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It is indeed a privilege to share this portion of the conference program with the distinguished Dr. Fredericks, but I think you will soon find that we hold little in common other than the same publisher—Grosset and Dunlap; our views on nutrition are poles apart.

Last year at the International Academy of Metabology Florida meeting, the banquet speaker, Dr. Emanuel Cheraskin, made a statement that deserves to be pondered: he said there is not a proper diet for arthritis, another completely different one for angina, and so on for other diseases. He said there is one optimal diet for man.

I am in complete accord; and it is my position that key characteristics of the optimal diet are the very low fat content and main dependence on food as grown—complex carbohydrates. Simple carbohydrates, as sugar, molasses and honey, are, of course, to be avoided.

An optimal diet should permit man to be free of the ravages of cardiovascular degenerative diseases, including angina and hypertension, and diabetes and arthritis, as one of its major attributes.

The low incidence of atherosclerosis in populations on low fat diets has been widely observed by many investigators. The coronary heart disease rate of Bantus in Africa who live on a 10% fat diet is almost zero. Hannah[1] reports that at the Nkana Nune Hospital, which has 300 beds for Africans and a high autopsy rate, no deaths were ascribed to coronary heart disease. In autopsies performed on 42 Bantus and 22 Europeans who died suddenly for any reason, only one Bantu had atherosclerosis; whereas 100% of the Europeans, even a youth of 15, had extensive artery damage. 35 of the Bantus lacked even a trace of fatty streak on the intima—the earliest clinical sign of artery damage.

Whyte[2] observed natives of New Guinea whose diet contained 10% fat and only 7% protein. Autopsies performed on 600 natives revealed only one case of death attributable to coronary heart disease. Blood pressure in New Guinea natives was found not only not to rise with age, but diastolic pressure dropped about 10 mm. when in their 60's.

Leaf[3] studied a population in Ecuador with an unusual number of aged people. Of the 800 villagers, a number were over 100 years old and one was 121, as authenticated by Catholic Church records. Cardiovascular disease was found to be virtually non-existent. The diet was mainly complex carbohydrate: corn, brown rice, beans, various other vegetables and fruits and a once weekly portion of animal protein.

Keys[4] has studied over 25 populations on a low fat diet: without exception, their heart disease rate was low.

Immunity to coronary heart disease lasts only so long as a low fat diet is maintained. Thus, in a study of Japanese who have changed their environments from their homeland to Hawaii and then to mainland United States, where different fat contents were consumed, it was found that as the fat intake increased, so did coronary heart disease death rates.

I would like to tell you about a small controlled study now taking place in Southern California where a diet such as is consumed by native atherosclerosis-free populations is being replicated with astonishing results. For this six month study, now almost half over, we selected 38 men with an average age of 60. Artery closure in all was so bad that most could walk only a few minutes on a 1½ MPH treadmill before calf pain stopped them. 80% of the men have had arterial reconstruction work and in most we found coexisting conditions of hypertension, diabetes, angina, cerebral ischemia and so on.

The extensive baseline testing done on all of the men has included an angiogram of all arteries from the aorta to the toes. The angiograms revealed very advanced atherosclerosis with occlusions in the larger arteries of each patient, even after their arterial bypasses and enarterectomies. Cholesterol levels for most were from 200 to 300 mg.%.

These normal western cholesterol levels contrast with the low cholesterol levels universal with atherosclerosis-free native populations. New Guinea natives have cholesterol levels around 100 mg.%, whether young or of advanced age; cholesterol levels of the African Bantus are 90-120 mg.%; the average cholesterol level of the Ecuadorian population studied by Leaf was 150 mg.% for a mature adult; and Keys found low blood cholesterol levels in all of the 25 atherosclerosis-free populations he studied.

In our study, half of the 38 men, the controls, have been instructed to walk daily and to try to cut out their smoking. They are having monthly examinations and are being given the best medical care—but their diet remains unchanged. The other half—the experimental group, for which I have dietary responsibility, have also been urged to walk daily and to cut out smoking, but, in addition, they are having all their meals at the study headquarters seven days weekly. Their diet simulates that of atherosclerosis-free populations in the world in that it is 80% carbohydrate, this being made up entirely of natural unrefined foods, so that no supplements are required.

We are getting some exciting results. In the first two weeks, blood cholesterol dropped an average of 30%. One triglyceride dropped from 360 to 85 mg.%. Our goal is to maintain cholesterol levels below 130 mg.% and triglycerides below 75 mg.%. At these levels, there is much evidence that atherosclerosis reverses.

After five weeks, treadmill performances increased 800%.

Even more gratifying were the drops in blood pressure in the nine hypertensives in the group. After a month, all nine—all of whom had been drug-treated for hypertension, were normotensive without drugs. One of them had been on

antihypertensive drugs for 20 years. In the fall of '73 he was taken off drugs when in the hospital for testing and his blood pressure rose to 210/110, staying there for ten days. He was put back on drug therapy and remained there—until his blood lipids were lowered recently on this experimental diet.

Repeat blood flow tests have demonstrated improvements. Repeat angiograms will be done in July at the end of the study and it is hoped that these will show that the regimen has caused the arterial damage to regress through plaque reversal.

The effect of the diet on the diabetics in our study's experimental group has been dramatic: the four diabetics were off their drugs in six weeks. This includes two who had been on insulin for 10 to 15 years. One of the two was taking 80 units daily for the last year and a half.

How can this fast reversal of diabetes be explained? Two studies in the literature clarify the diet relationships involved in the etiology of diabetes. Sweeney[5] put young medical students on different dietary regimens for two days. One group's diet was high in protein; another's was high in fat; a third group received no food; and a fourth group was fed a high carbohydrate diet. On the glucose tolerance test taken after the two day period, the high protein group tested borderline diabetic and the high fat and starvation groups were both quite diabetic with their two hour post glucose blood values exceeding 170 mg.%. Only one group tested normal: those on the high carbohydrate diet.

Anderson's study[6] required a longer period for ingested fat to raise the lipid level high enough to produce diabetes—two weeks instead of two days. Two diets utilizing corn oil as the fat and sucrose as the carbohydrate in varying percentages were used with 20 year old normal men. After two weeks, the group on the 80% sucrose and only 5% fat diet tested normal on a glucose tolerance test; but the second group, on a diet of 65% fat and 20% sucrose tested diabetic, with a glucose tolerance test two hour reading of 184 mg.%. Those on the low 5% fat diet continued this regimen for an additional seven weeks, and their glucose tolerance test at the end of this period was still normal. This result shows the efficacy of a low 5% fat diet in maintaining a normal glucose tolerance test response—even on a diet in which 80% of the total calories consumed were in the form of table sugar, sucrose. The heavy sugar intake did, however, reflect in steadily increasing triglycerides.

If a high fat diet can produce diabetes, can a low fat diet reverse it? Rabinowitch[7] used a 21% fat diet on thousands of diabetics in his Montreal hospital. In a five year controlled test 24% of insulin dependent diabetics on the low fat diet no longer required insulin, and those still on insulin had their dose reduced 58%.

Singh[8] used a diet of 12% fat and was even more successful. Starting with 80 insulin dependent diabetics, in less than five months, 72% of the diabetics were off insulin and the balance were on substantially lower dosages. Several were followed for five years, remaining unchanged.

With this historical background, our success in reversing diabetes with a low fat diet is no surprise. I believe our final angiographs will prove that a low fat diet can also reverse atherosclerosis. This confidence is based on primate and human

studies that show that cholesterol level is the best predictor of artery damage in atherosclerosis, and that when cholesterol level is lowered sufficiently, plaque regression occurs in primates.

The Framingham Study[9] which started in 1948 with 5200 people is relied upon as one of the more respected predictors of risk factors. Data from this study indicates that an individual with a cholesterol level above 260 mg.% had 400% more cardiovascular events than one with a level below 220 mg.%.

Keys' Minnesota study, [10] started in 1947, confirms the Framingham results. Using the amount of heart damage observed with maximum cholesterol levels of 200 mg.% as a standard of comparison, in both studies cholesterol levels of 220 to 239 mg.% were found to produce 250% more heart disease; from 240 to 259 mg.%, there was 400% more heart disease; and above 260 mg.%, there was 560% more coronary heart disease than was found in patients with levels below 200 mg.%. The risk was found to be directly proportional to the cholesterol level.

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All major population studies provide indirect evidence of this correlation between cholesterol level and heart deaths, but now we also have direct angiographic proof in the form of x-rays of the arteries. Welch[11] did angiographic studies of 723 men under 40 years. Half had two main coronary arteries more than half closed; the relationships of the amount of artery closure to cholesterol level were found to be almost identical to those in Keys' Minnesota study.

Welch observed that with cholesterol levels of 250 mg.%, 300% more men had closed arteries than those with levels under 200 mg.%. Almost 90% of the men with levels over 300 mg.% had some closed arteries, and these men were all under 40 years old. The youngest man, 17 years old, with no symptoms, had excellent blood levels by U.S. standards: cholesterol 184 and triglycerides 122. Yet the angiogram demonstrated a total occlusion of the anterior descending coronary artery.

The angiographic work of Page[12], from the famed Cleveland Clinic, made possible the construction of a table for prediction of artery closure based on only 3 factors: age, cholesterol level and triglyceride level. For example, at age 28, cholesterol level 140 mg.% and triglycerides 60 mg.%, only half of 1% would have artery closure; but if the cholesterol level was 360 and the triglyceride were 540, 73% would have artery closure—even at age 28!

To check the reliability of his predictions, Page did a double blind test on 60 new patients. Based on the three factors: age, cholesterol and triglyceride levels, he predicted which patients would demonstrate closed arteries in angiography. Independent groups did the angiograms and tabulated the results to eliminate any

human bias. Page's track record for predictions was nearly perfect: 59 of the 60 predictions were correct—a 98% degree of accuracy!

The indirect evidence from the population studies confirmed by the direct evidence of angiography paints a grim prognostic picture for most adults in the western world: Cholesterol levels over 140 mg.% and triglyceride levels over 75 mg. % are the blood environment initiating atherosclerotic damage.

Primate cholesterol levels are very similar to those in man when primates are placed on comparable diets. Armstrong[13] evaluated low fat, high fat and high unsaturated fat diets as to their potential for producing or regressing the atherosclerotic state, using 40 monkeys as subjects. On the high fat diet, which was also high in cholesterol and contained an amount of fat common in American diets—41%, the monkeys developed plaques that closed the arteries 58%. The monkeys were then placed on two cholesterol-free regression diets: one with only 4% fat, and the other with 40% fat, but of the unsaturated kind. Regression of plaques on the 4% fat diet permitted 400% more blood flow; but the 40% polyunsaturated fat diet produced 65% less regression, and plaque analysis showed 50% more cholesterol esters.

Rhesus monkeys eating an average American diet develop average American plaques as was demonstrated by Wissler[14] using a diet based on the 25 favorite American foods in proportions judged from food consumption tables. Other monkeys ate a second diet, the prudent diet, which omitted eggs, beef and pork fat, liver, cheese, butter and bacon. After two years, when the monkeys on both diets were sacrificed, it was found that those on the 25-favorite-foods diet, equivalent to 36% fat American meals, had average cholesterol levels of 383 mg.%; while those on the prudent diet had average cholesterol levels of 199 mg.%. On the 25-favorite-foods diet, almost half the monkeys had plaques covering more than 90% of the inner surface of the aorta; whereas the same amount of monkeys on the prudent diet had 16% of the aorta covered with plaques.

The prudent diet resulting in a cholesterol level of 199 mg.%, which is very normal by current standards, looks good, until one checks out Armstrong's controls[13] on a cholesterol-free low-fat diet. The 135 mg.% cholesterol level of these monkeys at the end of the five year period produced no plaques.

Two studies suggesting plaque reversal in humans by simple means should be cited. Morrison,[15] using proven coronary infarct patients in a 12 year study, put closely matched groups of 50 each on two different diets. The controls stayed on their normal western diet and the experimental group was recommended to restrict fat intake to 15% of total calories and cholesterol to 100 mg. daily. At the end of the 12 year period, 38% of the lower fat diet group were still alive, whereas the entire control group had died.

Lyons[16] started 280 patients with previous infarcts on a similar program. 155 were on a low fat low cholesterol diet and the control group of 125 continued on their average American diet. Four years later, those on the high fat diet had suffered 400% more deaths and 400% as many new coronary infarcts as those on the low fat diet.

It might be argued that even if a low fat diet prevents and reverses diabetes and heart disease, a diet which depends for 80% of its calories on natural carbohydrates would destroy health and vigor because vegetable proteins are incomplete.

This erroneous concept was fostered by the work of Osborne and Mendel[17] in the early 1900's. Working with rats, they concluded that plant proteins have a low PER (protein efficiency ratio) and hence have a much lower biological value than animal proteins. Their work and later work by others also demonstrating sub-optimal growth in rats on vegetable protein diets led to the assumption that this would also be true with humans. The PER rating of proteins was based on their efficiency with rats.

But the nutritional needs of rats and humans are not analogous. If they were, then human milk should not be fed to human infants because rats do poorly on human milk, it being only 1.2% protein compared to the 12% protein of rat's milk.

When humans were used as subjects by later investigators, two important findings were made: 1. Vegetable proteins are as adequate as animal proteins for human diets; 2. The amount of protein required for humans has been overstated. This throws new light on the world's supposed protein shortage, which, in fact, is really a calorie shortage. If calorie intake is insufficient for energy needs, the protein in the diet will be used for calories first and protein second. With a calorie deficit, no protein, regardless of its biological value, will permit normal growth in children or a positive nitrogen balance in adults.

These later investigations with humans into the adequacy of vegetable proteins used subjects of all ages. Knapp's subjects[18] were infants from 5 to 14 months admitted for treatment for diarrhea to his metabolic hospital in Corpus Christi, Texas. After the diarrhea was controlled, he fed them various isocaloric diets at two grams of protein per kg. of weight per day for three months. The reference protein, milk, was compared to: 1. rice and cottonseed; 2. rice plus peanut; and 3. wheat. It was found that there was no difference in growth rates or nitrogen retention in balance studies. In fact, the percentage of nitrogen excreted as urea was lower in all babies fed vegetable proteins compared with those fed milk.

Reddy[19] studied children aged 2 to 5 years old in India. He was concerned with the use of wheat as their principal protein because compared to egg it is low in lysine and in animal studies the addition of lysine to wheat diets significantly increases animal growth. Two diets were tested: one with unfortified wheat, the other wheat plus lysine. Nitrogen balance studies on the children showed no difference in results of the two diets so long as adequate calories were given.

In rice as with wheat diets, in rat nitrogen balance studies, there is lysine deficiency, and when some chicken was substituted for the rice, the PER (protein efficiency ratio) became considerably improved. But again, what is true for rats is not true for humans. Lee[20] at Purdue University put male students aged 20 to 27 in three different diets for 59 day periods for nitrogen balance studies. One diet relied on rice alone to supply all the protein; another used 85% rice and 15% chicken; and a third used 70% rice and 30% chicken. The total protein for each of the three diets was 6.5% of total calories, far less than considered essential by our present standards.

The 30% chicken diet demonstrated a positive nitrogen balance of 1.04 gms., or 13%. 15% chicken wasn't as good—2% less, making an 11% positive nitrogen balance. The 100% rice diet produced the highest nitrogen retention—13.5%, even though the rice used in the study was white rice, completely milled and unenriched. Yet in Kik's[21] study with rats using rice, even a 1% chicken supplement greatly improved their growth rate. Protein requirement studies with rats are not necessarily valid for humans.

College women aged 17 to 31 years were studied by Fisher[22] at Rutgers University. Only 5 gms. of nitrogen or 31 gms. of protein per day was necessary to keep them in nitrogen balance. The amino acid pattern was deliberately modified so as not to be equivalent to whole egg protein. Leucine, methionine and valine requirements were reassessed on this low protein diet. In the case of leucine, it was found that the 40 mg. per day required was only 1/4th to 1/10th the amount established by Rose and Leverton. Rose's study used twice as much protein and the whole egg was used as the amino acid pattern. Earlier studies by Fisher demonstrated lower requirements for both lysine and tryptophan when college women were on the low 5 gms. of nitrogen daily. Nitrogen balance is dependent upon the amount of protein fed as well as the amino acid pattern.

Krofranyi[23] determined that it required less protein to keep adult men in nitrogen balance when potato and egg was used rather than egg alone as the sole protein source. In fact, the essential amino acid requirements were as much as 50% lower than reported by Rose when the protein came from a potato-egg mixture. Next time you look up the biological value of protein foods as reported by Mitchell[24] and find that egg has a value of 94% and potato 67%, remember that this is valid for rats, not humans.

To summarize: assuming adequate calories, the nitrogen balance studies with human subjects ranging from infants to adults show no protein shortage when total protein was even as low as 7% of total calories and even when the sole protein was polished white rice.

Metabolic ward experiments are helpful, but the true test is in real life. The 50,000 Tarahumara Indians of Mexico are famed for their wooden kickball races. Men run, kicking a small wooden ball, for 48 hours continuously covering 180 miles in the process. Balke[25] authenticated these examples of Tarahumara fortitude:

- A 500 mile round trip run in 5 days;
- Carrying a 100 pound pack for 110 miles in 70 hours;
- Indian women playing the kickball game, but running continuously for only 50 miles.

The Tarahumara diet consists of 10% protein, 10% fat and 80% complex carbohydrate and comprises corn, peas, beans, squash and various other native plants and fruits. Animal protein is eaten perhaps a dozen times a year.

An average 40 year old Tarahumara man 5'7" weighs 125 pounds. Cholesterol levels, as might be expected from their diet, fall mostly in the 100 to 130 mg.%

range. Many physicians have examined these people and find them to be completely free of cardiovascular disease, hypertension or diabetes. If an 80% complex carbohydrate diet would seem to invite obesity, it should be noted that there are no fat Tarahumaras and, for that matter, substantial weight losses have occurred in obese men in our California study, where a similar diet is followed. When fat intake is drastically reduced, it is hard to consume enough calories to create weight gains.

Can a diet restricting fat to 10% produce fat deficiencies? The only fat the body cannot manufacture is linoleic acid. Winitz[26] has shown the daily requirement for linoleic acid to be only two grams. Recent findings by Press[27] demonstrate that only .1% of total calories as linoleic acid is required as a therapeutic dose to correct essential fatty acid deficiency: that is only 1/300th of an ounce per day.

In our preoccupation with protein needs, the resultant excess intake of fat and protein has been overlooked. There is a direct correlation between protein intake and excretion of minerals in humans. Margen[28] has been testing this concept since 1965 with over 2,000 University of California at Berkeley students. Findings on calcium are revealing: increasing protein intake from 0 to 90 gms. of nitrogen per day results in an 800% increase of calcium excretion, regardless of the calcium intake, which varied from 100 mg. to 2300 mg. This effect was noted without exception in all the studies conducted and was also repeated with synthetic amino-acid mixtures.

Confirming studies on mineral loss through high protein, low carbohydrate diets come out of Farleigh Dickinson University, where Dr. Fredericks has taught. Dr Yacowitz[29] found significant excretion losses in calcium, phosphorus, iron and zinc, as well as increased magnesium loss. His evidence of loss of bone matrix along with the calcium loss could be a factor in osteoporosis.

Several studies have indicated that young animals develop bone demineralization on meat diets.[30]

Ellis[31] has measured less aging bone loss in elderly vegetarians than in those on mixed diets.

Mazess[32] measured the bone mineral content of forearm bones in mature Alaskan Eskimos traditionally on high protein diets. He studied 413 Eskimos of all ages and found that after 40 years of age, Eskimos had 10-15% less bone density than whites. With each decade of age, the Eskimos had a 50 to 100% greater bone loss than whites. Bone loss started almost 10 years sooner than it did with whites even though calcium intakes were over 2000 mg. per day.

These studies may help us understand why western populations on high protein diets require so many mineral supplements,while underdeveloped populations like the Bantus on a 10% mostly vegetable protein intake require no supplements. On average calcium intakes of 350 mg. per day, Walker[33] reports that Bantu mothers give birth to an average of 9 children and yet their bones and teeth do not show loss of density.

I have already discussed another danger of high animal protein intake—the resultant ingestion of excessive amounts of cholesterol, leading to elevated plasma

cholesterol levels. High animal protein intake implies also the resultant ingestion of much animal fat. Fat consumption over 10 to 15% of total calories is excessive and toxic.

Excess fats, whether animal or vegetable, create damage in three ways: 1. Interference with carbohydrate metabolism which is a prime factor in diabetes; 2. Elevation of cholesterol and uric acid plasma levels resulting in the shifting of these substances into the tissues and extra-vascular spaces; and 3. The production of tissue anoxia.

One of the first to observe tissue anoxia produced by a high fat meal was Swank[34] who fed hamsters cream meals and then, through their transparent cheek pouches, observed the effects on the erythrocytes. As the chylomicrons started to pour in from the cream meal, the erythrocytes began to adhere to each other. In 3 to 6 hours after the feeding, the aggregation, now in rouleaux and irregular formations, completely blocked many capillaries. The aggregations directly affected the oxygen-carrying capacity of the erythrocytes, decreasing the plasma oxygen level to 68% of the starting value. It took 72 hours without food before the oxygen level reached 95% of the original value.

Could such a cream meal precipitate an angina attack because the oxygen-carrying capacity of the blood is lowered? Kuo's study[35] with 14 angina patients tested this possibility. After an overnight fast, each subject drank a glass of heavy cream, then rested quietly while half hour blood samples were drawn. In 5 hours, the influx of chylomicrons had caused the transparent fasting blood to become 600% more turbid on a plasma lactescence scale. At peak lactescence, 14 angina attacks occurred, simultaneous with ischemic ECGs and abnormal ballistocardiograms. The amazing similarity in the reaction of many individuals to fat was shown in almost identical lactescence curves for 13 of the 14 angina patients.

These same patients on another morning drank a fat-free drink with identical calories and bulk. After 5 hours, no increased blood turbidity, no angina and no abnormal ECG tracings could be noted.

Are unsaturated fats less harmful? Friedman[36] compared the effects of saturated and unsaturated fats on erythrocyte aggregation and blocking of arteries by correlating a series of microphotographs of conjunctival capillaries while monitoring blood lipids. Sludging and capillary blockage occurred after his subjects, 44 firemen, ingested a butterfat cream drink following an overnight fast, but this also occurred when the same subjects following the same procedure another day substituted a safflower oil drink of equivalent fat content. The unsaturated safflower oil aggregated the erythrocytes and blocked the capillaries just as the cream had, but worse, the safflower drink kept the 4 hour post prandial triglyceride level elevated even longer than had the butterfat drink. After the butterfat drink, the triglyceride value had returned to normal within 9 hours in every person; after the safflower oil drink, in some the triglyceride level did not even start to drop after 9 hours.

Platelet aggregation occurs under the same conditions that produce erythrocyte aggregation. A U.S. Department of Agriculture study, directed by Iacano, [37] placed normals on a 25% fat diet instead of their usual 40-45% fat intake. Not only did blood pressure and cholesterol levels drop, but there was a 50% drop in platelet aggregation. When the diet of 40-45% fat was resumed, the platelet aggregation returned to previous levels. Dr. Iacano said, "There is no question that these changes result from the changes in the amount of fat in the diet."

Even the so-called "good" fats like arachidonic acid aggregate platelets. Arachidonic acid is only found in animal sources; human enzyme systems quickly convert it into short-lived endoperoxides and these are metabolized into prostaglandins, which are potent triggers of irreversible platelet aggregation and thrombosis. Silver[38] found that arachidonic acid used intravenously can produce rapid respiratory death in rabbits from platelet occlusion of the small pulmonary vessels.

Hazlett,[39] chief of the pulmonary functions lab at Fitzsimmons Army Hospital in Denver, repeated Silver's work using rabbits and also humans. Using a fat very familiar to most of us, soybean lecithin, he tested male subjects between 19 and 25 years of age, all with normal pulmonary function. The lecithin-induced hyperlipidemia reducing lung function and producing erythrocyte aggregation lasted up to 48 hours—a confirmation of Swank's hamster tests.

Rabbits made hyperlipidemic with lecithin had severe impairment of their alveolar capillary gas exchange. Photos of their lungs showed fat droplets lining the capillary walls, surrounding erythrocytes and filling some capillaries. The investigator, Hazlett, said that in such a low pressure circulatory system as pulmonary capillaries, this could easily lead to obstruction.

Two years later, Pirkle,[40] a pathologist, confirmed Hazlett's apprehensions. He described 6 specific deaths of healthy young people 17 to 40 years old. The only abnormal findings were platelet aggregation in the small pulmonary vessels. The deaths were sudden and were marked by extreme respiratory distress lasting up to 30 minutes. Some of the aggregates were free-floating and others were blocking vessels. Pirkle believes these cases may explain thousands of unexpected natural deaths that occur each year.

Perhaps polyunsaturated fat containers should be labeled like cigarettes—"Warning: this could be dangerous to your health." For that matter, so should the marbelled steak and package of cheese. Dietary changes will not come easily, but for a nation suffering a million cardiovascular deaths yearly, what are the options? . . . Surgical? Coronary bypass is worse than palliative: 20% of bypasses completely close in the first 12 months . . . Pharmaceutical? The massive 8,000 man 5 year double blind Coronary Drug Project[41] is over. This evaluation of the most promising drugs available to the coronary patient indicate that if he had taken placebos he would have been better off than had he followed any recommended drug therapies. The drug testing included nicotinic acid at 3 grams per day, which was not only of no benefit, but had toxic side effects ranging from visual damage to increased cardiac arrhythmias.

Suppose, for the moment, that all the evidence presented so far about the dangers of excess fat and cholesterol were unknown. All that would be needed to discover the reality of these dangers would be to observe the effects of western diets on the gallbladder and intestinal tract. Population studies indicate that cholesterol gallstones are found exclusively in populations on the western diet. Tanaka[42] fed squirrel monkeys cholesterol-free diets. One diet used butterfat; the other, safflower oil. After a year, none of the monkeys had developed gallstones. Repeating the same diets but adding cholesterol produced gallstones in over 80% of the monkeys on both diets.

Taylor[43] used prairie dogs obtaining the same results. Without cholesterol, no gallstones; with cholesterol added in the form of egg yolks, 100% of the animals developed gallstones. In an attempt to dissolve the stones, he fed them lecithin at 3.6% of total calories; no effect was observed. But upon eliminating the dietary cholesterol, the stones dissolved.

Den Besten[44] followed this experimentation with humans. She fed them two isocaloric diets, one cholesterol-free, and the other containing 750 mg. of cholesterol in the form of egg yolk. In only three weeks, the cholesterol diet led to significant elevation of cholesterol in the bile, bringing 40% of the subjects into the lithogenic range, and in most of these, cholesterol crystals were now found in their bile.

Lithogenic bile—bile containing more cholesterol than the combined action of bile salts and lecithin can solubilize, can be manufactured at will: just add enough cholesterol.

Let's jump from the gallbladder into the intestine. Hill[45] and his group have been actively searching for the causes of colon cancer. From World Health Organization statistics, Hill noted the correlation between high fat intake and colon cancer was extremely high—.99. Checking the feces from people in high incidence areas, he found 100 times more anaerobic bacteria than in those people from low incidence areas. This was an exciting find, since the anaerobics are able to convert bile acids into carcinogens. Those on low fat and cholesterol diets were found to have mostly aerobics as enterococci that were unable to perform the bile conversion. This observation was confirmed by Wynder[46] who placed subjects who were on western diets on a non-meat diet and measured the significant drop in anaerobic bacteria and total bile acids.

The rate of bile degradation is dependent upon the quantity of bile as a substrate. With a 40% fat intake, typical of western diets, the bile acid concentration measured by Hill was 300-400% higher than with a 10% fat intake.

Testing 44 patients with colon cancer, Hill[47] found 82% with high fecal bile acid concentrations and coexisting anaerobic bacteria. In the same hospital, among 90 patients with other diseases, only 9% had high bile acids.

In other studies, Binder[48] found that the important function of bile acids in inhibiting anaerobic bacterial growth was prevented by lecithin.

Still another finding on high fat diets is the action by the lecithinase-negative Clostridia bacteria in converting the bile into estrogens, which could stimulate

breast tumor growth. Hill[49] noted that the correlation of high fat diets to breast cancer was .8, almost as high as with colon cancer.

These revelations concerning the relationship of fat and cholesterol to gallbladder and colon diseases result from a few dozen investigators, in less than the past 10 years, having asked the right questions. Added to other recent knowledge about the role of fat and cholesterol in degenerative diseases, the urgency for radical diet reform in the direction the data indicates becomes clear.

I have appreciated the opportunity to bring to the members of this audience, who are in the forefront of preventive medicine, a rationale for a high carbohydrate diet.

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