesterol levels were noted in birds kept on the high as compared with those on the low protein diet. It was interesting to note that almost twice as much total carcass fat was found at an E/P ratio of 22.6 than at an E/P ratio of 11.5 or approximately 9 and 5% respectively. Variations in dietary fat or dietary protein did not alter the percentage of total carcass fat significantly as long as the E/P ratio remained constant.

At a low protein level a methionine-deficient diet significantly elevated serum cholesterol levels. A diet low in choline also significantly increased serum cholesterol levels at the 15% protein level but not in those fed higher levels of protein. A low choline, low methionine diet significantly increased serum cholesterol levels, but this elevating effect was again not obtained at the higher protein levels (49).

These results were obtained in chickens on diets not ordinarily consumed by man. However a similar trend in response to changes in the proportion of the E/P ratio was noted in weanling rats fed evaporated milk in comparison with two popular proprietary infant formulas. On this study, the evaporated milk and infant formulas were diluted as directed on the label with an equal volume of water and fed under *ad libitum* and restricted conditions. The results indicated that evaporated milk had superior nutritional

value because of its higher protein content. When the proprietary infant formulas were supplemented with sodium caseinate and lactose so as to bring the protein and the caloric level to that of evaporated milk, no difference in growth rates was apparent. Although weanling rats grow more rapidly than human babies and therefore have a higher protein requirement, it is apparent that from the moment of birth the E/P ratio could influence the rate of growth and possible still unmeasured physiological factors. It is conceivable that an excess intake of protein is not desirable as the excess would be deaminated and the resulting deaminated amino acids could also be converted to acetyl coenzyme A, to fat, or to cholesterol. Although still fragmentary in the necessary detailed experimental evidence (Figure 2), a schematic diagram which involves at least 6 factors in the lipid carrying capacity of blood serum may be functional: 1) the total calories expended as mechanical energy and thus not available for conversion to lipid; 2) the fat, cholesterol, and glycogen storing capacity of the liver; 3) the rate of synthesis of cholesterol esters; (4) the rate of synthesis of cholesterol; 5) the rate of synthesis of fat (fatty acids, triglycerides, and phospholipids); and 6) the rate of synthesis of lipoproteins. The direction of synthesis of one or more lipid components can be shifted by "catalysts" involved in metabolic processes such as thyroid, insulin, sex hormones, and thiouracil.

Even some of the catalysts or cofactors which are necessary in metabolism have been shown to contain substantial amounts of lipid material. Cytochrome C has been shown to contain 50% lipid by weight (50) and the lipid portion of coenzyme Q has been shown to contain 5 to 8% neutral lipid and 92 to 95% phospholipid (51). The neutral lipid of coenzyme Q also contained cholesterol and at least three still unidentified components. As the lipid components in these cofactors become known and the exact function of vitamins A, D, E, and K in biological systems elucidated, such data will no doubt provide answers to some of the questions in regard to fat metabolism.

It is possible that the proportion of "hard" fats in the diet is too high. If so, this can be remedied by less selective hydrogenation. However the increased rancidity problems involved in unsaturated fats which have been largely stripped of tocopherols during deodorization is considerable. Such fats have caused anorexia and muscle involvement in dairy calves (52) and liver necrosis in rats (53). The linoleic acid content of brain lipids was increased by feeding methyl linoleate (54) and highly unsaturated fatty acids precipitate encephalomalacia in chicks in diets low in vitamin E (55).

To single out "hard" or "soft" fats or dietary fats in general as the culprit in the heart disease problem is premature. Even a lack of exercise may be important as good muscle tone in the heart and leg muscles cannot be gained without mechanical effort. The National Research Council has pointed out that it would seem wiser to gather more knowledge before decisions on the role of dietary fats to human health are made.

Summary

Dietary fats represent the most compact chemical energy available to man. They contain twice the caloric value of an equivalent weight of sugar. However dietary fats should not be thought of solely as providers of unwanted calories as fats are as vital to cell structure and biological function as protein.

If an individual consumes food items of high fat content, an adequate protein and vitamin intake should be assured in order to provide the lipotropic factors necessary for normal fat metabolism. It may be more judicious to control the total caloric intake under such circumstances rather than to resort to periods of semi-starvation or to drastically decrease the dietary fat intake which could result in an increase in hunger pangs and an actual increase in total caloric intake. If the excess calories furnished by carbohydrates are converted to fat *in vivo*, the problem of obesity could not be solved under conditions of increased total caloric intake. The problem could be solved by a curtailed intake of a diet which includes meat, milk, eggs, vegetables, fruits, and sufficient cereals and bread to provide for an adequate protein, vitamin, and caloric intake.

Dietary fats provide the essential linoleic acid which seems to have both a structural and functional role in animal tissue. Although the optimum total intake of linoleic acid by man has not been established, it is evident that the level of intake in the American dietary pattern could be increased. However the indiscriminate substitution of soft for hard fats seems undesirable as an excess consumption of highly unsaturated fatty acids may change the functional value of the triglycerides in the depot fats and may put an undue stress on the antioxidant supply available *in vivo*.

TABLE VI

Effect of Variations in Dietary Protein and Fat on Growth, Protein and Fat Composition and Carcass and Serum Cholesterol Levels of Chicks [a]

Dietary		Energy from fat (calories)	Average wt. of birds	Carcass composition		Cholesterol level	
Corn oil	Protein			Fat	Protein	Carcass	Serum
E/P ratio 22.6			*gm.*	%	%	*mg.* %	*mg.* %
%	%	%					
0.6	15.1	1.3	358	9.6	17.7	112	200
8.3	16.8	19.9	382	9.1	18.4	98	180
18.1	18.8	38.5	399	8.3	18.1	95	176
30.9	21.2	57.1	371	9.7	17.8	99	136
E/P ratio 11.5							
0.6	30.0	1.3	405	4.1	18.8	87	166
8.4	33.4	19.6	499	4.7	19.0	80	140
18.2	37.7	37.9	440	5.7	18.6	76	136
31.1	42.3	56.3	434	5.6	18.7	75	129

[a] In this series 240 one-day-old male chicks were kept on the same basal diet for 7 days and then divided into 8 treatments of 3 replicates of 10 birds each. They were kept in standard chick batteries in a randomized block design and fed the 8 experimental rations *ad libitum* for a 3-week period. Cholesterol values of 6 individual chicks from each treatment (2 from each replicate) were used for an analysis of variance. The figures reported in this table represent an average of 6 cholesterol values in each treatment.

Least significant difference of serum cholesterol = 26.3 and 38.0 mg. % at 5 and 1% levels respectively.

Least significant difference of carcass cholesterol = 13.3 mg. % at 5% level.

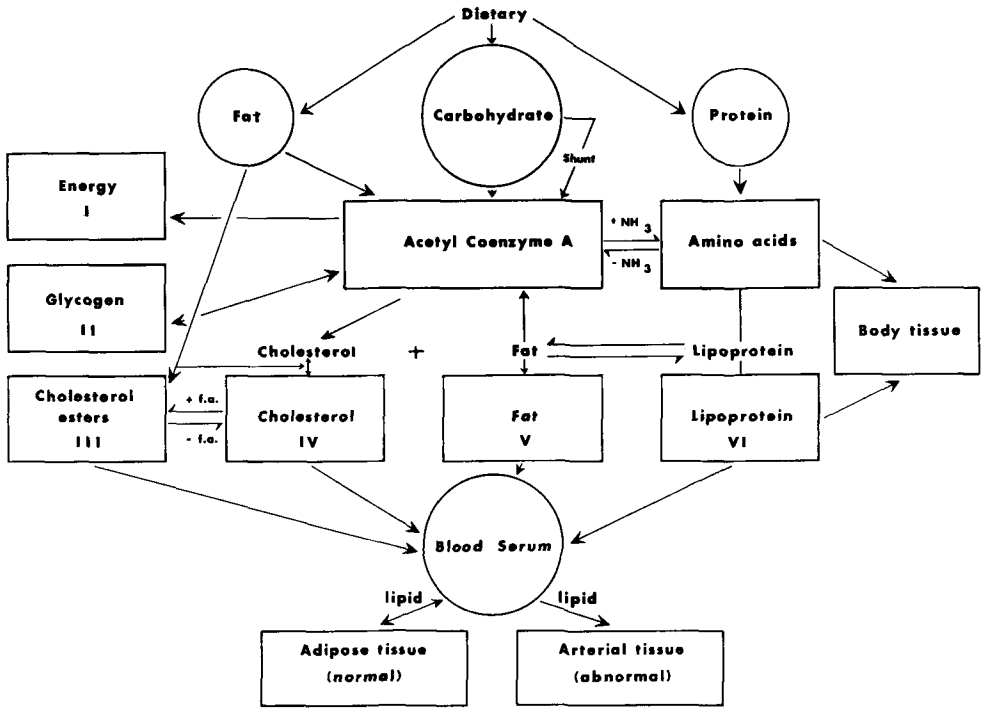

FIG. 2. Relationship between dietary factors and lipid-carrying capacity of blood serum.

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