RELEVANCE OF MIXED CROPPING SYSTEMS

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When tree crops are grown as sole crop under close spacing, each tree competes with the neighbouring one for the essential elements needed for the growth and production. They compete for moisture and nutrients because they tap them from the same soil depth for many years. They are also observed to compete for energy from sunlight from the time of their attaining physical maturity after a few years from planting, due to canopy overlapping. On the other hand, when the mono-culture trees is followed with wide spacing for avoiding these competitions, they fail to adequately utilize the available soil as well as air spaces.

When we consider the monocropping of trees from the labour utilization point of view again we find that barring the establishment stage, the labour absorption is low and seasonal for the remaining life span. For the regions with surplus labour, this type of systems may not be desirable.

Another economic disadvantage of mono tree crops is that the investment in establishing the crop till its bearing age becomes sizable which a smallholder may not be able to afford.

Plants are generally grouped a herbs, shrubs and trees depending upon their stature. In each group, there are wide variations in the rooting patterns and canopy structures, which change with age of plants as well as ecological distribution. The variations in the canopy shape and size of plants reflects on both the incident and intercepted radiation which are important components of growth. Based on their response to light, plants are classified further as photosensitive and photoinsensitive. While some are sun-loving plants, others thrive well either under partial shade or full shade.

4% safflower oil, which supplied 8% of calories as linoleic acid. The second group of dogs was "completely protected from the atherogenic process" with a concomitant loweing of the serum total cholesterol from a mean of 1294 mg/100 ml (all fat as HCNO) to a mean of 330 mg/100 ml. The authors ascribed the atherogenicity of the first diet to EFAD and to the presence of coconut oil.

Another canine model study revealed little information on coconut oil compared to other dietary oils (40). The design examined 10, 20 and 40% of calories as coconut oil for brief and therefore inadequate two week feeding periods. Essentially, the comparison was between high fat and low fat diets.

Coconut oil fed to miniature pigs produced a serum total cholesterol mean half as large as the mean from pigs fed tallow, even though the coconut oil group also received twice as much hog bile (35). More severe atherosclerotic lesions were observed with tallow feeding than with coconut oil feeding. Both diets were supplemented with menhaden oil to assess the protective effect of the omega-3 polyunsaturated fatty acids.

Although the tallow fed group received twice as much menhaden oil supplementation (6% by weight) as the coconut oil fed group, menhaden oil had no discernible protective effects on aortic or coronary atherosclerosis in the tallow fed pigs. Coconut oil fed pigs, receiving half as much menhaden oil, showed less severe lesions than controls without the supplement. Coconut oil did not block the beneficial effects of feeding marine oil and therefore can be regarded as neutral in terms of atherogenecity.

Monkeys have also been used as a model of human atherogenesis. An early study published in 1962 by Wissler et al. using twelve Cebus monkeys, compared coconut oil, butterfat, and corn oil as 48% of calories with a 0.5% by weight cholesterol supplement (41). Each dietary group contained only four animals. Although the investigators prudently employed

principles of 'pair' feeding, the coconut oil group suffered marked weight loss (from 1450 gm at week 0 to 1200 gm at week 45), while the other groups did not; the general appearance of the monkeys fed coconut oil was not remarked upon by the authors.

The authors speculated that the relatively "hard" coconut oil was incompletely absorbed by the gastrointestinal tract. No definition of "hard" was included in the text, but the term probably refers to hydrogenation.

Since no chronic diarrhea or steatorrhea was observed, increased thermogenesis from the medium chain triglycerides in coconut oil may explain the weight loss. The animals may have also lost weight due to a lack of essential fatty acids over an extended period of time. Regardless of the cause, weight loss in one group of animals precludes valid comparison with other groups not experiencing weight change.

In the same study, serum lipids were measured. All three fats with supplemental cholesterol caused increases in serum cholesterol over time. No statistical analyses were performed "trends" were noted. The initial serum cholesterol values given for the three groups recorded the coconut oil mean as 50% larger than the means for the corn oil and butterfat groups. Either the study did not report true baselines (called "week 0" by the authors), or the baseline given for the coconut oil group is unreasonably disparate from the other baselines. The defects in the study design prevent the careful reader from making any conclusions from the data on coconut oil's atherogenicity.

EPIDEMIOLOGY

Epidemiology does not conclusively prove coconut oil's nutritional safety, yet a brief review of the reports raises a valid question: If coconut oil is truly dramatically hypercholesterolemic, why are

hypercholesterolemia and coronary heart disease rare in populations consuming 35-50% of calories as fat largely derived from coconut?

Prior et al. studied two populations on Polynesian atolls, finding no harmful effects from the high saturated fat intake practiced by both groups (42). The two groups derived 63% and 34% respectively of their energy requirements from coconut. The observed serum total cholesterol values were lower than the values predicted from the Key's equation by 68, 75, 80, and 82mg/ 100 ml in the four groups examined. The predicted values were approximately 40% higher than actual measured values. The prevalence of vascular disease was low for both groups compared to populations in New Zealand, highlighting highlights the need to distinguish between medium and long chain saturated fats in terms of hypercholesterolemia etiology.

In an earlier epidemiological report sponsored by the Medical Research Council of New Zealand, Prior compared two populations of Polynesians of the same broad ethnic type (Pukapukans and Rarotongans) from the Cook Islands (43). Pukapuka was described as "one of the most isolated atolls in the Pacific", while Rarotonga was considered much more westernized. The average diets differed in total calories, percent of calories from fat, and type of fat.

Pukapukans ate 300 kcal/day less than Rarotongans, and derived 35% of calories from fat versus 27% among the Rarotongas. Seventy five percent of the fat in the Pukapukan diet was coconut oil compared to twenty five percent in the Rarotongan diet. Even though Pukapukans ate more coconut oil and fat overall, their serum cholesterol levels were considerably lower than those measured in Rarotongans. The range of cholesterol values for all Pukapukans was from 155 mg/100 ml to 195 mg/ 100 ml. Ethnically similar groups were compared to control for any strongly confounding genetic factors.

Therefore, coconut oil appears, at worst, to be neutral in terms of atherogenesis. Humans consuming large of amounts of coconut oil in a varied diet do not demonstrate hypercholesterolemia. Yet the same individuals develop hypercholesterolemia when subjected to migrational changes in diet and environment. Polynesians are not genetically immune to hypercholesterolemia (44).

HUMAN STUDIES

As other reviewers have noted, very little corroboration of the animal data has been found in clinical studies of dietary coconut oil (26). Some of the earliest clinical trials of dietary lipids were of the rather non-scientific case study variety. Kinsell et al. reported the effects of supplemental coconut oil feeding in two patients (45). One patient suffered xanthelasma and diabetes, while the other has sustained two myocardial infarctions. The formula feeding protocol used by Kinsell is considered by many investigators as non-physiological. No statistical analysis was performed. The synthetic mixtures containing various fats were fed in a non-random order.

Other clinical studies have shown that removal from an ***ad libitum*** diet to a synthetic formula type diet causes a dramatic and prolonged decrease in serum cholesterol levels independent of the fat component (46, 47). Thus, the pattern of serum cholesterol values observed cannot be entirely ascribed to diet without eliminating the effects of removal from a self-selected diet.

Grande et al. presented the data from their crossover trial of diets supplemented with 29 gm/day stearic acid (C18:0). The results clearly showed that the second diet fed produced lower serum cholesterol levels regardless of the fatty acid content (48). The authors somehow corrected for the time trend to obtain a statistically significant difference (7.7 3.5 mg/100 ml) in the serum cholesterol values observed

with each diet; the time trend correction figure was 19 mg/100 ml. Concluding that lauric acid is more hypercholesterolemic by 8 mg/100 ml than stearic acid, as the authors assert, seems to be a biased and anticipated analysis of the data.

Malmros and Wigand ran clinical trials of different dietary fats as 40% of calories (31). The fat was added as salad-dressing, mayonnaise, and oil for frying, thus enabling the investigators to examine the fats as part of a physiologic diet. The authors noted the markedly depressing effect of corn oil and safflower-seed oil on serum total cholesterol. "Coconut fat (hydrogenated non-hydrogenated) has had no such effect". coconut oil was not found to be hypercholesterolmic or labeled as such. Coconut oil did not alter the baseline serum cholesterol values. When fed in a mixture with a third on the fat calories as corn oil, coconut oil did not block the serum cholesterol depressing action of the corn oil. Unfortunately, the absence of statistical analysis of the data weakens any conclusions made in Malmros and Wigand's otherwise impressive article.

Ahrens et al. employed an oral feeding technique of liquid formulas to assess more precisely the effect of different dietary fats on serum cholesterol (30). Again, the diets might not have been physiologically relevant. From the data, the investigators elaborated a schematic representation of their hypothesis in the form of a three-dimensional plot with columns representing serum cholesterol levels protruding from the surface. The intricacies of the diagram are complex and confusing, and, therefore, the salient features are easily overlooked.

Coconut oil was tested in only three subjects who had serum cholesterol values of 213,367, and 186 mg/100 ml. The subject with a cholesterol of 367 mg/ 100 ml had serum cholesterol levels of 450 mg/ 100 ml on his regular diet. Baseline serum cholesterol levels were not given for the other subjects. All serum cholesterol values were compared to values obtained with a reference corn oil diet. Coconut oil

is less effective at depressing serum cholesterol levels than corn oil, but the data do not indicate that coconut oil increases cholesterol in the blood.

A previously mentioned, Keys et al. developed a regression equation predicting changes in serum total cholesterol from changes in the percentage of calories as polyunsaturated and saturated fats (5). Ordinary foods were used in physiologic diets with 40% of calories as the experimental fats. The subjects were typically on the test diets for two to four weeks. The experimental periods were terminated when the cholesterol levels were subjectively deemed as steady-state.

The authors recognized and remarked upon the inaccuracy of the equation in regard to corn oil and hydrogenated coconut oil. The mean serum cholesterol value for the twelve, otherwise healthy schizophrenic men on coconut oil was 224.3 8.4 mg/100 ml. A Duncan's multiple range test to differentiate between serum cholesterol means was not performed. Means from trials of other oils were often higher than or very close to the coconut oil group mean: house diet 232 and 225 mg/100 ml, butterfat 241, 235, and 232 mg/100 ml.

Frantz and Carey selected twelve healthy male subjects with "high normal levels of serum cholesterol" from a hundred volunteers (49). Half of the subjects received an ounce of corn oil before each of three ad libitum American type meals a day, while the other six took three ounces of hydrogenated coconut oil a day. The fat supplements comprised an additional 810 kcal/day, yet body weightds remained constant. Liver cholesterol levels were obtained via biopsy, and blood was drawn for cholesterol analysis.

After one month of dietary fat supplementation, the mean serum cholesterol value for the mean eating hydrogenated coconut oil was not significantly different from their initial mean value. Dietary coconut oil appears to be neutral in terms of any effect on serum cholesterol. Liver cholesterol levels

also did not change significantly in the men fed coconut oil. Corn oil, however, caused a decrease in serum and liver cholesterol (p 0.5).

Hashim et al. fed 42% of a formula diet as fat to ten hypercholesterolemic patients (8). Within the metabolic ward, the subjects received either safflower oil or an equivalent amount of a 50% safflower oil and 50% coconut oil mixture in a crossover design. The coconut oil/safflower oil mixture and safflower oil alone each caused decrease in serum cholesterol of the same magnitude from the initial central values. In fact, the mixture produced a slightly lower serum cholesterol mean, 256 mg/100 ml versus 268 mg/100 ml with safflower oil alone, but the difference was not significant. Thus, coconut oil is not hypercholesterolemic when consumed as part of a mixed fat diet typical of human dietary patterns.

The Austrian investigators Halden and Lieb fed 80 grams of coconut oil as the major dietary fat with 5% of the experimental fat as sunflower oil and another 5% as olive oil (50). The fats were used as cooking oils and spreads in a normal physiological diet in twelve hypercholesterolemic but otherwise healty volunteers. The authors reported an initial range of serum cholesterol from 170 to 370 mg/100 ml.

The coconut oil diet, with a tenth of fat calories as sunflower and olive oils, caused the range to decrease after ten weeks to 140-240 mg/100 ml. Removal from the diet caused an increase in serum cholesterol to 1709-320 mg/100 ml. Resumption of the diet again decreased the serum cholesterol to a range of 150-260 mg/100 ml. A subsequent switch to 100% coconut oil caused a slight increase in serum cholesterol to 170-279 mg/100 ml, but the range remained well below the baseline range.

Halden's study was primarily descriptive and without analytical statistics, yet a significant amount of dietary coconut oil was clearly shown not to block the reduction of serum cholesterol probably caused by other features of the dietary management.

In 1967, Bierenbaum et al. reported the result of an impressive five year study of the dietary management of 100 young men with coronary disease evidence by electrocardiographically confirmed myocardial infarctions (7). All the subjects were placed on a strictly reduced fat diet with 28% of calories as various fat mixtures. Diet 1 was supplemented with one ounce (slightly less than half of the total fat) of a 50/50 mixture of corn and safflower oils; Diet 2 contains an ounce of a 50/50 mixture of coconut and peanut oils. After one year and five years, both reduced fat diet groups experienced a significant reduction in serum cholesterol while non-dietary-managed controls did not.

The reduction in serum cholesterol with Diet 1 containing corn oil was not significantly different from the reduction observed with Diet 2 containing coconut oil. The polyunsaturated to saturated fat ratio was 3:1 in Diet 1 and 1:3 in Diet 2. The Keys equation does not accurately predict the data. Coconut oil caused no adverse effects on the serum cholesterol values, atherosclerotic status, or the mortality of the fifty men consuming the supplement.

Some evidence suggests that the quality and quantity of fatty acids alter the effects of dietary cholesterol on plasma cholesterol (51). A study by Schonfeld demonstrated that a diet high in polyunsaturated fatty acids did not adversely affect serum cholesterol when dietary cholesterol was increased (52). The type of fatty acids appears to exert the most consistent control over the plasma lipid levels (53); a dietary change from saturated fats to polyunsaturated fat will decrease serum cholesterol levels in most patients. However, the degree of this lowering effect varies from individual to individual. The effect of dietary fatty acids on serum cholesterol depends not only on whether or not the acids are saturated on unsaturated, but also on their chain length, as in coconut oil, and quantity ingested.

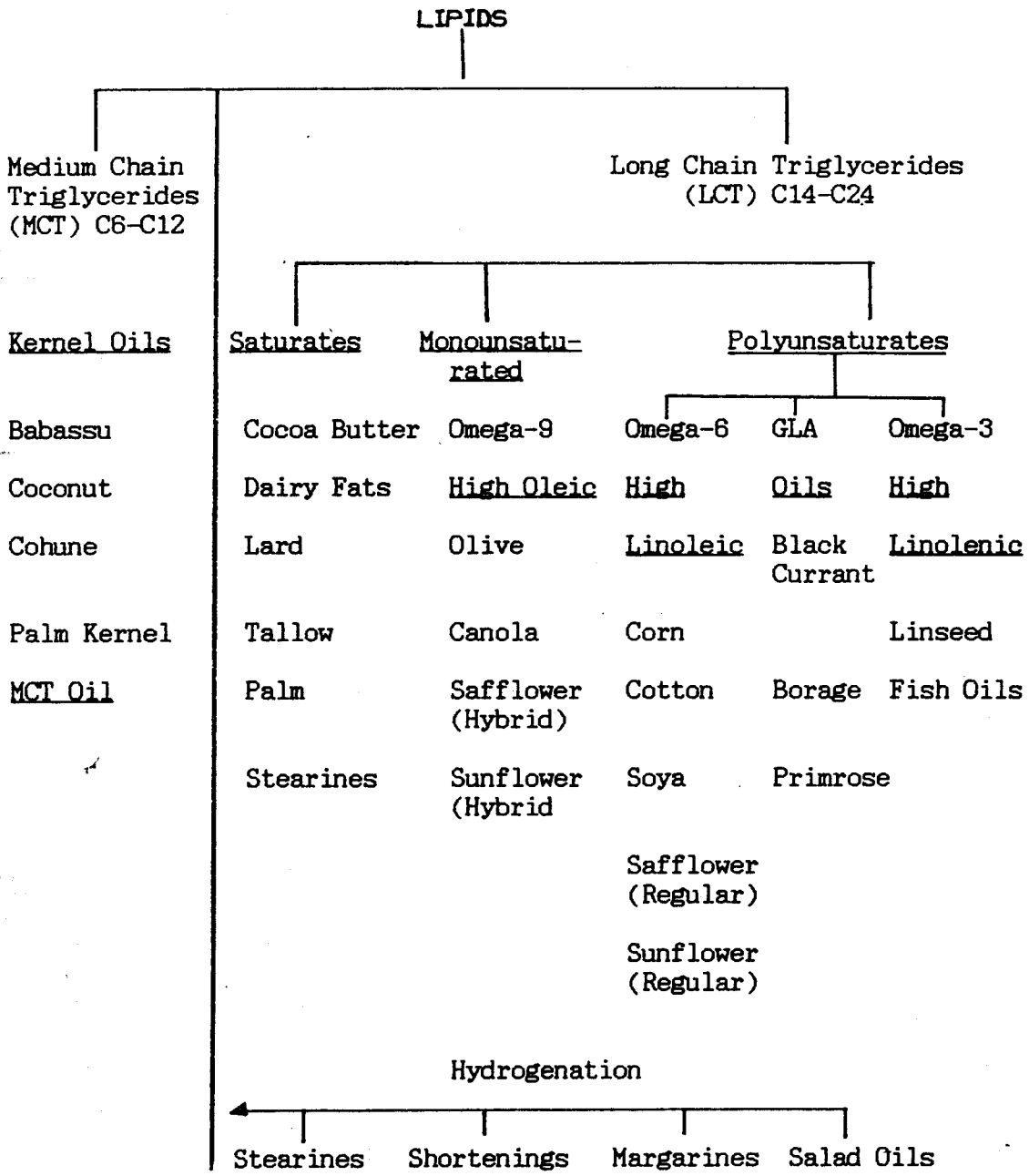
TABLE 1
TYPICAL LCT OILS

	Lauric	Myristic	Palmitic	Palmitoleic	Margaric	Stearic	Oleic	Linoleic	Arachidic	Gadoleic	Eicosadienoic	Behenic	Lignoceric	
Corn Oil			12.2	0.1		2.2	27.5	57.0	0.9	0.1				
Peanut Oil		0.1	11.6	0.2	0.1	3.1	46.5	31.4		1.5	1.4	0.1	3.0	1.0
Safflower Oil		0.1	6.5			2.4	13.1	77.7		0.2				
Soybean Oil		0.1	11.0	1.0		4.0	23.4	53.2	7.8	0.3			0.1	
Sunflower Seed Oil	0.5	0.2	6.8	0.1		4.7	18.6	68.2	0.5	0.4				

TABLE 2
TYPICAL LAURIC FTS AND OILS

	Caproic	Caprylic	Capric	Undeca-noic	Lauric	Myristic	Palmitic	Stearic	Oleic	Linoleic	Arachidic
Babassu	0.4	5.3	5.9		44.2	15.8	8.6	2.9	15.1	1.7	0.1
Coconut Oil	0.5	8.0	6.4		48.5	17.6	8.4	2.5	6.5	1.5	0.1
Cohune Oil	0.3	8.7	7.2	0.1	47.3	16.2	7.7	3.2	8.3	1.0	
Palm Ker-nel Oil	0.3	3.9	4.0		49.6	16.0	8.0	2.4	13.7	2.0	0.1
Tacum Oil	0.2	2.9	2.3		51.8	22.0	6.8	2.3	9.3	2.4	

TABLE 3


LIPIDS
Medium Chain Triglycerides (MCT) C6-C12
Long Chain Triglycerides (LCT) C14-C24
Kernel Oils
Babassu
Coconut
Cohune
Palm Kernel
MCT Oil
Saturates
Cocoa Butter
Dairy Fats
Lard
Tallow
Palm
Stearines
Monounsaturated
Omega-9
High Oleic
Olive
Canola
Safflower (Hybrid)
Sunflower (Hybrid
Polyunsaturates
Omega-6
High
Linoleic
Corn
Cotton
Soya
Safflower (Regular)
Sunflower (Regular)
GLA
Oils
Black Currant
Borage
Primrose
Omega-3
High
Linolenic
Linseed
Fish Oils
Hydrogenation
Stearines
Shortenings
Margarines
Salad Oils

CONCLUSION

The foregoing review of the literature on dietary coconut oil should not be misconstrued as an attempt to discredit the cited investigators or their work. In a sharp rebuttal to Reiser's review of dietary saturated fats, Keys et al. counter some of Reiser's criticism of the clinical studies of dietary lipids (54). Some of the most heated controversy easily degenerates into trivial argument and bickering. Some of Reiser's arguments are adroitly refuted by Keys et al., and yet the controversy remains. Any conclusions from the research on dietary coconut oil's effect on serum cholesterol are made without complete confidence, since too many incongruencies exist, and doubt is not dispelled by any of the reviews. Clearly more studies, free from the inadequacies noted in this review and others, are required. McNamara has focused on some of the inconsistencies regarding the effects of dietary cholesterol and quality of fat on serum cholesterol in humans (53, 55). The data suggest that individuals vary in response to dietary cholesterol. Therefore, dietary intervention should be tailored toward the individual's specific need. Animal studies can also be misleading because of the models' dissimilarly from the human. Extrapolation from the animal data is therefore not always prudent. Grundy et al. warn, "it is important to note that hypercholesterolemia induced by diet in most species is associated with unusual types of lipoproteins... which normally do not occur in significant quantities in man" (56).

Recently, Michael S. Brown elaborated on the role of lipoproteins and their effects on the atherosclerotic process. A suppression of the HDL receptors in the liver may result from a diet high in cholesterol and fat. The decrease in receptor sites allows the LDL cholesterol to accumulate in the blood, which may accelerate atherogenesis. Also, plasma concentration of lipoprotein (a) has been correlated with atherosclerosis (57). This link between certain serum lipids and apolipoprotein levels in indisputable. Clearly, further investigation focusing

on these intricate chemical processes and its relationship to atherogenesis needs to be undertaken.

Human studies have suffered from an inadequate number of subjects compounded by subjective or descriptive interpretation of the data. A few carefully designed with the proper statistical analyses might resolve much of the long-standing conflict. Utilization of improved technology has led to a more efficient way to study cholesterol homeostasis in the free-living population.

McNamara et al. (53) used a combination of three procedures to evaluate the effect of dietary fat and cholesterol on serum cholesterol: (1) refined dietary cholesterol measurement; (2) quantification of the dietary cholesterol absorption by the isotope ratio method; and (3) analysis of changes in endogenous cholesterol rates. These methods may help alleviate the clinical problems relevant to studying dietary lipids.

Humans tend to eat mixtures of fats, yet very rarely have dietary lipids been examined as mixtures. Early animal experiments are of limited value because these animals developed essential fatty acid deficiency. This would have been avoided by feeding mixtures of fats, as the interaction of different fatty acids might not be simply additive. In the few trials of coconut oil as part of a mixed fat diet, a hypercholesterolemic response was not observed.

The diet-heart hypothesis has slowly gained credence even though the debate continues. Progressively more convincing data have emerged. The AHA strongly recommends a reduction of fat calories from the current average of 40% to 30%, with a specific reduction in saturated fats (58). Coconut oil is currently included in the AHA's list of deleterious saturated fats.

As previously noted, coconut oil is an unusual edible oil. The literature does not support lumping coconut oil with palm oil, a saturated fat with 43% palmitic acid (C16:0) and 39% oleic acid (C18:1). The

loose categorization of dietary lipids, as saturated, polyunsaturated or monounsaturated, promulgated by the scientific community has led to even greater inaccuracy by the media.

A recent editorial in the New York Times refers to coconut oil, palm oil, and palm kernel oil as "the cheaper, artery-clogging oils from Malaysia and Indonesia" (59). The authors's bias can be detected in phrases like ".... American farmers stand to gain from promotion of products made with domestically produced, unsaturated corn and soybean oil".

The AHA recommends an intake of polyunsaturated fats with an upper limit of 10% of calories. Excessive amounts of polyunsaturated fat in the diet have been shown to have co-carcinogenic potential in laboratory animals (60, 61). Also, high polyunsaturated fat diets may increase the incidence of gallstones (62). This new recommendation would limit omega-6 PUFA to 5% and allow for the remaining 5% of PUFA to be provided by omega-3. In the same 1982 AHA committee report cited earlier, Grundy et al. write, "At present, no inexpensive sources of natural monounsaturated fats similar to olive oil are readily available in the U.S." (63), thus recognizing the unlikelihood of Americans replacing a large percentage of saturated fat with monounsaturated fat.

Advanced clinical and laboratory lipid research technology needs to be directed at identifying safe, available dietary fats. Since the American public is disinclined to reduce readily the amount of highly palatable fat in the diet, nutrition research must expose in detail which fats are health promoting and safe.

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