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NUTRITIONAL PROPERTIES OF COCONUT OIL*

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ABSTRACT

Coconut oil has been one of the most widely used vegetable oils since the agricultural revolution. Only in recent years has there been controversy over the desirability of its use. Controversy has usually stemmed from observed disturbances of calcium or cholesterol metabolism when hydrogenated coconut oil was fed, frequently with inadequate linoleate supplementation, to experimental animals. Furthermore, in many of the studies involving cholesterol, entirely unphysiological amounts of cholesterol have been included in the diet. It is contended here that the findings in such studies are the consequence of abnormal nutrition rather than inherent defects in coconut oil. Evidence from epidemiological studies of arteriosclerosis in populations consuming large amounts of coconut oil are cited to show that coconut oil in a natural diet is not dis-advantageous and may even be of advantage. The high level of medium chain fatty acids in coconut oil is discussed from the point of view that they may contribute to beneficial effects on the part of coconut oil under some abnormal conditions.

The coconut palm grows so easily in tropical climates on sandy soil near the ocean and is so useful to humans that it has replaced other vegetation in large areas since prehistoric times (1-3). However, this development has had its dangers to evolutionary ecology. Each of the several million trees in existence in India, Malaysia, China and the Philippines may yield 1-2 gal of coconut oil annually for a total production of several million metric tons providing the main fat intake and a large part of the calorie requirements for a large area of our planet.

The rise in population and concomitant increasing demands for sources of dietary fats have made it an urgent problem to consider whether the nutritional properties of coconut oil are such that its production should be expanded or whether the consumption of other easily grown oils should be recommended. One could have expected that the mass nutritional experiment which has been under way with coconut oil for so many thousand years would have settled the question of its desirability compared to other oils; indeed, no objections to its use were raised until a few decades ago. At present, the somewhat vague ideas on the relationship of so-called saturated fats to arterio-sclerosis have raised some doubts about the usefulness of this oil. This has been particularly true since the introduction of hydrogenated coconut oil and its administration to experimental animals in purified diets.

On reviewing the nutritional work with coconut oil, one is overwhelmed by the magnitude of work that has been carried out and at the same time by the confusion and contradictions existing in relation to such topics as the occurrence of vascular lesions (so-called experimental atherosclerosis), cholesterol and calcium metabolism.

Because of the vastness of the literature on coconut oil, one can at best recognize the barest outlines of the many problems connected with the nutritional aspects of this fat. However, a review of pertinent material may focus attention at some existing contradictions and may help to clarify them.

Some of the chemical properties of coconut oil are uniquely different from those of other dietary fats and oils. Table I shows that about 80% of the constituent fatty acids of the triglycerides of coconut oil have a chain length of fewer than 16 carbon atoms. Laurate accounts for nearly half of the fatty acids.

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Except for other palm kernel oils, these acids occur in other dietary fats only in trace amounts; only butter contains 10-25% of short or medium chain length fatty acids. The many differences in the metabolic fate of these fatty acids compared to those having carbon chains of 16 or more have been repeatedly reviewed (4,5). Triglycerides containing the shorter acids hydrolyze more easily, and the shorter the acids, the higher is the percentage directly absorbed by the blood vessels rather than the lymph circulation. Most of the acids with carbon chain length of less than 12 are a ready source of energy in that they are rapidly converted into CO_2 (6); only traces are deposited in mammalian tissues.

Considerable interest has centered around the problem as to whether the high laurate content of coconut oil is biologically advantageous. Doubts are sometimes expressed because of adverse effects in experiments in which ethyl laurate is fed to rats (7). Such experiments are not pertinent to the question of whether triglycerides containing laurate are biologically advantageous not only because the nutritional effects of ethyl esters cannot be compared with those of triglycerides but particularly because such experiments often have been carried out with purified, essential fatty acid deficient diets. When glyceryltrilaurate was fed to rats in the presence of methyl linoleate, no abnormal effects were noted (8). At present, no work has been reported suggesting untoward effects in mammalians fed laurate-containing triglycerides in the presence of linoleate.

Caprylic acid (C_8), which is most readily metabolized by the liver (9), was found to have rather specific metabolic effects. Its feeding to rats strongly counteracted signs of vitamin B deficiency (10); its application reduced the carbon clearance time in rats suggesting that caprylate stimulates the RES (11, 12). Such effects gradually decreased as the chain length of the fatty acids increased.

Coconut oil triglycerides are characterized by a high laurate content in the beta position (13). This fact should be kept in mind because the retention of the laurate in the 2 position during absorption may be important (14).

The delicate liquid filling the immature coconut (coconut milk), which eventually turns into the lipid rich meat of the ripened fruit contains a large variety of pharma-cologically active substances. There is voluminous literature about growth factors for plant seeds (15, 16), materials preventing senescence of wheat leaves (17), etc. These materials probably deserve the nutritionists' attention because current work suggests that differences in the effects of dietary fats may be due to the action of minor constituents. It has been reported that feeding of coconut oil to lactating cows enhances the fat content of the milk (18).

Information about the effects of coconut oil on such general parameters as mortality and morbidity is extremely sketchy. Attempts to evaluate these factors in wide human populations have proved to be a hopeless task. In one study on mice (19) the animals fed coconut oil lived longer than the controls fed safflower oil. In this laboratory, a long term study is being carried out on the survival rates of groups of 80 male rats of the Charles River strain which have been fed, from the time of weaning, various dietary fats supplied at level of 20% in a purified diet (20). When the hydrogenated coconut oil diet was supplemented with 0.5% of a linoleate concentrate containing about 75% of linoleic acid, 17 of 80 animals died during the first year. When, at the end of the first year, the linoleate concentrate was raised to 1.5% of the diet (or 7.5% of the hydrogenated coconut oil), the mortality among the animals declined markedly. The mortality rates of the various groups between 360 and 500 days, based on those alive at 360 days, are given in Table II.

Evaluation by a chi square test with 9 degrees of freedom gave a value of 16.6 ($P < -0.05$) suggesting that differences between the animals with high and low mortality were real.

TABLE I
Fatty Acid Composition of Coconut Oil

Chain length	%	*Chain length*	%	*Chain length*	%
6 : 0	0.5	12 : 0	49.5	18 : 0	2.0
8 : 0	6.5	14 : 0	19.5	18 : 1	6.0
10 : 0	6.0	16 : 0	8.5	18 : 2	1.5

Data as to terminal histopathological findings in coconut oil-fed animals supplied with essential fatty acids are particularly scarce. In one such study, no differences between animals fed coconut oil or butter were noted (21).

Considerably more is known about the digestibility of coconut oil in men. It is nearly completely absorbed as are most other natural dietary fats (22-24). stools of children were the same after the feeding of coconut oil or milk fat (25). The growth rate of animals fed coconut oil has frequently been compared to that of controls fed butter or other natural dietary fats. No major differences have been reported (24, 26-28).

Coconut oil, like other fats, exerts a strong influence on the composition of the body's triglycerides especially those of the depot fat (26). Predictable changes in the composition of the depot fat could be recognized one week after feeding had started (29). When randomized saturated medium chain triglycerides (MCT) derived from coconut oil were fed to rats (30) the resulting depot fat contained above 50% laurate and myristate.

Coconut oil contains only 2-3% linoleate; it shares such a low linoleate content with olive oil (the second of the time-honored vegetable oils) and with milk fat, the most specific mammalian fat. One may theorize that the relatively low linoleate content of coconut oil and olive oil not only protects these fats from rapid rancidification but offers other (evolutionary?) advantages, a speculation suggested by the low linoleate content of milk fat. Modern vegetable oils may contain about 50% linoleate or high percentages of linolenic or eleostearic acid.

When the physical properties of coconut oil are changed by hydrogenation, it becomes more suitable for margarine production; therefore, mainly hydrogenated coconut oil containing at best 0.5% linoleate has been used for nutritional experiments during the last decades.

Coconut oil intake is associated with high linoleate requirements which can be concluded from the observation that deficiency signs occur early after the start of feeding (31). In view of this finding, it is of interest that the essential fatty acid requirements of rats fed MCT compared to those of rats maintained on saturated long chain (palmitate and stearate) triglycerides were higher in the latter group. At the same time, the linoleate content of the epididymal fat of the animals fed the longer chains was higher than that of the animals fed MCT (30). Rats fed hydrogenated coconut oil behave like those given triglycerides with longer chains because it was noted that feeding of hydrogenated coconut oil was associated with higher values of tissue linoleate (32). This emphasizes the higher linoleate requirements of animals fed hydrogenated coconut oil.

These observations probably explain a number of seemingly contradictory findings. It is obvious that the absence of an essential dietary element, such as essential fatty acids, produces deficiency symptoms. Comparisons of the effects of hydrogenated coconut oil with those produced by other fats should only be made if the diets contain all essential elements including essential fatty acids; otherwise animals having a deficiency disease are compared to controls. The violation of this principle greatly limits the conclusions which can be drawn from many studies. Thus, histological lesions were noted in rats fed hydrogenated coconut oil without essential fatty acids over a protracted period (33). The authors concluded that saturated fats were the cause of the lesions. However, it seems more probable that the lesions were due to the induced linoleate deficiency. Similar objections must be raised to a study in which the effects of alternately feeding rabbits 40% corn oil and 40% hydrogenated coconut oil were compared to effects in controls receiving a mixture of 20% of each. The animals receiving only hydrogenated coconut oil for

TABLE II

Mortality Rates of Male Rates Fed Various Fats

Fat	*No. of Animals*	%	*Fat*	*No. of Animals*	%
Cottonseed oil	15/80	19	Corn oil	7/77	9
Olive oil	10/78	13	Lard	7/79	8
Chicken fat	9/77	12	MCT (40 in group)	3/37	8
Soybean oil	8/78	10	Coconut oil	5/63	8
Butter	8/78	10	Beef fat	3/80	4

periods of 10 weeks were on diets deficient in an essential dietary element but the controls were not (34). These two papers have been singled out of many for a more detailed discussion because of the great care with which the experiments had been carried out.

Conditions are further complicated by the fact that the essential fatty acid requirements are also enhanced by the use of highly purified diets but the nature of the protective effect of diets made up of natural food stuffs (laboratory chow) has not been identified (35).

All of this has an important bearing on the discrepancies one encounters in the literature about the calcium metabolism of experimental animals fed coconut oil. When a rachitogenic diet made up of natural food stuffs was fed to rats. the inclusion of coconut oil had antirachitic properties; it improved the quality of the damaged bones (36, 37). When rachitogenic diets composed of purified materials including refined coconut oil were fed, negative Ca balances with reduced utilization of Ca were noted (28,38,39).

Some of these contradictions could be explained by the outcome of the above discussed experiment with male rats of the Charles River strain. Seventeen of 80 rats (21%) supplied with coconut oil containing 2.5% of a linoleate concentrate died during the first year of observation whereas only 3% of the rats fed other fats died. Autopsy findings (gross and histological) on the dead animals fed coconut oil have revealed the presence of advanced secondary hyperparathyroidism in three of the 17 animals so far examined. The renal insufficiency which is the basic cause of this condition is associated with phosphorus retention, causing hypocalcemia; this in turn stimulates the parathyroid glands which hypertrophy and excrete large amounts of parathormone leading to calcium mobilization from the bones and widespread calcification of organs. This disturbance of Ca metabolism is frequent among old rats but was not seen in animals fed other fats below the age of 500 days. When the linoleate level of the animals fed coconut oil was raised, the death rate was reduced and no gross symptoms of secondary hyperparathyroidism were observed in the animals dying from that time up to the age of at least 500 days.

Therefore, this disturbance of Ca metabolism was somehow related to essential fatty acid deficiency, which may also have played a part in experiments in which adverse effects of hydrogenated coconut oil have been noted. Further work about the properties of bones of animals fed coconut oil is desirable.

No topic related to coconut oil has been given more prominence than its relation to cholesterol metabolism in mammals.

Whether or not human societies consuming large amounts of coconut oil develop more or less arteriosclerosis than other societies and whether any differences are due to the fats consumed can not be answered with any degree of certainty.

However, it has been claimed that the inhabitants of Thailand have a low rate of heart attacks and strokes although coconut oil is their leading dietary fat (40). In a study on two groups of Polynesians, it was found that the group eating 89% of their fat as coconut oil had lower blood pressure values than those eating 7%. Heart attacks were not observed in either group. There were, of course, many other differences between the groups (41). In another study on Polynesians, it was found that Pukapukans consuming large amounts of coconut oil had lower serum cholesterol levels and a lower incidence of arteriosclerosis than the Maoris (and Europeans) who consumed a European type of diet (42).

During the fifties, it was established that the intake by humans of ethyl linoleate or of vegetable oil containing large amounts of linoleate reduced serum cholesterol levels (43, 44); this was true especially when liquid formula diets were used. The subsequent feeding of the same formula diet, but containing coconut oil, raised the level of serum cholesterol although not to the level previous to the intake of the liquid diet (45). At the same time, it was noted that the effect given by coconut oil was less pronounced than had been anticipated from its saturation (46).

Other observations about the effect, in man, of coconut oil on lipid metabolism include the observation that alimentary lipemia is less pronounced after the intake of coconut oil than after consumption of other fats (47, 48) and that coconut oil containing 5% of safflower oil, when added to the standard diet of inmates of a mental institution, reduced serum cholesterol levels (49).

Coconut oil has been used in a vast number of animal studies concerned with the pathogenesis of vascular lesions. The magnitude of this work is such that one can, at best, hope to recognize a few general principles.

The experimental work can roughly be divided into three categories: (a) studies in which coconut oil was fed in the presence of a sufficient supply of essential fatty acids; (b) studies in which hydrogenated coconut oil was supplied without additional fatty acids; (c) studies in which hydrogenated coconut oil was part of essential fatty acid deficient diets to which cholesterol had been added. This latter category constitutes the majority of the reports.

When coconut oil was fed to animals as a constituent of diets made of natural food stuffs, tissue lesions and serum and tissue cholesterol levels hardly differed from those found with many other natural dietary fats. Thus, rabbits maintained on laboratory chow supplemented with hydrogenated coconut oil, hydrogenated vegetable oil, or butter showed no arterial lesions after one year (50). Rabbits maintained on laboratory stock diets and given daily supplements of 5 cc of hydrogenated coconut oil, cottonseed oil, or peanut oil showed no effect on serum cholesterol levels after nine weeks (51). Rhesus monkeys eating a normal diet supplemented with coconut, mustard and sesame oils for nine months showed the lowest aortic cholesterol values in the coconut oil fed animals (52).

When purified, essential fatty acid deficient diets containing hydrogenated coconut oil were used, the results were so strongly influenced by species, age, previous diet, etc. that it is hardly possible to arrive at a common denominator. In the careful study discussed above rabbits developed widespread histopathological changes (including some in the arteries) after one year. In other species it was more difficult to produce histological lesions. Thus, growing chicks given a low fat diet supplemented with coconut, corn, or olive oil showed no differences in plasma and aortic cholesterol (53). Dogs are quite immune to the induction of vascular changes by an essential fatty acid deficient hydrogenated coconut oil diet unless the deficiency state is compounded by the induction of hypothyroidism (54). One strain of pigeons developed coronary lesions on the essential fatty acid deficient diet containing hydrogenated coconut oil (55). When two chimpanzees were given a purified essential fatty acid deficient diet containing hydrogenated coconut oil, one developed vascular lesions exceeding those of the controls (56).

It has been demonstrated again and again that essential fatty acid deficient diets with added cholesterol will eventually induce lesions of the coronary arteries regardless of the origin of the usually hydrogenated fat. This has been found in various birds and is probably true for all mammaliam species. The amounts of cholesterol which the animals are forced to eat are unphysiological; they lead to cholesterol deposits in various organs especially the RES before the coronary arteries are visibly involved. Whether or not results obtained by inducing a deficiency state and an unphysiological dietary stress are relevant to human arteriosclerosis is questionable. The fact that the histological appearance of some of the vascular lesions produced in animals has some resemblance to human arteriosclerotic lesions is probably of no etiological significance because of the limited response potential of arteries to any chronic injuries. However, there is a need for well controlled long term experiments with primates in which the effects of various dietary fats are compared under more physiological conditions.

The medium chain saturated acids of coconut oil have been reconstituted into randomized triglycerides (MCT). Feeding of one of these mixtures containing predominantly C_6 and C_8 acids beneficially modified the effects of various stress conditions under which rats had been kept (4). Related studies have been carried out with coconut oil in deficiency states, in disturbances of carbohydrate metabolism, in infections, and with cancerogenic substances.

It was found that the signs of vitamin B deficiency in rats could be diminished when coconut oil was added to the diet (10). The deficiency signs developed by young calves fed a fat free diet could be prevented by 2% of coconut oil while 4% of lard was necessary to give the same effect (57).

Some of the metabolic effects of MCT simulate those of carbohydrates (5). This metabolic behavior of medium chain fatty acids may explain that feeding of hydrogenated coconut oil to rats tends to increase their liver glycogen (58). Similarly it was noted that, in rats fed laboratory chow and given various fats by stomach tube, liver glycogen was highest in animals fed hydrogenated coconut oil (59). This may

have some bearing on the observation that the diabetogenic action of aloxan in lard-fed rats was counteracted by coconut oil (60). However, disturbances of glucose tolerance and diminished liver glycogen were noted in rabbits fed 25% of saturated fats such as hydrogenated coconut or peanut oils in essential fatty acid deficient diets (61).

Injection of extracts of coconut milk seemed to retard experimental tuberculosis in mice (62); this may be related to the finding that feeding of coconut oil as compared to olive and linseed oil retarded experimental tuberculosis in mice (63).

Numerous studies have been reported about the development of liver tumors in experimental animals fed carcinogenic azo dyes in diets containing various fats. Feeding of coconut oil (and of laurate) was associated with lower tumor incidence (64). In the cited experiment with Charles River rats, chloromatous leukemia, frequently noted in rats fed other fats, was not seen in those given coconut oil or MCT.

Finally it deserves to be mentioned that, in rats, re-establishment of normal spleen after whole body irradiation was facilitated by feeding of coconut oil (65).

It seems permissible to conclude that intake of coconut oil as part of a mixed diet containing enough essential fatty acid has evidently no disadvantages and may have some advantages.

We have pointed out that all dietary fats have their individual properties which cannot be explained by any single chemical characteristic. The example of coconut oil shows how useless and misleading it is from a nutritional standpoint, to classify a particular fat simply as saturated.

There is a great need for long term nutritional studies with coconut oil and other dietary fats especially in primates although their long life span (compared to rodents) makes the execution of such studies by young researchers mandatory. Desirable levels of essential fatty acid in hydrogenated coconut oil will have to be established taking into consideration that not only low levels but also too high ones may be disadvantageous. The nature of the materials responsible for relatively lower essential fatty acid requirements in diets made of natural food stuffs needs attention. Finally, and perhaps most important, more work is necessary to establish why a variety of stress conditions were found to be beneficially influenced by MCT and coconut oil.

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