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American
Heart
Association



NUTRITION COMMITTEE

AHA NATIONAL CENTER ROOM C-3

Thursday, April 5, 1979

8:30 am-5:00 pm

Friday, April 6, 1979

8:30 am- noon

ROBERT SHANK, M.D.

ADDITIONAL AGENDA ITEMS

D. Nutrition

AHA SUBGOAL IN NUTRITION: To influence Americans to modify their dietary habits to conform with AHA recommendations.

The development and progression of coronary heart disease is influenced by many factors: heredity, environment, and lifestyle. Among these factors, a diet rich in calories, saturated fat, and cholesterol can contribute to hyperlipidemia, obesity, and increased risk for developing coronary heart disease.

There is substantial evidence that the diet recommended herein will aid in the control of serum lipid levels in man. Present evidence also suggests that maintaining serum lipids at reduced levels will lower the incidence of heart attack caused by atherosclerosis of the coronary arteries. Diets similar to the one recommended have been consumed by many persons in the United States for periods of more than ten years without evidence, clinical or biochemical, of harmful effects. Worldwide population studies have yielded similar findings.

The recommended diet includes:

- Caloric intake adjusted to achieve and maintain ideal body weight;
- A reduction in total fat calories to no more than 35% of calories. This is achieved by a substantial reduction in dietary saturated fatty acids;
- A substantial reduction in dietary cholesterol to approximate 300 mgs. daily;
- Increased dependence on foods containing complex natural carbohydrates, with reduction in the use of refined sugar; and
- Avoidance of excessive salt in the diet.

These recommendations describe the minimum in terms of dietary restrictions and are appropriate for the population in general. However, there are many individuals who because of genetic or environmental influences require major diet changes characterized by greater reductions in saturated fat and cholesterol.

The practical implementation of these diet recommendations by the public of the United States is difficult because of their limited knowledge of the composition of foods and widespread consumption of processed food-stuffs. The food industry and government can alleviate the situation in the following ways:

1. Nutritional Labeling

Nutritional labeling is an effective way to provide the consumer with nutrition information about food. The AHA recommends:

- Efforts that will expand the ability of the public to obtain this information to improve the nutritional quality of their diet.
- Efforts to expand the number of items of nutritional information on the label which will benefit those who need specific information, e.g., cholesterol, salt and saturated fat content.
- Efforts to provide adequate government funds for monitoring labeling.

2. New Food Products

Ready availability of food products low in fat, refined sugar and salt content at reasonable cost is essential for those attempting to modify their diets in these respects. The AHA recommends:

- Government and industry programs to stimulate the development and testing of these products.

3. Nutrition Advertising

The public is in need of nutritional guidance to assist them in making wise food choices. Food advertising is one of the most powerful means of communicating nutrition information. To insure its accuracy and honesty, the AHA recommends:

- Increased government monitoring of food advertising, together with full use of "cease and desist" and other administrative powers to eliminate false, deceptive or misleading health claims in such advertising.

4. Government Food Programs

Government food programs have an important impact on the American diet, either by directly influencing daily nutritional intake or by affecting the development of eating habits. These include school lunch, food stamp, and other supplementary food programs, as well as government-administered food services, such as the Armed Forces or Veterans Administration facilities. These programs provide an excellent opportunity for improving the diet for a sizeable segment of the U.S. population. Therefore the American Heart Association recommends:

- Revision of policies, regulations, practices and educational aspects of government food programs accordingly to promote and facilitate the consumption of a nutritionally balanced, fat-modified diet for all people who wish to do so.

5. Nutrition Education

Educational programs at all levels should be designed to promote understanding and appreciation of the need for primary prevention and inform the public and health professions of ways of selecting and preparing foods consistent with good nutritional practices. The American Heart Association recommends:

- Government or private action for the development of comprehensive and sustained public and professional nutrition education programs, including medical school curricula.

6. Nutrition Research

Successful preventive and therapeutic measures for cardiovascular disease can only be realized through effective application of existing scientific knowledge and a continuous flow of new knowledge. The American Heart Association recommends:

- Nutrition research on a basic and applied clinical level to fill the existing gaps in knowledge relative to the relationship of nutrition to disease.
- Research programs to obtain more specific information about how various groups in the population can better benefit from specific nutrition information.
- Research designed to detect and measure nutritional deficiencies.
- Research relative to nutrition and diet as a therapeutic modality in the treatment of disease, especially cardiovascular disease.
- Research on the interaction of nutrients with each other and with drugs.
- Research on the cause of obesity as well as the social and psychological aspects of overeating, and how people can modify their eating practices toward more healthful patterns.
- Support funding for analysis of commonly used foods, including content of calories, cholesterol, total fat, saturated fatty acids and unsaturated fatty acids, sodium and potassium, fiber, and the various carbohydrates.
- Funding for research efforts to fill the important gaps in knowledge relative to nutrition and diabetes mellitus, e.g., specially controlled prospective studies to determine an optimal diet for diabetics that will avoid long term complications of diabetic neuropathy, microangiopathy, and atherosclerotic vascular disease.

7. Nutrition and the Nation's Health Care System

Nutritional adequacy is a prerequisite for good health. Delivery of good nutritional care is essential to the prevention of conditions related to cardiovascular disease. The American Heart Association recommends:

- The development of a national nutrition policy characterized by the incorporation of appropriate goals and standards on nutrition for healthy persons as well as those whose nutritional needs have been altered by chronic disease.
- The establishment of nutrition education programs at state and local health agency levels.
- The development of model nutrition education programs for those in training for health professions.
- The incorporation of third party payments for nutrition counseling.

8. Allocation of Responsibility for Federal Nutrition Programs

Human nutrition is both a health science and an agricultural science. The American Heart Association recommends:

- That health research and education be the responsibility of those government agencies whose responsibilities encompass health.
- That the agricultural sciences be the responsibility of the agencies that specialize in dealing with agricultural problems.

E. Other Issues in Primary Prevention

1. Rheumatic Fever

Means are available for the control of rheumatic fever and rheumatic heart disease. The first step in primary prevention of rheumatic fever and rheumatic heart disease is the recognition of streptococcal infection and its confirmation by culture. The second step is effective antibiotic therapy for the infection. Since the incidence rates for rheumatic fever and rheumatic heart disease are highest among disadvantaged groups, it may be necessary to have government programs to assist in effecting both of these steps. For these reasons, the American Heart Association recommends:

- Programs which insure local availability of throat culture services, at little or no cost to the patient, and optimum patient follow-up.
- Public financial coverage for rheumatic fever prevention drugs for low income patients who do not qualify for Medicaid.

2. Sedentary Living

The benefit of physical activity in preventing the initial manifestations of atherosclerotic disease remains undetermined, but regular physical activity probably decreases the risk of coronary disease and may promote convalescence after heart attack. Accordingly, the American Heart Association recommends:

- Programs which create incentives for more physical activity as a part of balanced living in the general population.

3. Water

The American Heart Association is often asked for its position on the fluoridation of drinking water. The Council on Epidemiology has repeatedly held and the Steering Committee has accordingly affirmed that there is no evidence that adjustment of the fluoride content of public water supplies to a level of not more than 1 ppm has any deleterious effect on the cardiovascular system.

Investigators in several countries have observed that hard water supplies are associated with lower cardiovascular death rates. Despite 49 epidemiologic studies from nine countries the role water plays in cardiovascular disease is by no means certain. It is still not clear whether there are protective trace elements in hard water, harmful elements in soft water or whether water quality reflects a more fundamental geochemical influence possibly unrelated to water. Therefore, the American Heart Association recommends:

- Government sponsored studies of water quality, including its trace metal content, particularly in areas which have or are likely to change their water supply, in relation to cardiovascular disease rates.

EARLY INTERVENTION, DIAGNOSIS AND TREATMENT

F. High Blood Pressure

AHA SUBGOAL IN HIGH BLOOD PRESSURE: To reduce the prevalence of uncontrolled high blood pressure.

High blood pressure is a leading public health problem in this country. One every six adults has some elevation of blood pressure. High blood pressure can result in stroke, congestive heart failure, and is a major risk factor in coronary artery disease.

Below is a comprehensive legislative approach to meet current high blood pressure priorities of the American Heart Association.

1. Detection (Screening)

High blood pressure may be asymptomatic until the onset of cardiovascular complications and in the asymptomatic stage can only be detected by measuring blood pressure. In addition, high blood pressure is unusually common in Black populations which are economically

Absorption of food iron¹

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Iron lack is the commonest deficiency state in man. In contrast with other dietary deficiencies, iron deficiency is not closely linked with socioeconomic status but is widely prevalent both in developing countries and in highly industrialized nations. Although rarely encountered in men, it has been estimated that more than 5 million adult women in this country have iron deficiency anemia and that at least twice this number have no iron reserves to meet the physiologic needs of menstruation and pregnancy. Because the normal diet contains more than 5 times the total amount of iron needed to maintain iron balance, the problem is not so much inadequate intake but rather poor availability of dietary iron. It is this issue of food iron availability with which the present review is concerned.

METHODOLOGY

The development of our present knowledge of food iron availability has paralleled refinements in methods to measure iron absorption. No meaningful data were obtained in early studies using chemical iron balance, and it was not until the introduction of radioiron about 3 decades ago that the first reliable measurements of iron absorption were obtained. A measured quantity of radioiron is given orally and after several days to allow complete excretion of unabsorbed radioactivity, absorption is determined by measuring either the unabsorbed fecal radioactivity or more commonly the radioactivity retained in the body. While the latter can be measured by a whole body counting, the most common approach in recent years is to measure the incorporated red cell radioactivity in blood drawn 2

weeks following the test dose. In normal subjects the only errors with this method are due to variations in the red cell incorporation of absorbed radioactivity (80–100%) and estimating total blood volume from surface area. Both these errors are eliminated by performing multiple tests in the same subject and determining relative rather than absolute absorption, as discussed below.

A vexing methodologic problem is the enormous variability in iron absorption measurements. Most of this variation is not due to errors in actually determining absorption but rather to true fluctuations in absorption. Some of this variability is due to differences within the same subject when tested on different days. The effect of this physiologic variability can be reduced by administering the test dose over several days and by including a sufficient number of subjects to attain reasonable statistical precision. Of greater magnitude is the variation in iron absorption among apparently healthy subjects due to differences in iron status. The effect of this biologic variation can be reduced by performing multiple absorption tests in the same subject and thereby determining relative absorption from different forms of administered iron. By using dual radioiron tracers (⁵⁵Fe and ⁵⁹Fe) and measurements of incorporated red cell radioactivity, a total of four separate iron absorption tests can be performed in the same individual as two sets of parallel absorption measurements. The use of absorption ratios eliminates not only the effect of biologic or subject-to-subject variation but also eliminates any errors in estimating blood volume or assuming a constant red cell incorporation of absorbed radioactivity.

It is important with this approach to measure absorption in all subjects from a standard reference dose of inorganic iron. By expressing the absorption of iron from a given food relative to this reference dose, studies performed in subjects with widely different iron status can be compared.

Intrinsic labeling

The most important methodologic aspect of radioisotopic studies of food iron absorption is not the method of determining absorption but rather the nature of the administered radioiron. Until recently, biosynthetic labeling of foods, first introduced by Moore and co-workers more than a quarter of a century ago (21), has been considered the only valid approach. Vegetable foods are tagged intrinsically by growing in hydroponic mediums containing radioiron. Animal foods or fish are similarly tagged by injecting radioiron several months prior to sacrifice. Test meals containing a single labeled food item are prepared as they would normally be eaten and administered to fasting subjects in amounts that provide 3–5 mg iron or about 20–25% of the total daily iron intake.

Extensive data with intrinsic labeling have been obtained in recent years in a collaborative program by the Seattle and Caracas workers (12). Studies of 12 different foods in a total of 520 subjects have recently been summarized (18). All studies included absorption measurements from a

¹ From the American Institute of Nutrition Symposium on *Iron Absorption and Nutrition* presented at the 60th Annual Meeting of the Federation of American Societies for Experimental Biology, Anaheim, CA, April 14, 1976.

Abbreviations: STD, standard; SS, semisynthetic.

reference dose of 3 mg ferrous sulfate to allow corrections for differences in iron status. Overall mean absorption with seven vegetable foods was about 3-4%. Lowest absorption was observed with rice and spinach (about 1%) and highest with soybean (7%). Intermediate absorption was observed with black bean (3%), maize (4%) and wheat (5%), all of which are staple foods for a large portion of the world's population. The most significant finding in these studies was the consistently higher absorption obtained with foods of animal origin. Mean absorption of iron from veal liver, fish, and veal muscle were all greater than 10% and in many cases exceeded absorption of the reference dose of ferrous ascorbate. Iron absorption from purified animal sources, hemoglobin and ferritin, was intermediate between animal and vegetable sources of food iron.

Because none of the above foods would normally be eaten alone, data obtained with biosynthetic labeling would only be applicable if absorption was the same when the labeled food is given alone as when administered as part of a complete meal. However, studies in which two foods tagged with different radioisotopes of iron were administered simultaneously revealed major food interactions (17, 19). For example, iron absorption from maize increased about twofold when it was administered with fish or beef muscle while the absorption from fish decreased significantly when it was eaten with a vegetable food. In view of the complexities of a modern diet it became obvious that biosynthetic tagging would provide little practical information about food iron assimilation.

Extrinsic labeling

The most important advance in studies of food iron absorption is the use of an extrinsic tag to measure iron absorption from a complete meal. Using double radioiron labels, it was originally observed that when a small quantity of inorganic radioiron (extrinsic tag) is simply mixed with a food that has been biosynthetically labeled (intrinsic tag), absorption of the two tracers is virtually identical (4). Furthermore, their absorption remained identical when the dose of the extrinsic tag (ferric chloride) was

varied between 0.001 and 0.5 mg iron, when the extrinsic tag was added to the food either before or after cooking and when a variety of foods were tested (maize, wheat, soybean and black bean). The ratio of the extrinsic:intrinsic tag was also close to unity when absorption was decreased by adding desferrioxamine to the test meal and when absorption was increased by adding meat or ascorbic acid.

The validity of an extrinsic tag to measure the absorption of nonheme iron has now been confirmed with additional foods and by several laboratories (2, 3, 22). There have been a few exceptions. In a study with biosynthetically tagged rice (3), absorption of the extrinsic tag was higher than the intrinsic label when fed as rice grain but equal when fed as rice flour. The extrinsic radioiron apparently did not completely permeate the polished rice grain. Certain insoluble iron salts such as sodium ironpyrophosphate or ferric orthophosphate may have a lower absorption than the radioiron in foods fed simultaneously (5). Finally, it has been shown recently that labeled ferritin and hemosiderin may be less well absorbed than nonheme radioiron in foods tagged either intrinsically or extrinsically and administered in the same meal (16). While it has been suggested that these biologic iron complexes constitute a second pool of nonheme food iron, they probably do not represent a sufficient quantity of food iron to warrant separate consideration. Indeed, none of these exceptions to the validity of extrinsic tagging seriously limits the usefulness of the method.

Studies with an extrinsic tag have provided convincing evidence that there are no unique biologic complexes of nonheme iron in foods in terms of the absorption of dietary iron. An important corollary is that the absorption of nonheme iron from different foods ingested in the same meal will be the same. Evidence in support of this was presented in the original report (4). Iron absorption was measured from a complete meal that contained a small quantity of biosynthetically tagged maize. An extrinsic radioiron tag was either mixed with the meal after homogenization or simply taken as a drink during the meal prepared and served

as it would normally be eaten. In both instances absorption of the intrinsic and extrinsic tag were very similar. Additional evidence has been obtained using two foods biosynthetically tagged with separate radioiron tracers (3). When served separately, iron absorption from eggs and bread was 1-2 and 30% respectively; when administered simultaneously absorption was 5.0 and 5.3% respectively. These studies indicate that a common pool of nonheme iron is formed by foods ingested in the same meal and that iron absorption from this pool can be measured by extrinsic tagging.

In addition to the major fraction of nonheme iron in food, there is a second pool of heme iron that must be considered separately. It has been shown in both animals and man that heme iron is not degraded within the intestinal lumen but is absorbed into the mucosal cell as an intact porphyrin complex. The absorption of this heme iron differs markedly from nonheme iron in that it is not affected by enhancing substances such as ascorbic acid or by inhibiting substances such as phytate or desferrioxamine, which have a profound influence on the absorption of nonheme iron. Recent studies have established that extrinsic tagging can also be used to measure the absorption of dietary heme iron by adding a trace amount of labeled hemoglobin to the test meal (11, 13, 15). Furthermore, by using double radioiron tags of heme and nonheme iron, absorption from an entire meal containing both animal and vegetable foods can be determined simultaneously.

The use of dual radioiron tags to measure absorption from a complete meal has recently been validated in a study by Bjorn-Rasmussen and co-workers (1). Meals containing proportional amounts of all foods consumed in a typical 6-week diet were labeled with double extrinsic tags and administered to 32 young men. The daily intake of iron in these men was 17.4 mg of which 1 mg was in the form of heme iron. Absorption of heme and nonheme iron averaged 37 and 5.3% respectively or 0.37 and 0.88 mg daily. The total absorption of 1.25 mg iron daily is in reasonable agreement with estimates of daily iron loss. Similar data have been obtained by Martinez-Torres and Layrisse (13), who studied a meal of meat, black

bean, maize and rice containing a total of 4.5 mg iron. In normal persons, absorption from the 1.5 mg of heme iron was 27% or 0.34 mg as compared with an absorption from 3 mg nonheme iron of 6% or 0.12 mg. Thus nearly three-quarters of the 0.46 mg total absorption was derived from heme iron. With iron deficiency, absorption of heme and nonheme iron increased to 37% (0.52 mg) and 14% (0.43 mg) respectively, giving a total absorption of 0.95 mg of iron.

These studies clearly indicate the importance of animal sources of food iron. When meat is regularly consumed the relatively small amounts of heme iron it contains may account for as much as one-third of the total iron absorbed daily. However, heme iron is of lesser importance in studies of dietary factors that influence iron assimilation. Meat is seldom contained in the diets of populations where iron deficiency is most prevalent. More importantly, the absorption of heme iron is little influenced by the nature of the diet and can therefore be predicted from chemical measurements of the proportion of dietary iron ingested as heme iron.

DIETARY FACTORS

Despite the important capability of measuring iron absorption from a complete meal, the complexities of a modern diet present a significant challenge in evaluating food iron assimilation. It is now apparent that it is not the iron complexes in food that determine absorption but rather the composite effect of substances that either block or facilitate absorption from a common pool of iron. There remains then the important task of developing a model that will define these biochemical factors and determine their relative importance. The following model was developed for this purpose.

The starting point was to select a meal considered to represent a typical American dinner, which will be referred to as the standard (STD) meal. The meal contains lean beef, corn meal, potatoes, bread, margarine, peaches and ice milk. The total iron and caloric content is 4.1 mg and 700 kcal, respectively. This STD meal, prepared and served as it would normally be eaten, is tagged extrinsically by mixing 0.1 mg radioiron as ferric chloride with the potatoes.

One difficulty in using such a meal is the inability to vary one biochemical parameter while keeping other parameters constant. Because of this, a second meal was designed in parallel that matched precisely the total chemical composition of the STD meal. This semisynthetic (SS) meal contains dextrimaltose as carbohydrate, corn oil as fat, and egg albumin as the source of protein. Calcium and phosphorus are added as dibasic calcium phosphate and dibasic potassium phosphate. The meal is tagged extrinsically in the same manner as the STD meal and administered as a chilled suspension.

In an initial study, absorption of the two meals was measured in each of 32 female subjects (6). Absorption from the STD meal ranged from 1 to 43% with an overall mean of 10.0%. A much lower mean absorption of 1.8% (range, 1–28%) was observed with the SS meal. Because meat is known to enhance the absorption of nonheme iron, the 100 g beef in the STD meal was believed to account for its much higher availability. To study this the subjects were divided into 4 groups and fed the SS meal containing an additional 25, 50, 75, or 100 g beef. At each level of substitution egg albumin and other constituents were reduced to maintain the same total composition. With 25, 50, 75 and 100 g beef, the absorption ratio of substituted SS:SS meal averaged 1.14, 1.88, 2.11 and 3.55 respectively, as compared with 5.56 for the STD/SS meal. These results confirmed that beef accounted for most or all of the higher assimilation from the STD meal.

This model employing parallel meals of defined chemical content has been employed in several studies to examine factors influencing food iron availability. The first study concerned the effect of animal protein on nonheme iron absorption (7). The importance of animal protein in iron nutrition is recognized in current World Health Organization recommendations of daily iron intake. In adult women, an intake of 28 mg per day is recommended when less than 10% of calories are derived from animal protein as compared with only 14 mg per day when more than 25% of calories are supplied by animal protein. These recommendations assume that all animal proteins are equivalent in their enhancing effect on iron absorption. A study was therefore per-

formed to compare the enhancing effect on nonheme iron absorption of a variety of animal proteins.

Four iron absorption tests were performed in each of 70 female subjects divided into 9 groups of 7–10 subjects each. Absorption was measured in all subjects from the STD and SS meal. For the third and fourth absorption test, various sources of animal protein were substituted for the egg albumin in the SS meal and the beef in the STD meal. The substituted animal proteins included pork loin, lamb, beef liver, chicken breast, fish, whole milk, cheese, and powdered egg. Each food was added in amounts that replaced the protein supplied by 100 g beef in the STD meal (20 g protein). In one study group, the beef in the STD meal and the egg albumin in the SS meal were interchanged to provide a comparison of a total of 10 protein sources. When substitutions were made in the STD meal no adjustments in the other components of the meal were attempted. However, with substitutions in the SS meal, the other components were varied to maintain the same total chemical content in each substituted meal.

Two major categories of animal foods were defined. Animal tissues including pork, lamb, chicken and fish were all identical with beef in their ability to sustain the high absorption from the STD meal and to significantly enhance absorption from the SS meal. In contrast, when dairy products (egg, milk and cheese) were substituted for egg albumin in the SS meal, absorption did not change significantly and when substituted for beef in the STD meal, absorption decreased by 60–80%. Clearly, dairy products do not enhance the absorption of nonheme iron and should be excluded when estimating the enhancing effect of animal protein.

The SS and STD meals have also been used to evaluate the effect of certain chelates on the absorption of nonheme iron. Depending on their affinity for iron, chelates may either facilitate absorption by forming a complex with iron that remains soluble at the higher pH of the duodenum or, with stronger chelates such as desferrioxamine, may completely block the absorption of nonheme iron by effectively competing for iron with the gastrointestinal mucosa.

The most widely used iron chelate in our present diet is EDTA, which

is added to prevent oxidative damage to food by free metals. Food and Drug Administration regulations permit EDTA in certain foods at levels ranging from 25 to 800 mg per kg. We evaluated the effect of this chelate on iron absorption in the following manner (8). A preliminary study was performed to determine whether iron EDTA undergoes complete exchange with the nonheme pool of dietary iron. A tracer quantity of ^{55}Fe was first mixed with 50 mg EDTA and then added to either the SS or STD meal that had been tagged extrinsically with $^{59}\text{FeCl}_3$ in the usual manner. A mean absorption ratio of 0.94 for $^{59}\text{Fe}:^{55}\text{Fe}$ was obtained indicating essentially complete isotopic exchange. With this amount of EDTA (molar ratio of EDTA:iron approximately 2:1) absorption from both the SS and the STD meal was reduced by about 50%. In additional studies, the amount of EDTA added to the STD meal was varied over a range of 6–500 mg EDTA. Absorption was not significantly reduced with 6 or 25 mg EDTA. However, with 50 mg or more of the chelate, absorption progressively fell to about 20% of basal when 500 mg was added. Although the exact quantity of EDTA in our diet is not known, we have estimated that the diet may contain as much as 50 to 100 mg, levels shown in this study to significantly impair the absorption of nonheme iron.

Ascorbic acid is a dietary constituent with a much different effect on iron absorption. Vitamin C increases iron absorption by maintaining iron in a reduced, more soluble form and by forming a chelate with ferric iron at a low gastric pH that remains soluble when the pH rises in the small intestine. The enhancing effect of ascorbic acid is quite pronounced. For example, 60 mg added to a meal of rice more than tripled absorption and in another study, 150 g papaya containing 66 mg of ascorbic acid increased the absorption of iron from maize more than fivefold (15). Indeed, recent studies have indicated that food fortification with ascorbic acid may be more effective than iron in improving iron nutrition in countries with a high prevalence of iron deficiency.

The relationship between iron absorption and ascorbic acid has become more important because of Paulings' suggestion that large amounts (250–

4,000 mg daily) may reduce the incidence and severity of the common cold. To determine the effect on iron absorption of these higher levels, we measured absorption from the SS meal alone and when taken with various doses of ascorbic acid ranging from 25 to 1,000 mg (9). The mean ratios for absorption with:without ascorbic acid were as follows: 1.7 with 25 mg, 2.5 with 50 mg, 4.2 with 100 mg, 4.7 with 250 mg, 6.7 with 500 mg, and 9.6 with 1,000 mg. When 100 mg of ascorbic acid was added to the STD meal, the relative increase was somewhat less (mean ratio 1.67 with:without ascorbic acid) suggesting that animal protein and vitamin C may enhance iron absorption by a similar mechanism. The administration of ascorbic acid 4 or more hours prior to a test meal had no effect on absorption.

To relate these findings to current dietary practices, we conducted a limited telephone survey of 100 subjects. Sixty-three were taking supplemental vitamin C ranging from 50 to 2,000 mg daily. The median intake in the entire group of 100 individuals was 200 mg daily or an amount that would increase iron absorption 200–300%, depending on whether it was taken as a single morning dose or as divided doses with each meal. The long-term effect on iron status of these large amounts of vitamin C deserves further study.

There are many other dietary factors that may influence the absorption of food iron although the majority of these have not been sufficiently well defined to assess their overall importance in iron nutrition. Phosphate and to a lesser extent calcium have been reported to reduce food iron assimilation although most studies have been performed in animals rather than man. In a recent study, we have observed that iron absorption from the SS meal was not decreased with separate addition of calcium or phosphate, whereas the addition of both reduced absorption by 50–75% (20). Bran has recently been shown to produce a linear decrease in iron absorption from bread presumably due to its content of phytate. Disler and co-workers have recently shown that tea produces a marked depression in the absorption of nonheme iron due to the formation of insoluble iron tannate complexes (10). Other important dietary

factors will undoubtedly emerge in future studies.

IRON FORTIFICATION

The main importance of studies of food iron availability is in designing programs of iron fortification. Recent proposals by the Food and Drug Administration to increase the level of iron fortification in this country have generated strong opposition and the controversy still remains unresolved. Although few would question the desirability of reducing the prevalence of iron deficiency in adult women, there is concern that certain segments of the population not requiring additional iron may be at risk of developing iron overload. Those that oppose fortification have also argued that there is little or no evidence that any national iron fortification program has been efficacious. Undoubtedly, the benefit of an iron fortification program will depend on the nature of the diet. For example, in a study by Layrisse and co-workers (14) the addition of 60 mg iron as ferric chloride to a meal of maize resulted in an absorption of only 0.3 mg. When the same amount of ferric chloride was added to a meal containing both meat and maize, absorption increased to 1.2 mg and when added to meat alone, absorption increased to 3.3 mg iron. Because animal foods are seldom eaten in areas where iron deficiency is most prevalent, fortification of the diet with a substance such as ascorbic acid which increased the availability of native food iron may be a more reasonable approach than adding more iron to the diet.

Studies with an extrinsic tag suggest certain guidelines for food iron fortification irrespective of the amount of iron added. Although it has previously been considered that the vehicle for iron fortification is important, it is now apparent that an available iron salt will have the same absorption as the nonheme iron of the diet regardless of the vehicle. The choice of vehicle should be dictated by factors other than iron availability, such as distribution within the population and undesirable reactions of iron with the vehicle which may alter its taste or color.

From studies of extrinsic labeling, it is unlikely that any iron salt used for fortification will be better absorbed

than the nonheme iron of the diet. However, there are iron salts presently used for fortification that are less well absorbed. For example, it has been shown that the absorption of sodium pyrophosphate is only 5% and ferric orthophosphate only 30% the absorption of ferrous sulfate or reduced iron of small particle size (5). It is possible that as much could be achieved by employing iron salts for fortification that undergo complete isotopic exchange with nonheme iron as could be realized by increasing the level of iron fortification.

SUMMARY

The development of more sensitive measures of iron status in recent years has identified a continuing high prevalence of iron deficiency in adult women of the childbearing age. Most of this iron deficiency is nutritional in the sense that the diet does not contain sufficient quantities of available iron to replace physiologic losses associated with menstruation and pregnancy. Studies of food iron absorption are of central importance in understanding and counteracting this

iron deficiency. A major advance in this field has been the recent development of extrinsic tagging to measure absorption of nonheme iron from a complete diet. Such studies have highlighted the importance of animal tissues and ascorbic acid in improving food iron availability. Absorption inhibitors such as EDTA, phytate and tea may be equally important, although their exact role in iron nutrition requires further elucidation. Existing methodology has and will provide the basis for more rational approaches to food iron fortification. 57

REFERENCES

1. Bjorn-Rasmussen, E., L. Hallberg, B. Isaksson, et al. Food iron absorption in man—Applications of the two-pool extrinsic tag method to measure heme and nonheme iron absorption from the whole diet. *J. Clin. Invest.* 53: 247–255, 1974.
2. Bjorn-Rasmussen, E., L. Hallberg and R. B. Walker. Food iron absorption in man—I. Isotopic exchange between food iron and inorganic iron salt added to food: Studies on maize, wheat, and eggs. *Am. J. Clin. Nutr.* 25: 317–323, 1972.
3. Bjorn-Rasmussen, E., L. Hallberg and R. B. Walker. Food iron absorption in man—II. Isotopic exchange of iron between labeled foods and between a food and an iron salt. *Am. J. Clin. Nutr.* 26: 1311–1319, 1973.
4. Cook, J. D., M. Layrisse, C. Martinez-Torres, et al. Food iron absorption measured by an extrinsic tag. *J. Clin. Invest.* 51: 805–815, 1972.
5. Cook, J. D., V. Minnich, C. V. Moore, et al. Absorption of fortification iron in bread. *Am. J. Clin. Nutr.* 26: 861–872, 1973.
6. Cook, J. D., and E. R. Monsen. Food iron absorption. I. Use of a semisynthetic diet to study absorption of nonheme iron. *Am. J. Clin. Nutr.* 28: 1289–1295, 1975.
7. Cook, J. D., and E. R. Monsen. Food iron absorption. III. Comparison of the effect of animal proteins on non-heme iron. *Am. J. Clin. Nutr.* 29: 859–867, 1976.
8. Cook, J. D., and E. R. Monsen. Food iron absorption in man. II. The effect of EDTA on absorption of dietary nonheme iron. *Am. J. Clin. Nutr.* 29: 614–620, 1976.
9. Cook, J. D., and E. R. Monsen. Vitamin C, the common cold and iron absorption. *Am. J. Clin. Nutr.* 30: 235–241, 1977.
10. Disler, P. B., S. R. Lynch, R. W. Charlton, J. D. Torrance, T. H. Bothwell, R. B. Walker and F. Mayet. The effect of tea on iron absorption. *Gut* 16: 193–200, 1975.
11. Hallberg, L., and E. Bjorn-Rasmussen. Determination of iron absorption from whole diet—A new two-pool model using two radioiron isotopes given as haem and non-haem iron. *Scand. J. Haematol.* 9: 193–197, 1972.
12. Layrisse, M., J. D. Cook, C. Martinez-Torres, et al. Food iron absorption—A comparison of vegetable and animal foods. *Blood* 33: 430–443, 1969.
13. Layrisse, M., and C. Martinez-Torres. Model for measuring dietary absorption of heme iron—Test with a complete meal. *Am. J. Clin. Nutr.* 25: 401–411, 1972.
14. Layrisse, M., C. Martinez-Torres, J. D. Cook, et al. Iron fortification of food—Its measurement by the extrinsic tag method. *Blood* 41: 333–352, 1973.
15. Layrisse, M., C. Martinez-Torres and M. Gonzalez. Measurement of the total daily dietary iron absorption by the extrinsic tag model. *Am. J. Clin. Nutr.* 27: 152–162, 1974.
16. Layrisse, M., C. Martinez-Torres, M. Renzy and I. Leets. Ferritin iron absorption in man. *Blood* 45: 689–698, 1975.
17. Layrisse, M., C. Martinez-Torres and M. Roche. The effect of interaction of various foods on iron absorption. *Am. J. Clin. Nutr.* 21: 1175–1183, 1968.
18. Martinez-Torres, C., and M. Layrisse. Interest for the study of dietary absorption and iron fortification. *World Rev. Nutr. Diet.* 19: 51–70, 1974.
19. Martinez-Torres, C., and M. Layrisse. Iron absorption from veal muscle. *Am. J. Clin. Nutr.* 24: 521–540, 1971.
20. Monsen, E. R., and J. D. Cook. Food iron absorption in human subjects. IV. The effect of calcium and phosphate salts on the absorption of non-heme iron. *Am. J. Clin. Nutr.* 29: 1142–1148, 1976.
21. Moore, C. V., and R. Dubach. Observations on absorption of iron from foods tagged with radioiron. *Trans. Assoc. Am. Physicians.* 64: 245–256, 1951.
22. Sayers, M. H., S. R. Lynch, P. Jacobs, et al. The effects of ascorbic acid supplementation on the absorption of iron in maize, wheat and soya. *Br. J. Haematol.* 24: 209–218, 1973.

SODIUM AND HYPERTENSION

ANIMAL EXPERIMENTS

METABOLIC WARD

TREATMENT OF HYPERTENSION

CROSS CULTURAL

WITHIN CULTURES

COMMUNITY CHANGE

SALT and SODIUM

1 mEq sodium = 23 mg sodium

1 mg sodium = 0.04 mEq sodium

1 g salt = 17.1 mEq sodium

1 g salt = 0.39 g sodium.

ANIMAL EXPERIMENTS

(rat, rabbit, chicken, dog)

- 1) \uparrow NaCl \rightarrow Hypertension
- 2) \uparrow NaCl early in life \rightarrow Hypertension
and then normal salt
- 3) \downarrow NaCl \rightarrow No Hypertension
- 4) Sensitivity to Salt Varies

PATHOGENESIS

- 1) \uparrow ECF
- 2) \uparrow Reactivity to Pressor Stimuli
- 3) "Waterlogging"
- 4) Structural Change

PROTECTION - POTASSIUM

Animals

- 1) Modifies BP Elevation
- 2) Modifies Damage

Humans

- 1) Overbeck
- 2) Sasaki

TREATMENT OF HYPERTENSION

	Ambard, Beaujard	1904
	Allen, Sherrill	1922
	Kempner	1948
severe restriction	Dahl	1958
	Owens	1978
	Kawasaki	1978
	Parijys	1973
moderate restriction	Morgan	1975
	Morgnani	1976
	Morgan	1978

COMMUNITY INTERVENTION

	Sasaki	1977
	Farquhar	1978

CROSS-CULTURAL COMPARISONS

Show linear regression (i.e., dose response) of salt intake on:

1. Blood pressure levels
2. Prevalence of "hypertension"

WITHIN CULTURE COMPARISONS

Japan

Solomon Islands

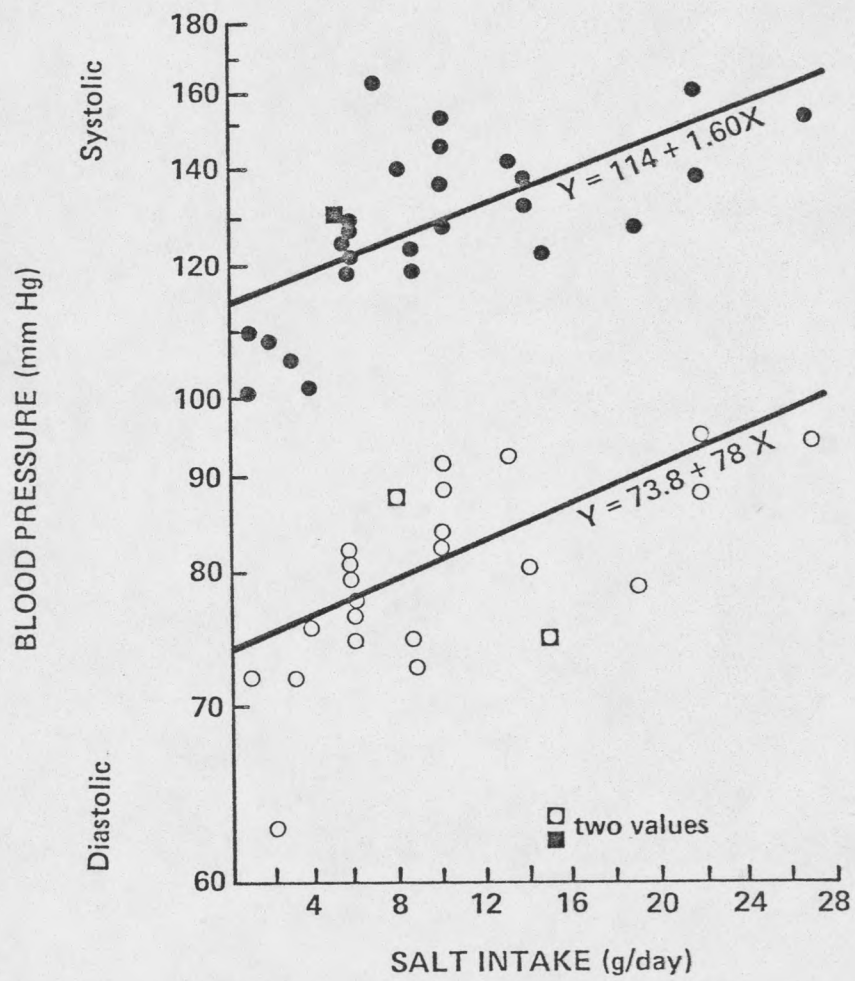
U.S.

Dahl yes

Dawber no ✓

Frank yes → *Bozulina*

(Calabrese, Farquhar)



INTERVENTION STRATEGY

No Comment

Saying nothing implies status quo is ok.

Procrastinate ?

E. Corday

"[Lipid disorders] It would appear to be a causative factor in only 5 to 20% of the American Public. It would seem unwise to tamper with the diets of the great majority of the U.S. population until we have more knowledge about whether dietary modifications in the clinical trials can prevent arterisclerosis in modern man."

Choose Target ?

1. American Academy of Pediatrics - Committee on Nutrition, 1976

"Eighty percent of population not affected by salt [because they do not have hypertension]."

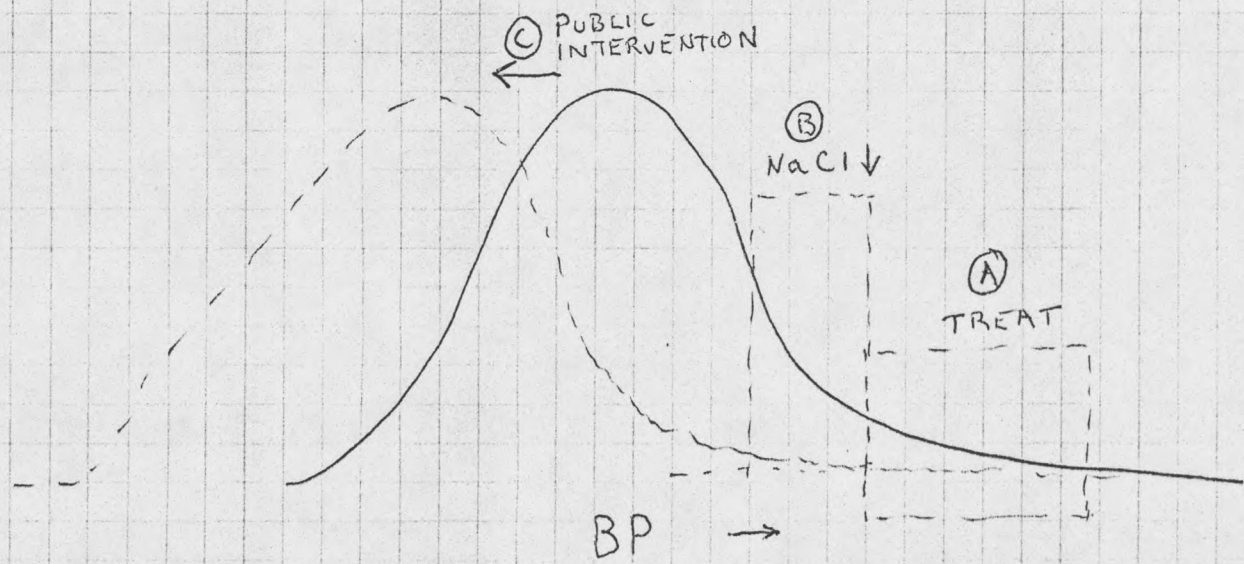
2. How?

Salt taste.

Family history.

BP level.

↑
EVENT
RATE



PICKING A NUMBER

EFFECTIVENESS

REQUIREMENTS

PALATABILITY

AVAILABILITY

PROPORTIONAL REDUCTION (PRESENT CONSUMPTION?)

SAFETY

SODIUM vs. SALT

FRESH FOOD

FOOD INDUSTRY  FOOD LABELLING
SODIUM CONTENT

TABLE SALT

WATER

MEDICATIONS

No intake - controlled for weight
ref - Copese + Stomach



Estimation of available dietary iron^{1, 2}

Elaine R. Monsen,³ Ph.D., Leif Hallberg,⁴ M.D., Miguel Layrisse,⁵ M.D.,
D. Mark Hegsted,⁶ Ph.D., James D. Cook,⁷ M.D., Walter Mertz,⁸ M.D.,
and Clement A. Finch,³ M.D.

ABSTRACT Dietary iron requirements are dependent on the amount and availability of food iron ingested. On the basis of recent studies of food iron absorption employing extrinsic tag techniques, the availability of heme iron has been defined and estimates of the availability of nonheme iron based on the amounts of enhancing substances appear possible. A model has been developed whereby the availability of iron in a given meal may be estimated. Calculations are made on a meal basis of 1) the amount of heme iron and its availability, and 2) the amount of nonheme iron and its availability as influenced by the meal's content of enhancing factors. Examples of these calculations are provided. *Am. J. Clin. Nutr.* 31: 134-141, 1978.

It is recognized that frank and occult iron deficiency is a relatively common problem in the United States. Considerations of requirements have focused on those of the adult woman since she represents the population consuming a general diet who is at greatest risk. The amount of iron a woman must absorb varies greatly and is dependent on the amount of iron lost during menstruation (1-3). It is estimated that some 1.8 mg of iron must be absorbed to meet the needs of 80 to 90% of women (4). The current Recommended Dietary Allowance for food iron intake was thus established at 18 mg/day, assuming an absorption of 10% (5). However, this amount can rarely be achieved with the ordinary foods available. Furthermore, these estimates were made at a time when there was limited information concerning the availability of food iron.

Accumulating evidence demonstrates that the amount of iron potentially available from foods depends not only upon the amount of iron supplied but the nature of that iron and the composition of the meal with which it is consumed (6-8). The total iron content of the diet is thus a relatively poor indicator of the adequacy of the diet with regards to iron. Although numerous

questions remain to be answered about iron availability and iron needs, sufficient information is now at hand so that better estimates of iron need in relation to diet can be made, and such information should be used in the development of diets and in making dietary recommendations.

Food iron absorption

The availability of food iron is best discussed under the headings of heme and nonheme iron, since considerable experi-

¹ Supported in part by a grant from the United States Department of Agriculture.

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TABLE 1
Factors for estimating percent absorption of dietary iron at increased levels of iron status (indicated by the quantity of iron stores)

	Women			Men
	Iron stores			
	mg			
I. Heme iron	0	250	500 ^a	1,000
II. Nonheme iron	35%	28 ^b	23 ^{a, b}	15
A. Low availability meal	5	4	3 ^a	2
1. Meat, poultry, or fish <30 g (i.e., <1 ounce) lean, raw wt or				
2. Ascorbic acid <25 mg				
B. Medium availability meal	10	7	5 ^a	3
1. Meat, poultry, or fish 30-90 g (i.e., 1-3 ounce) lean, raw wt or				
2. Ascorbic acid 25-75 mg				
C. High availability meal	20	12	8 ^a	4
1. Meat, poultry, or fish >90 g (i.e., >3 ounce) lean, raw wt or				
2. Ascorbic acid >75 mg or				
3. Meat, poultry, or fish 30-90 g plus ascorbic acid 25-75 mg				

^a The factors for 500 mg iron stores are suggested for most dietary calculations. ^b These factors are approximate values based on a semi-logarithmic relationship between iron stores (the linear function) and heme iron absorption (the logarithmic function).

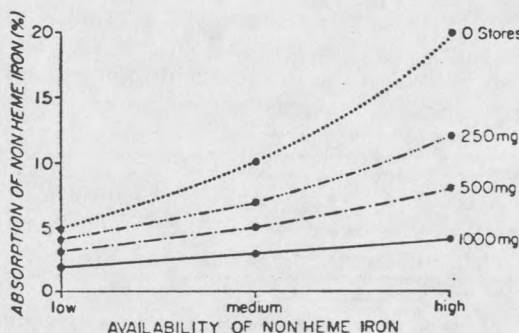


FIG. 1. The percent absorption of nonheme iron by individuals with no body iron stores, 250, 500, and 1000 mg body iron stores is shown as influenced by the availability of nonheme iron in a given meal.

mental data indicates that each of these forms a different pool of iron (6, 9). Heme iron which comes from hemoglobin and myoglobin, is absorbed directly as the intact iron porphyrin complex, and its iron is freed in the intestinal mucosal cell (10-13). The proportion of heme iron which humans usually absorb from their diet is high in compar-

ison with nonheme iron (14, 15). An individual who has no iron stores may be expected to absorb approximately 35% of heme iron when ingested as meat, while an individual with an adequate iron store of 500 mg would be expected to absorb approximately 25%. Analysis of a variety of foods has shown that approximately 30 to 40% of the iron in pork, liver, and fish and 50 to 60% of the iron in beef, lamb, and chicken is in the form of heme (16).

The nonheme iron pool consists of iron from other foods such as vegetables, grains, fruits, eggs, and dietary products as well as from the nonheme iron of meats, poultry, and fish and from soluble iron supplements. Although numerous isotopic studies have shown virtually complete mixing of the nonheme iron from all foods in a complex meal (9, 17, 18), certain forms of dietary iron may not be so completely available. Only a portion of the iron of ferritin and hemosiderin appears to enter this pool, the remainder being unavailable (19). The availability

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of iron in contaminating dirt may vary depending on the chemical properties of the dirt iron. Insufficient information is available to make any quantitative predictions of the absorption of such iron which seems to form only a minor part of the dietary

iron intake with usual American meals. Certain forms of fortification iron are also of limited availability, especially pyrophosphate, orthophosphate, and some preparations of ferrum reductum (20, 21).

Unique to nonheme iron is the fact that

TABLE 2
Examples of method to calculate absorbable iron from single meals

Food	Wt g	Total iron mg	Heme factor	Heme iron	Nonheme iron mg	Ascorbic acid (as served)
I. Meat, poultry, fish-containing—high availability (26 g protein; 650 kcalories)						
Beef-vegetable stew						
Beef, lean, raw, 3 ounce	85	2.7	0.4	1.1	1.6	0
Potatoes, 1/2 cup	78	0.4			0.4	13
Carrots, 2 tablespoon	20	0.1			0.1	1
Onions, 2 tablespoon	15	0.1			0.1	2
Green pepper, raw, 2 slices	20	0.2			0.2	26
Breadstick, 2 medium	35	0.3			0.3	Trace
Margarine, 2 teaspoon	10	0			0	0
Peaches, canned, 1/2 cup	128	0.4			0.4	4
Gingerbread	63	1.0			1.0	Trace
Totals		5.2		1.1	4.1	46
Ascorbic acid 46 mg						
Meat, fish, poultry 3 ounces, raw						
			% absorbable iron	23%	8%	
			Absorbable iron (mg)	0.25	0.33	
			total absorbable iron (mg)	0.58 mg		
II. Meat, poultry, fish-containing—medium availability (27 g protein; 700 kcal)						
Macaroni, tuna fish, and cheese casserole						
1/2 cup macaroni and cheese	120	0.9			0.9	Trace
1 ounce tuna, drained	29	0.6	0.4	0.2	0.4	0
Green peas, 1/2 cup	80	1.5			1.5	10
Cucumber, 3 large slices	14	0.2			0.2	1
Molasses cookies, 2	65	1.4			1.4	0
Blueberry muffin, 1	40	0.6			0.6	Trace
Totals		5.2		0.2	5.0	11
Ascorbic acid 11 mg						
Meat, fish, poultry 1 ounce, raw						
			% absorbable iron	23%	5%	
			Absorbable iron (mg)	0.05	0.24	
			Total absorbable iron	0.29 mg		
III. Nonmeat, -poultry, or -fish containing—low availability (22 g protein; 730 kcal)						
Beans, navy, cooked 1/2 cup	95	2.6			2.6	0
Rice, brown, cooked 1/2 cup	98	0.5			0.5	0
Cornbread, 1 piece	78	0.9			0.9	1
Margarine, 1 tablespoon	14	0			0	0
Apple slices, 1/2 cup	55	0.1			0.1	1
Walnuts, black, raw 1 tablespoon	8	0.5			0.5	0
Almonds, raw, 1 tablespoon	8	0.4			0.4	Trace
Yogurt, skim milk, 1 cup	226	0.1			0.1	2
Totals		5.1			5.1	4
Ascorbic acid 4 mg						
Meat, fish, poultry, 0 ounces, raw						
			% absorbable iron		3%	
			Absorbable iron (mg)		0.15	
			total absorbable iron	0.15 mg		

TABLE 2—continued

Food	Wt	Total iron	Heme factor	Heme iron	Nonheme iron	Ascorbic acid (as served)
	g	mg			mg	
IV. Nonmeat, -poultry, or -fish containing—high availability (23 g protein; 650 kcal)						
Red kidney beans, 1/2 cup	93	2.2			2.2	
Tomato sauce, 2 tablespoon	30	0.2			0.2	4
Broccoli, 2/3 cup	120	0.9			0.9	70
French bread, 1 slice	35	0.8			0.8	Trace
Margarine, 1 tablespoon	14	0			0	0
Cottage cheese, 1/4 cup	55	0.2			0.2	0
Pineapple, canned, 2 large slices	210	0.6			0.6	14
Banana, sliced, 1/4 cup	37	0.3			0.3	4
Totals		5.2			5.2	92
Ascorbic acid 92 mg						
Meat, fish, poultry, 0 ounces, raw						
			% absorbable iron		8%	
			Absorbable iron (mg)		0.42	
			Total absorbable iron		0.42 mg	

the amount absorbed can be modified markedly by components of foods ingested concomitantly (22). Dietary factors which dramatically increase the absorption of nonheme iron, as much as four fold, are ascorbic acid (23-25) and a "meat factor" present in meat, poultry, and fish (16). As the quantities of these substances in a complex or composite meal increase, iron absorption increases. Neither protein nor animal protein per se enhance iron absorption. While beef, lamb, pork, chicken, liver, and fish substantially raise the rate of nonheme iron absorption, milk, cheese, and eggs do not increase and may decrease iron availability (16). These enhancing factors play a very important role, for if they are not present in a meal, the absorption of nonheme iron is very low. It is also apparent that a group of other less well defined substances act to decrease nonheme iron availability. Tannic acid in tea (26), phosvitin of egg yolk (27), phytates (28), calcium and phosphate salts (29), EDTA (30), and antacids clearly suppress absorption when present in sufficient amounts. There may be other substances as well, and further studies are needed to clarify their role and particularly to determine the effect of the interaction of blocking and facilitating substances.

The absorption of nonheme iron depends not only on the general composition of the meal but along with heme iron is further affected by the iron status of the individual. Absorption of nonheme iron in the iron

deficient individual may be as much as 20% when enhancers are abundant (to be contrasted with about 35% heme iron absorption). A much greater difference is observed in the iron replete subject with a meal lacking enhancers and/or with high levels of blocking substances, where nonheme absorption is about 2% as contrasted to about 15% heme iron absorption. Even though the percent absorption of nonheme iron is considerably below that of heme iron, the quantity of nonheme iron in the diet is many fold above that of heme iron; thus, in most meals, the major contribution of available iron is made by nonheme iron.

Calculation of available food iron

In calculating the amount of iron absorbed from different meals, the amount of heme and nonheme iron ingested must be considered separately. Present evidence indicates that the nature of the meal has little or no influence on the amount of heme iron absorbed, with the possible exception of heme iron when used for fortification. Percentage absorption of heme iron will, however, be influenced by iron status in an inverse logarithmic function; in subjects with 0, 250, 500, and 1000 mg of iron stores, absorption from heme iron is estimated to be 35, 28, 23, and 15%, respectively, from the amounts of heme iron present in meat, poultry, and fish in usual American meals (Table 1). The proportion of heme iron contained in various animal tis-

meals. Cereals also of pyrophosphate preparations). The fact that

Ascorbic acid (as served)

0
13
1
2
26
Trace
0
4
Trace
46

Trace
0
10
1
0
Trace
11

0
0
1
0
1
0
Trace
2
4

%
15

TABLE 3
A sample day's calculation of absorbable iron

Time of Day	Food	Wt g	Total iron mg	Heme factor	Heme iron	Nonheme iron mg	Ascorbic acid	Meat, fish, poultry (raw) oz	Level of availability	% absorb- able iron	Total ab- sorbable iron mg
7:30 AM	Cornflakes, 1 ounce	30	0.4			0.4	0				
	Milk, 1 cup	240	trace			2					
	Sugar, brown, 2 teaspoons	10	0.3			0.3	0				
	Toast, buttered whole wheat	20	0.5			0.5	Trace				
	Honey	10	trace			Trace					
Subtotal	Orange juice, 1/2 cup	120	0.1			0.1	54	0	Medium	5%	0.065
						1.3	56				
10:30 AM	Coffee, 1 cup	240	0.1			0.1	0				
	Coffee cake, 1 piece	60	1.0			1.0	Trace				
Subtotal						1.1	0	0	Low	3%	0.033
12:30 PM	Chili con carne	160	2.7			1.0					
	With beans, 2/3 cup (contains 1/2 ounce beef)		1.6	0.4	0.6	1.1					
	Cheddar cheese, grated on top 1/2 ounce	15	0.2			0.2	0				
	Coleslaw, 1/2 cup	100	0.4			0.4	29				
Rye crisp crackers	30	1.2			1.2	0					
Peach, fresh	110	0.6			0.6	8					
Coffee, 1 cup	240	0.1			0.1	0					
Subtotals	Nonheme iron		0		0.6	4.6	37	1 1/2	High	8%	0.368
	Heme iron		0		0.6	4.6				23%	0.138
4:00 PM	Soda pop, 1 cup	240	0			0	0	0			
Subtotal						0	0	0	Low	3%	
7:00 PM	Roast turkey	120	2.8			1.7	0				
	Cranberry sauce, 1 tablespoon	15	trace			trace					
	Stuffing, 1/2 cup	100	1.0			1.0	trace				
	Acorn squash, baked, 1 piece	100	1.1			1.1	13				
	Tossed green salad	100	1.2			1.2	12				
	Roquefort dressing, 1 tablespoon	15	trace			trace					
	French roll	45	1.1			1.1	trace				
	Margarine, 2 teaspoon	5	0			0					
	Plums, canned, 1/2 cup	110	1.0			1.0	2				
	Chocolate nut brownie	30	0.6			0.6	trace				

100	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.1
100	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
15	trace	trace	trace	trace	trace	trace	trace	trace	trace
45	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.1	1.1
5	0	0	0	0	0	0	0	0	0
110	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
30	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6
12	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2	1.2
trace	trace	trace	trace	trace	trace	trace	trace	trace	trace
0	0	0	0	0	0	0	0	0	0
2	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
trace	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6
4	7.7	7.7	7.7	7.7	7.7	7.7	7.7	7.7	7.7
High	8%	23%	8%	23%	8%	23%	8%	23%	8%
0.616	1.473	1.473	1.473	1.473	1.473	1.473	1.473	1.473	1.473
1.473	1.473	1.473	1.473	1.473	1.473	1.473	1.473	1.473	1.473

Total absorbable iron = 1.473 = 1.5 mg

- Stalling, 1/2 cup
- Acorn squash, baked, 1 piece
- Tossed green salad
- Roquefort dressing, 1 tablespoon
- French roll
- Margarine, 2 teaspoon
- Plums, canned, 1/2 cup
- Chocolate nut brownie

Subtotals

Nonheme iron

Heme iron

Totals

2500 kcal

90 g protein

120 mg ascorbic acid

16.4 mg total iron content

1.7 mg heme iron

14.7 mg nonheme iron

1.5 mg absorbable iron

199

200

sues undoubtedly varies. However, these differences are probably not of sufficient magnitude to justify separate factors for each type of animal tissue. We have, therefore, assumed a heme iron content of 40% of the iron in all animal tissues including meat, liver, poultry, and fish.

In addition to iron status, the absorption of nonheme iron is markedly influenced by the nature of the meal. There are several factors which have been studied which either enhance or inhibit the absorption of nonheme iron. At this point, however, only two enhancing substances have been sufficiently well defined on a quantitative basis to allow estimates of their effect. These two factors are the amount of ascorbic acid and the quantity of animal tissue. The availability of nonheme iron from different meals has been classified somewhat arbitrarily as low, medium, and high according to the content of these substances. Because much or all of ascorbic acid contained in the meal may be inactivated by heating and oxidation during the storage and preparation of the food, estimates of ascorbic acid should be based on the actual amount contained in the meal as eaten. In contrast, the enhancing effect of animal tissues is not diminished during preparation of the meal.

In estimating the enhancing effect of either ascorbic acid or animal tissue, we have considered 1 g of meat roughly equivalent to 1 mg of ascorbic acid as eaten. More recent studies (4) have defined the effect of these substances when added separately to a meal, and it is much less clear at present whether their effect is additive when both are contained in the same meal. We have assumed for the present calculations that these effects are additive and increase in proportion to the amount present in a meal (Fig. 1).

The method proposed to estimate the quantity of absorbable iron in a given meal requires computation of 5 sums: 1) total iron, 2) heme iron, 3) nonheme iron, 4) ascorbic acid, and 5) meat, poultry, and fish. The iron in the meal can then be classified as having high, medium, or low availability. It is suggested that the reference level for body iron stores of 500 mg be used for calculations. Examples have

meal by meal!


been prepared for 4 meals to illustrate the effects of ascorbic acid and meat tissues on the estimated amount of iron absorbed (Table 2). Each meal has been carefully constructed to provide 5 mg of iron and approximately 25 g of protein and 700 kcal and could serve as the major meal of the day. While the meals are similar in these regards, the amount of iron which could be absorbed differs four-fold. Iron absorption from the low availability nonmeat tissue containing meal is estimated to be 0.15 mg and from the medium availability meal containing meat, poultry, or fish to be 0.29 mg of iron. In contrast, the estimated iron absorption is two to four times higher from the high availability meals, one containing meat, poultry, or fish and the other without meat tissue. To calculate the iron absorption from a day's diet, each meal and snack must be computed separately, with appropriate factors for iron availability assigned to each meal and snack. Subsequently, the iron absorption from each meal and snack can be tallied together (Table 3).

The basic assumptions in this paper are: 1) that an average of 1.4 mg iron/day will be required to be absorbed by the menstruating woman to replace physiologic losses and that 2.8 mg/day will be required to replace the total body iron losses of up to 95% of menstruating women; 2) that an inverse relationship exists between iron stores and absorption so that the higher iron stores, the lower will be the percent of available iron absorbed; 3) that iron stores of about 500 mg are desirable if women are to meet the requirements of pregnancy without supplemental iron.

Three examples will demonstrate the application of these estimates. A woman with no iron stores who has a daily intake of 18 mg of iron and a need to absorb 2.8 mg would absorb about 0.6 mg of heme iron (about 35% of the 1.5 to 2 mg of iron present in this form) and 2.2 mg of nonheme iron (10 to 15% of the 16 mg of nonheme iron) to meet her iron needs. The second example is an average premenopausal woman with 250 mg iron stores who has a need to absorb 1.4 mg would absorb 0.5 mg of heme iron and about 1.1 mg of nonheme iron (assuming 7% absorption). The final example would be a man with

iron stores of 1000 mg and a daily iron intake of 20 mg would be expected to absorb about 0.4 mg of heme iron and 0.7 mg of nonheme iron (3 to 4%). The adequacy of the diet must be based on a composite of a number of individual meals over several days in order to see a meaningful analysis.

Several assumptions have been made in the development of the proposed model which may restrict its application. All types of heme iron have been assumed to be equivalent. However, as mentioned earlier, there are certain forms of nonheme iron which are not entirely exchangeable with the nonheme pool. Particularly, fortification iron involving iron compounds of low availability cannot be included in these calculations. The calculations have not dealt with inhibitory factors and, obviously, the presence of tea, phytates, antacids, etc., will significantly affect the amount of available iron. In future calculations of iron absorption, it may be feasible to consider the content of inhibiting substances, especially if excessive amounts are present. Further, it is recognized that as the quantity of available iron in a meal is increased, percent absorption will decline; it is unlikely, however, that any adjustment would be necessary for single meals supplying up to 15 mg of iron.

The inference of these calculations is that 95% of women may attain iron balance with most meals of high iron availability at an intake of 15 mg or moderate availability with an 18 mg daily iron intake. Nutrition may be improved by increasing either the amount of iron in the diet or its availability. It is likely that both will be necessary if current prevalence of iron deficiency in premenopausal women is to be decreased to $\leq 5\%$, and if average iron stores of 250 to 350 mg are increased to 500 mg. The proposed dietary calculations are believed to provide a more accurate means of estimating the adequacy of diets with respect to iron. 

References

1. BALDWIN, R. M., P. J. WHALLEY AND J. A. PRITCHARD. Measurements of menstrual blood loss. *Am. J. Obstet. Gynecol.* 81: 739, 1961.
2. HALLBERG, L., A. M. HOGDAHL, L. NILSSON AND G. RYBO. Menstrual blood loss—a population

- study. Variation at different ages and attempts to define normality. *Acta Obstet. Gynecol. Scand.* 45: 320, 1966.
3. COLE, S. K., W. Z. BILLEWICZ AND A. M. THOMSON. Sources of variation in menstrual blood loss. *J. Obstet. Gynaecol. Brit. Commonwealth* 78: 933, 1971.
 4. Control of nutritional anemia with special reference to iron deficiency. Technical Report Series 580, World Health Organization, Geneva, 1975.
 5. Recommended Dietary Allowances, (8th ed). Food and Nutrition Board, National Research Council, National Academy of Sciences, Washington, D. C., 1974.
 6. HALLBERG, L., AND E. BJORN-RASMUSSEN. Determination of iron absorption from whole diet. *Scand. J. Haematol.* 9: 193, 1972.
 7. HALLBERG, L., L. GARBY, R. SUWANIK AND E. BJORN-RASMUSSEN. Iron absorption from Southeast Asian diets. *Am. J. Clin. Nutr.* 27: 826, 1974.
 8. LAYRISSE, M., C. MARTINEZ-TORRES AND M. GONZALEZ. Measurement of the total daily dietary iron absorption by the extrinsic tag model. *Am. J. Clin. Nutr.* 27: 152, 1974.
 9. COOK, J. D., M. LAYRISSE, C. MARTINEZ-TORRES, R. WALKER, E. MONSEN AND C. A. FINCH. Food iron absorption measured by an extrinsic tag. *J. Clin. Invest.* 51: 805, 1972.
 10. CALLENDER, S. T., B. J. MALLETT AND M. D. SMITH. Absorption of haemoglobin iron. *Brit. J. Haematol.* 3: 186, 1957.
 11. TURNBULL, A. L., F. CLETON AND C. A. FINCH. Iron absorption. IV. The absorption of hemoglobin iron. *J. Clin. Invest.* 41: 1897, 1962.
 12. CONRAD, M. E., B. I. BENJAMIN, H. L. WILLIAMS AND A. L. FOY. Human absorption of hemoglobin iron. *Gastroenterology* 53: 5, 1967.
 13. LAYRISSE, M., AND C. MARTINEZ-TORRES. Model for measuring dietary absorption of heme iron: test with complete meal. *Am. J. Clin. Nutr.* 25: 401, 1972.
 14. BJORN-RASMUSSEN, E., L. HALLBERG, B. ISAKSSON AND B. ARVIDSSON. Food iron absorption in man. Applications of the two-pool extrinsic tag method to measure heme and nonheme iron absorption from the whole diet. *J. Clin. Invest.* 53: 247, 1974.
 15. BJORN-RASMUSSEN, E., L. HALLBERG, B. MAGNUSSON, L. ROSSANDER, B. SVANBERG AND B. ARVIDSSON. Measurement of iron absorption from composite meals. *Am. J. Clin. Nutr.* 29: 772, 1976.
 16. COOK, J. D., AND E. R. MONSEN. Food iron absorption in human subjects. III. Comparison of the effects of animal protein on nonheme iron absorption. *Am. J. Clin. Nutr.* 29: 859, 1976.
 17. BJORN-RASMUSSEN, E., L. HALLBERG AND R. B. WALKER. Food iron absorption in man. II. Isotopic exchange of iron between labeled foods and between a food and an iron salt. *Am. J. Clin. Nutr.* 26: 1311, 1973.
 18. BJORN-RASMUSSEN, E. Food iron absorption in man. V. Validity of the extrinsic tag two-pool method for measurement of dietary nonheme iron absorption in patients with various clinical disorders. *Scand. J. Gastroenterol.* 8: 645, 1973.
 19. LAYRISSE, M., C. MARTINEZ-TORRES, M. RENZY AND I. LEETS. Ferritin iron absorption in man. *Blood* 45: 689, 1975.
 20. COOK, J. D., V. MINNICH, C. V. MOORE, A. RASMUSSEN, W. B. BRADLEY AND C. A. FINCH. Absorption of fortification of iron in bread. *Am. J. Clin. Nutr.* 26: 861, 1973.
 21. ELWOOD, P. C., W. E. WATERS AND P. SWEETNAM. The haematinic effect of iron in flour. *Clin. Sci.* 40: 31, 1971.
 22. LAYRISSE, M., C. MARTINEZ-TORRES AND M. ROCHE. Effect of interaction of various foods on iron absorption. *Am. J. Clin. Nutr.* 21: 1175, 1968.
 23. SAYERS, M. H., S. R. LYNCH, P. JACOBS, R. W. CHARLTON, T. H. BOTHWELL, R. B. WALKER AND F. MAYETT. The effects of ascorbic acid supplementation on the absorption of iron in maize, wheat, and soya. *Brit. J. Haematol.* 24: 209, 1973.
 24. BJORN-RASMUSSEN, E., AND L. HALLBERG. Iron absorption from maize. *Nutr. Metabol.* 16: 94, 1974.
 25. COOK, J. D., AND E. R. MONSEN. Vitamin C, the common cold, and iron absorption in man. *Am. J. Clin. Nutr.* 30: 235, 1977.
 26. DISLER, P. B., S. R. LYNCH, R. W. CHARLTON, J. D. TORRANCE, T. H. BOTHWELL, R. B. WALKER AND F. MAYET. The effect of tea on iron absorption. *Gut* 16: 193, 1975.
 27. MOORE, C. V., AND R. DUBACH. Observations on the absorption of iron from foods tagged with radioiron. *Trans. Assoc. Am. Phys.* 64: 245, 1951.
 28. BJORN-RASMUSSEN, E. Iron absorption from wheat bread—influence of various amounts of bran. *Nutr. Metabol.* 16: 101, 1974.
 29. MONSEN, E. R., AND J. D. COOK. Food iron absorption in human subjects. IV. The effects of calcium and phosphate salts on the absorption of nonheme iron. *Am. J. Clin. Nutr.* 29: 1142, 1976.
 30. COOK, J. D., AND E. R. MONSEN. Food iron absorption in human subjects. II. The effect of EDTA on absorption of dietary nonheme iron. *Am. J. Clin. Nutr.* 29: 614, 1976.

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ALLEY AND J. A. menstrual blood 1: 739, 1961. L. L. NILSSON AND loss—a population

post-testing, using the same questionnaire.

Various personal characteristics of the participants, as well as data relating to the diabetic state, were also examined for possible relationships to test scores and to one another.

References

- (1) STUCKY, V.T.: Tape recording, booklet teach diets. *Hospitals* 42: 78 (Dec. 16), 1968.
- (2) McDONALD, G.W.: The diabetes supplement of the National Health Survey. 1. Introduction and overview. *J. Am. Dietet. A.* 52: 118, 1968.
- (3) ETZWILER, D.D.: Developing a regional program to help patients with diabetes. *J. Am. Dietet. A.* 52: 394, 1968.
- (4) STULB, S.C.: The diabetes supplement of the National Health Survey. 4. The patient's knowledge of the food exchanges. *J. Am. Dietet. A.* 52: 391, 1968.
- (5) HOLLAND, W.H.: The diabetes supplement of the National Health Survey. 3. The patient reports on his diet. *J. Am. Dietet. A.* 52: 387, 1968.
- (6) ROBINSON, C., AND LAWLER, M.R.: Normal and Therapeutic Nutrition. 14th ed. N.Y.: Macmillan Co., 1972.
- (7) GORMICAN, A.: Controlling Diabetes with Diet. Springfield, Ill.: Charles C Thomas, 1971.
- (8) McDONALD, G.W., AND O'SULLIVAN, J.B.: Screening for diabetes mellitus. In Fajans, S.S., and Sussman, K.E., eds.: Diabetes Mellitus: Diagnosis and Treatment. Vol. III. N.Y.: Am. Diab. Assoc., 1971.
- (9) ETZWILER, D.D.: Developing a System for Patient Education. A speech to Post-graduate Conf. on Diabetes, Diabetes Assoc. of the Cincinnati Area, May 23, 1973.
- (10) ETZWILER, D.D.: The patient is a member of the medical team. *J. Am. Dietet. A.* 61: 421, 1972.
- (11) KAUFMAN, M.: Many dimensions of diet counseling for diabetics. *Am. J. Clin. Nutr.* 15: 45, 1964.
- (12) YOUNG, M.A.C., BUCKLEY, P.H., WECHSLER, H., AND DEMONE, H.W., JR.: A demonstration of automated instruction for diabetic self care. *Am. J. Pub. Health* 59: 110, 1969.
- (13) REARDON, E.: Can sub-professionals assist in teaching patients with diabetes mellitus? *J. Am. Dietet. A.* 52: 405, 1968.
- (14) TRAYSER, L.M.: A teaching program for diabetics. *Am. J. Nurs.* 73: 92, 1973.
- (15) HENDERSON, S.E.: Dietetics and human ecology. *Hospitals* 44: 78 (July 16), 1970.
- (16) PHARAYIL, P.: Patients learn how to calculate special diets in hospital classes. *Mod. Hosp.* 112: 100 (June), 1969.
- (17) OHLSON, M.A.: Suggestions for research to strengthen learning by patients. *J. Am. Dietet. A.* 52: 401, 1968.
- (18) ECHOLS, I.J.: Comparative group approaches. *J. Am. Dietet. A.* 59: 460, 1971.
- (19) HAYS, W.: Statistics for Psychologists. N.Y.: Holt, Rinehart, & Winston, 1963.

Analyzed vs. calculated values

Composition of diets containing 25 and 35 per cent calories from fat

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Can nutrient values in food composition tables be used with assurance in calculating diets?

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The National Diet-Heart study showed that dietary studies with "free-living" participants are feasible and capable of yielding important information on the effect of diet in reducing risks, such as elevated blood cholesterol associated with increased incidence of coronary heart disease (1, 2).

Consequently, several groups have recommended dietary intervention to reduce the risks of heart disease. Changes recommended by the Inter-Society Commission for Heart Disease Resources (ICHDR) (3) include: (a) reducing total fat intake to supply 35 per cent or less of total calories; (b) maintaining a daily intake of less than 10 per cent of total calories from saturated fats and up to 10 per cent from polyunsaturated fats, with the remainder of fat supplied by monounsaturated fats; and (c) maintaining a daily intake of less than 300 mg. cholesterol. The Food and Nutrition Board of the National Academy of Sciences-National Research Council and the Council on Foods and Nutrition of the American Medical Association, in a joint pronouncement on "Diet and Coronary Heart Disease" (4), have also supported such dietary intervention, as has the Council on Nutrition of the American Heart Association (5). The former

group included recommendations (4) for the production of modified foods low in fat and cholesterol to help meet the requirements of fat-controlled diets.

Although the recommended reductions in dietary fat of the ICHD were modest—from 45 to 35 per cent—the recommendation for reducing cholesterol from 600 to 1,000 mg. in average American diets to a level of 300 mg. or less was more drastic. However, Friend, in a report outlining the nutrients available in the food supply for civilian consumption per capita per day, pointed out (6) that the fatty acid content of the food supply has changed. Shifts in food consumption have resulted in a marked rise in the consumption of polyunsaturated fat (linoleic acid) with the level rising steadily—due to greater use of salad and cooking oils, margarine, and shortening—from 15 to 24 gm. per capita per day in twenty-five years. Thus, the polyunsaturated fat in the food supply was actually closer to the ICHD recommendation (up to 10 per cent of calories) than was known when the recommendations were made.

In our laboratory, we conducted a dietary study to test the effect of some of the ICHD recommendations on blood lipids and other parameters of persons in a "free-living" situation by feeding diets low in fat and cholesterol, using U.S. foods commonly available in the open market and easily prepared at home.

This is not a report of the results of that study—rather, of what the participants ate, i.e., the determined composition of the diets. The preparation of the meals in our laboratory afforded an opportunity to assess the validity of data from calculated diets by comparing determined nutrient content of homogenates of meals with the composition of foods calculated from data in Agriculture Handbook No. 8 (7). The results allowed us to assess the degree of confidence that can be placed in calculated data when they are used to correlate nutrient intakes with such parameters as blood lipids. Further, in keeping with the recommendations of the Food and Nutrition Board and Council on Foods and Nutrition of the American Medical Association to insure that dietary advice does not compromise the intake of essential nutrients, the determined nutrient composition of the diets was compared with the 1974 Recommended Dietary Allowances (8).

Results of the study, including the effects of the diets on the various metabolic parameters measured, are reported in separate papers.

Procedures

Briefly, the study was divided into two main periods of forty days each. During the first period, twenty-one healthy volunteers, ten men and eleven women, fifty to sixty years old, were fed diets containing 25 per cent calories from fat, 60 per cent calories from carbohydrate, and less than 300 mg. cholesterol. During the second period, the same subjects received diets containing 35 per cent calories from fat and 50

per cent calories from carbohydrate, with an average of about 300 mg. cholesterol, depending on individual caloric intake, with an upper limit of 500 mg. cholesterol in the 3,600-kcal meals. Protein was maintained at 15 per cent of calories, simple (mono- and di-saccharides) and complex (poly-saccharides) carbohydrates in a ratio of about 1 (0.8 to 1.2), and the ratio of polyunsaturated fat (linoleic acid) to saturated fat (total saturated acids) at 1 (0.8 to 1.2).

Ten menus, with caloric levels varying from 1,600 to 2,800 kcal for the 25-per-cent-fat-calorie diets, were used during the first forty-day period. For the second period, the same menus were modified to contain 35 per cent calories from fat, with caloric levels varying from 1,600 to 3,600. During each period, the ten menus were fed for four (ten-day) cycles. Caloric levels varied in increments of 400 kcal. Thus during the 35-per-cent-fat-calorie period, sixty menus were calculated to meet individual needs. Caloric intakes were adjusted throughout the study to maintain body weights, since loss of body weight causes a reduction in blood lipids. Sample menus for two days are shown in Table 1. All ten menus were described in another report in this series (9).

The nature and rationale of formulating fat-controlled diets have been discussed (10). Diets low in total and saturated fat and cholesterol must contain a high proportion of fruits, vegetables, and cereal grains—all cholesterol-free and containing little or no fat. Meat, such as beef and pork, must be lean and/or trimmed of visible fat; fish and poultry are used frequently. Our diets conformed to these general principles. Eggs were used sparingly in the 25-per-cent-fat-calorie meals, but two to three eggs a week were used in the 35-per-cent-fat-calorie diets. Two per cent fat milk and "soft" margarine—both usually recommended for fat-controlled diets to reduce saturated fat—were used. However, these foods have been readily available to consumers for more than a decade and, for this study, are not considered as special foods. In the 35-per-cent-fat-calorie diets, both 2 per cent and whole milk were used, as well as some bacon and sausage for the breakfast meals.

The meals were readily accepted by participants, and most said they would volunteer to eat them again.

When possible, meat and chicken breasts were purchased from a single retail market where the butcher prepared individual portions of such items as steaks, pork chops, and chicken breasts. Approximate yield on cooking was taken into account when individual servings were ordered. Visible fat and bone were trimmed from the portions, and skin was removed from chicken breasts. Frozen fish was purchased; after thawing, allowance was made in weights of portions to correct for cooking losses. Fat was trimmed from round roasts and ham prior to cooking, and individual portions were cut and weighed after cooking. Turkey roll, commercially prepared from all white meat, was heated before individual portions were cut

Table 1: Examples of menus planned to contain 25 and 35 per cent calories from fat with low cholesterol content and a P:S ratio of 1*

food item	measure	weight	25 per cent calories from fat		35 per cent calories from fat	
			2,000 kcal	2,800 kcal	2,000 kcal	2,800 kcal
Day No. 1						
BREAKFAST						
apple juice	8 oz.	248	8 oz.	8 oz.	4 oz.	8 oz.
Rice Krispies or Cream of Rice	1 c.	25	25 gm.	25 gm.	25 gm.	25 gm.
toast, white	1 sl.	22	2 sl.	2 sl.	2 sl.	2 sl.
total soft margarine†	1 tsp.	4.6	23 gm.	37 gm.	23 gm.	37 gm.
jelly	1 Tbsp.	18	2 Tbsp.	1 Tbsp.	1 Tbsp.	1 Tbsp.
total sugar‡	1 tsp.	3.6	4 tsp.	5 tsp.	4 tsp.	4 tsp.
2 per cent milk	8 oz.	246	8 oz.	8 oz.	—	—
whole milk	8 oz.	244	—	—	4 oz.	4 oz.
cream, half and half	1 Tbsp.	15	—	—	—	1 Tbsp.
bacon, broiled	1 sl.	7.5	—	—	1 sl.	3 sl.
coffee-tea, <i>ad libitum</i>						
NOON MEAL						
beef, round, cubes	1 oz.	30	90 gm.	90 gm.	60 gm.	90 gm.
noodles	1 c.	160	80 gm.	80 gm.	80 gm.	80 gm.
whole kernel corn, frozen	—	—	100 gm.	100 gm.	—	100 gm.
broccoli, frozen	—	—	—	—	125 gm.	—
salad:						
apple, diced	—	—	50 gm.	50 gm.	50 gm.	50 gm.
carrots, grated	—	—	25 gm.	25 gm.	25 gm.	25 gm.
raisins	—	—	20 gm.	20 gm.	20 gm.	20 gm.
mayonnaise	1 Tbsp.	15	1 Tbsp.	1 Tbsp.	1 Tbsp.	1 Tbsp.
lettuce leaf	1	25	25 gm.	25 gm.	25 gm.	25 gm.
bread, white	1 sl.	22	1 sl.	2 sl.	1 sl.	1 sl.
gingerbread	1 pc.	63	63 gm.	63 gm.	63 gm.	63 gm.
pears, water packed‡	—	—	—	—	—	—
pears, with sirup	—	—	—	127 gm.	—	127 gm.
corn oil (for beef)	1 tsp.	4.6	—	—	2 tsp.	2 tsp.
cream, half and half	1 Tbsp.	15	—	—	1 Tbsp.	—
coffee-tea, <i>ad libitum</i>						
EVENING MEAL						
pork chops (muscle only)	1 oz.	30	60 gm.	96 gm.	60 gm.	96 gm.
spiced apple ring	—	—	50 gm.	50 gm.	50 gm.	50 gm.
rice	1 c.	205	102 gm.	250 gm.	—	—
sauerkraut	1 c.	235	—	235 gm.	117 gm.	235 gm.
mixed vegetables, frozen	—	—	100 gm.	100 gm.	100 gm.	100 gm.
lettuce wedge	—	—	150 gm.	150 gm.	150 gm.	150 gm.
oil and vinegar dressing#	1 Tbsp.	15	1 Tbsp.	1 Tbsp.	2 Tbsp.	2 Tbsp.
bread, rye	1 sl.	25	—	2 sl.	—	2 sl.
angel food cake	—	—	—	53 gm.	—	53 gm.
strawberries, frozen	1 c.	149	149 gm.	149 gm.	149 gm.	149 gm.
2 per cent milk	8 oz.	244	—	—	8 oz.	6 oz.
cream, half and half	1 Tbsp.	15	—	—	—	—
coffee-tea, <i>ad libitum</i>						

*All food items were either weighed or measured. Measures were converted to gram weights as indicated for calculating nutrient content.

†Margarine was weighed and sugar was allocated for individuals on a daily basis. Both were provided with breakfast to be distributed over the menus for each day.

‡Served with lower caloric level.

#Two tsp. cottonseed oil plus 1 tsp. vinegar.

Table 1: Concluded

food item	measure	weight	25 per cent calories from fat		35 per cent calories from fat	
			2,000 kcal	2,800 kcal	2,000 kcal	2,800 kcal
<i>gm.</i>						
Day No. 3						
BREAKFAST						
pineapple juice	8 oz.	249	8 oz.	8 oz.	8 oz.	8 oz.
corn cereal, flakes	1 c.	25	25 gm.	25 gm.	25 gm.	25 gm.
toast, white	1 sl.	22	2 sl.	2 sl.	1 sl.	2 sl.
total soft margarine†	1 tsp.	4.6	23 gm.	41 gm.	32 gm.	46 gm.
jelly	1 Tbsp.	18	1 Tbsp.	2 Tbsp.	1 Tbsp.	2 Tbsp.
total sugar†	1 tsp.	3.6	21 gm.	21 gm.	5 tsp.	6 tsp.
2 per cent milk	8 oz.	246	8 oz.	8 oz.	—	—
whole milk	8 oz.	244	—	—	4 oz.	4 oz.
whole egg (hard cooked)	1	50	—	—	1	1
cream, half and half	1 Tbsp.	15	—	—	—	1 Tbsp.
coffee-tea, <i>ad libitum</i>						
NOON MEAL						
baked filet haddock	1 oz.	30	60 gm.	90 gm.	78 gm.	60 gm.
with tomato sauce	½ c.	121	121 gm.	121 gm.	121 gm.	121 gm.
lemon wedge	—	10	10 gm.	10 gm.	10 gm.	10 gm.
parsley boiled potato	—	—	122 gm.	122 gm.	—	—
baked potato	—	—	—	—	122 gm.	122 gm.
spinach, frozen	—	—	—	90 gm.	90 gm.	90 gm.
sliced beets	½ c.	123	123 gm.	246 gm.	123 gm.	123 gm.
cole slaw:						
cabbage	1 c.	90	90 gm.	90 gm.	90 gm.	90 gm.
vinegar	1 Tbsp.	15	1 Tbsp.	1 Tbsp.	1 tsp.	1 tsp.
sugar	1 tsp.	3.6	1 tsp.	1 tsp.	1 tsp.	1 tsp.
mayonnaise	1 Tbsp.	15	—	—	1 Tbsp.	1 Tbsp.
gelatin-cherry	—	—	65 gm.	65 gm.	65 gm.	65 gm.
banana	—	—	—	175 gm.	—	175 gm.
bread, whole wheat	1 sl.	25	1 sl.	2 sl.	1 sl.	2 sl.
chocolate chip cookie	1	10	30 gm.	30 gm.	10 gm.	20 gm.
corn oil (for fish)	1 tsp.	4.6	—	—	1 tsp.	2 tsp.
2 per cent milk†	8 oz.	246	—	—	—	—
coffee-tea, <i>ad libitum</i>						
EVENING MEAL						
roast beef (round)	1 oz.	30	60 gm.	120 gm.	60 gm.	120 gm.
whipped potato	½ c.	100	—	—	100 gm.	100 gm.
baked potato	1	99	99 gm.	99 gm.	—	—
asparagus, frozen	—	—	122 gm.	122 gm.	122 gm.	122 gm.
salad:						
tomato wedges	—	—	200 gm.	200 gm.	200 gm.	200 gm.
lettuce leaf	1	25	25 gm.	25 gm.	25 gm.	25 gm.
mayonnaise	1 Tbsp.	15	1 Tbsp.	1 Tbsp.	1 Tbsp.	1 Tbsp.
pan roll	1	28	2 rolls	2 rolls	1 roll	2 rolls
fruit cocktail, water pack	—	—	100 gm.	—	100 gm.	—
fruit cocktail with sirup	—	—	—	256 gm.	—	—
cream, half and half	1 Tbsp.	15	—	—	1 Tbsp.	—
2 per cent milk	—	—	—	—	6 oz.	8 oz.
oatmeal cookie with raisin	1	14	28 gm.	28 gm.	28 gm.	28 gm.
coffee-tea, <i>ad libitum</i>						

and weighed. The same brand of milk was used throughout. Both the 2 per cent fat and whole milk were fortified with vitamins A and D.

The same brands of meat, packaged and canned foods, bread, rolls, corn muffins, cookies, cereals, and frozen vegetables and fruits were used with few exceptions. Fresh vegetables and fruits were purchased as needed to assure maximum freshness. One brand of soft margarine, selected for its high alpha-tocopherol and linoleic acid contents (11) and fortified with vitamin A, was used throughout.

Bread and rolls were served as purchased. Although the same brands were used as much as possible, bread slices varied 2 to 3 gm. and rolls up to 6 gm. each.

Table salt was not restricted. Salt shakers were weighed and furnished to individuals for their use with meals. The shakers were reweighed periodically, and individual salt intakes were calculated. In general, a minimal amount of salt was added to vegetables during cooking. No salt was added to meat during cooking, except when called for in a recipe, such as for meatballs.

Cooked food was served within 1 hr. after cooking. Meals taken for analysis were sampled at random times throughout the serving period.

PREPARATION OF DIETS FOR ANALYSIS. During the first forty-day period (meals containing 25 per cent calories from fat), two trays containing each meal for each day of the 2,000-kcal diet were saved and later analyzed. During the second period (meals containing 35 per cent calories from fat), one tray of each meal for the 2,800-kcal diet was sampled for later analysis. Meals of these two caloric levels were selected because they were most representative of the caloric intakes at the start of the test periods.

After removal of inedible material, such as apple cores, banana peels, and so forth, the three meals with beverages, except coffee and tea, were accumulated for each day in a weighed plastic box, sealed with freezer tape, and frozen at -20°C . or below. They were then reweighed, resealed, and kept frozen until prepared for analysis. The meals were subsequently partly thawed, transferred to a heavy-duty blender, and homogenized with a small amount of glass redistilled water. The homogenates were transferred quantitatively to weighed stainless steel pans, refrozen at -50°C . in a commercial freeze-dryer, and dried under vacuum at -20°C .

On removal from the freeze-dryer, homogenates were weighed, pulverized, and kept at -40°C . in sealed plastic bags. Prior to analysis, an aliquot of 25 per cent by weight of each day's freeze-dried homogenate was combined by days for each cycle within each fat-calorie period. For example, for the 25-per-cent-fat-calorie period, aliquots for Day 2 for each of the four cycles were combined. These combinations resulted in twenty pooled samples representing homoge-

nates of nearly all meals fed throughout the study. Finally, samples from Day 4 of all four cycles fed during the 35-per-cent-fat-calorie period were analyzed individually to assess the variation in the same menus served during each cycle. Samples from meals for seven of the seventy-nine days were omitted from the pooled homogenates for various reasons. Three of these were combinations of meals that were changed in an emergency due to a power failure.

ANALYSIS OF DIETS. The frozen pooled homogenates were shipped to an analytical laboratory for analysis and kept frozen until analyzed². Calories, proximate composition, ten vitamins, fourteen minerals and trace minerals, and cholesterol were determined. In addition, alpha-, gamma-, and delta-tocopherols; fatty acids; phospholipids; and sterol composition were determined in the Lipid Nutrition Laboratory at Beltsville. (All of the Beltsville data will be reported in detail in separate papers.)

Proximate analysis of the freeze-dried homogenates, including residual moisture, total fat (acid hydrolysis), total protein ($\text{N} \times 6.25$), crude fiber, and ash were determined by official methods of the Association of Official Analytical Chemists (12). Carbohydrate was calculated by difference.

The following vitamins were analyzed by methods of the Association of Official Analytical Chemists (11th edition—except folic acid, 8th edition): niacin, with *L. plantarum* (turbidimetric); folic acid, with *Streptococcus faecalis* (titrimetric); and riboflavin, with *L. casei* (turbidimetric). Thiamin was determined fluorometrically. Preformed vitamin A (official method for mixed feeds) and beta-carotene were analyzed separately; colorimetric measurements were made after chromatographic separation of carotene from vitamin A. These data were combined to calculate total vitamin A activity.

Additional vitamins determined were: biotin (13), using *L. plantarum*; pantothenic acid (double-enzyme system) (14); vitamin B₁₂ (15), using *L. leichmannii*; vitamin B₆ (16), with *S. carlsbergensis*; and ascorbic acid, reduced and dehydroascorbic acid (17). The latter two were combined and reported as total ascorbic acid.

Major mineral elements determined were: calcium, phosphorus, iron, sodium, potassium, and magnesium—all by direct reading emission spectroscopy.

Trace minerals analyzed, also by emission spectroscopy, were: copper, manganese, zinc, chromium, aluminum, barium, boron, and strontium.

Total cholesterol was analyzed by gas-liquid chromatography after acid hydrolysis and extraction of the samples (12).

Although data were obtained for meals from each

²Laboratory analyses were made by the Wisconsin Alumni Research Foundation Institute, Inc., Madison, working under contract with the Lipid Nutrition Laboratory, Nutrition Institute, Agricultural Research Service, U.S. Department of Agriculture.

Table 2: Nutrients in diets designed to contain 25 and 35 per cent calories from fat: Averages for ten days for various caloric levels

constituents	calculated*				analyzed*			
	1,600 kcal	2,000 kcal	2,400 kcal	2,800 kcal	3,200 kcal	3,600 kcal	2,000 kcal	2,800 kcal
Diets containing 25 per cent calories from fat†								
food energy (kcal)	1,664	2,027	2,449	2,863			1,878 (2,052)	
carbohydrate (gm.)	251.3	306.1	366.0	423.1			273.0	
per cent of calories	60.4	60.4	59.8	59.7			58.1	
protein (gm.)	67.8	79.6	94.9	110.4			75.3	
per cent of calories	16.3	15.7	15.5	15.6			16.0	
fat (gm.)	47.4	58.6	72.7	84.3			53.8	
per cent of calories	25.4	26.0	26.7	26.7			25.9	
saturated acids (gm.)	13	15	19	22			11‡	
per cent of calories	7.0	6.7	7.0	7.0			5.2	
oleic acid (gm.)	17	22	28	32			14‡	
per cent of calories	9.2	9.8	10.3	10.1			6.9	
linoleic acid (gm.)	12	15	18	20			12‡	
per cent of calories	6.5	6.7	6.6	6.3			5.6	
P:S ratio#	0.92	1.0	0.95	0.91			1.08	
cholesterol (mg.)	140	164	205	236			141	
Diets containing 35 per cent calories from fat‡								
food energy (kcal)	1,750	2,135	2,397	2,830	3,261	3,651	2,594 (2,854)	
carbohydrate (gm.)	212.1	260.6	289.2	348.7	417.4	451.9	314.9	
per cent of calories	48.5	48.8	48.3	49.3	51.2	49.5	48.4	
protein (gm.)	70.3	85.0	96.2	112.5	125.1	139.0	108.5	
per cent of calories	16.1	15.9	16.0	15.9	15.3	15.2	16.7	
fat (gm.)	73.0	88.1	99.5	114.8	126.7	148.9	100.6	
per cent of calories	32.6	37.1	37.3	36.5	35.0	36.7	34.9	
saturated acids (gm.)	19	23	26	31	34	40	20‡	
per cent of calories	9.8	9.7	9.8	9.8	9.4	9.9	7.1	
oleic acid (gm.)	25	31	36	41	47	54	25‡	
per cent of calories	12.8	13.1	13.5	13.0	13.0	13.3	8.7	
linoleic acid (gm.)	20	24	26	29	32	38	21‡	
per cent of calories	10.3	10.1	9.8	9.2	8.8	9.4	7.3	
P:S ratio#	1.05	1.04	1.0	0.94	0.94	0.9	1.04	
cholesterol (mg.)	221	248	277	317	343	376	236	

*Percentages may not add to 100.0 due to rounding.

†2,000-kcal meals analyzed.

‡Analyzed by different method from that for total fat; see text.

#Linoleic acid:saturated acids.

‡2,800-kcal meals analyzed.

day, only the values for the ten-day averages are presented for total saturated fatty acids and oleic and linoleic acids, determined by gas-liquid chromatography at Beltsville. The P:S ratios were calculated from the analyzed samples. Details of methodology and daily variations of the fatty acids will be presented in a separate publication.

Tocopherols were analyzed by gas-liquid chromatography (18) after extraction of the homogenates with ethanol, saponification in the presence of pyrogallol, and purification by thin layer chromatography prior to preparing trimethylsilyl ethers. Ten-day averages also are presented for the tocopherols. Further details of these results will be reported later.

Results and discussion

Table 2 shows the composition of food energy and carbohydrate, protein, fat, and cholesterol as calculated from Agriculture Handbook No. 8 (7) for the different caloric levels and as analyzed in the 2,000

and 2,800-kcal levels of the 25-per-cent- and 35-per-cent-fat-calorie diets, respectively. The computer tape, No. 8-1-1 (19), was used for tabulating data from the 1963 edition of Agriculture Handbook No. 8 as revised recently specifically to update fat and cholesterol data. Basic food item codes in the Handbook as modified were followed in consideration of the type of fat in some foods, particularly baked goods (19).

Table 3 shows the comparisons of the analyzed and calculated data for the ten-day averages of diets containing 25 and 35 per cent calories from fat. These comparisons include data for food energy, proximate composition, fatty acids, and cholesterol. In general, analyzed and calculated data are close. There is no ready explanation for the consistently lower values for analyzed diets, but none of the differences shown in this table is considered of great importance except cholesterol in the 35-per-cent-fat-calorie diets. Data for calculated cholesterol are based on modified and updated data on tape (20) and reflect recent analyses of

Table 3: Analyzed and calculated composition of daily diets containing 25 and 35 per cent calories from fat: Ten-day averages of proximate components, fatty acids, and cholesterol

constituent	25 per cent calories from fat*			35 per cent calories from fat†		
	analyzed‡	calculated#	per cent difference¶	analyzed‡	calculated#	per cent difference¶
Proximate components, fatty acids, and cholesterol						
food energy						
calculated (kcal)**	1,878 ± 20††	2,027 ± 25	- 7.4	2,594 ± 39	2,830 ± 35	- 8.3
combustion (kcal)‡‡	2,052 ± 23	—	—	2,854 ± 36	—	—
total protein (gm.)	75.3 ± 1.6	79.6 ± 1.9	- 5.4	108.5 ± 3.9	112.5 ± 1.9	- 3.6
carbohydrate (gm.)	273.0 ± 3.0	306.1 ± 4.8##	-10.8	314.9 ± 6.2	348.7 ± 6.3##	- 9.7
total fat (gm.)	53.8 ± 1.5	58.6 ± 3.0	- 8.2	100.6 ± 1.8	114.8 ± 2.8	-12.4
saturated acids (gm.)	10.8 ± 0.5	15.2 ± 0.7	¶¶	20.4 ± 0.6	31.2 ± 1.0	¶¶
oleic acid (gm.)	14.3 ± 0.5	22.1 ± 0.9	—	25.0 ± 0.8	41.3 ± 1.1	—
linoleic acid (gm.)	11.6 ± 0.5	14.7 ± 1.3	—	21.1 ± 1.0	29.4 ± 1.4	—
P/s ratio	1.08 ± 0.04	0.96 ± 0.07	—	1.04 ± 0.06	0.95 ± 0.05	—
cholesterol (mg./gm. fat)	2.63 ± 0.14	2.84 ± 0.13	- 6.1	2.36 ± 0.32	2.76 ± 0.28	-14.5
total cholesterol (mg.)	141 ± 7	164 ± 6	-14.0	236 ± 31	317 ± 33	-25.6
crude fiber (gm.)	5.8 ± 0.3	6.7 ± 0.2	-13.4	7.4 ± 0.4	7.7 ± 0.2	- 3.9
ash (gm.)	14.9 ± 0.5	16.1 ± 0.4	- 7.5	20.1 ± 1.1	21.5 ± 0.4	- 6.5
total moisture (%)	76.6 ± 0.5	—	—	75.4 ± 0.4	—	—
average weight (gm., 3 meals/day)	1,814 ± 43	—	—	2,248 ± 54	—	—

*2,000-kcal meals analyzed.

†2,800-kcal meals analyzed.

‡Average results of laboratory analyses of homogenates for ten menus.

#Average results of data calculated from Agriculture Handbook No. 8 for ten menus.

¶analyzed - calculated
calculated × 100.

**Sum of calories from protein, fat, and carbohydrate calories, using the factors 4, 9, 4 applied to contents after analysis of protein, fat, and calculation of carbohydrate by difference.

††Mean ± standard error.

‡‡Obtained after bomb calorimetry.

##Calculated ratio simple:complex carbohydrates averaged over ten days for 25 per cent fat calories was 1.10 ± 0.08; for 35 per cent fat calories, 1.02 ± 0.07.

¶¶Fatty acids determined at Beltsville. Per cent differences between analyzed and calculated values were not calculated because the methods for determining total fat were different—acid hydrolysis by WARF vs. chloroform:methanol extraction used at Beltsville. The latter method produced lower values for total fat.

some food items by gas-liquid chromatography (21). Cholesterol values in some foods are lower than earlier data in Agriculture Handbook No. 8, which reflected total sterols and not just cholesterol.

The small differences in total fat content (Table 3) in the analyzed and calculated diets were probably due to use of the leanest meat available to us; most of the visible fat was trimmed prior to cooking.

Crude fiber in the diets per day was 5.8 gm. in the 25-per-cent-fat-calorie diet with 2,000 kcal, and 7.4 gm. in the 35-per-cent-fat-calorie diet with 2,800 kcal. In the average American diet, as calculated from nutrients available for civilian consumption in 1970, daily crude fiber was 4.3 gm. for diets with food energy value of about 3,300 kcal (22).

In Table 4, analyzed and calculated values are shown also for minerals and vitamins listed in Table 1 of Agriculture Handbook No. 8. Again, analyzed and calculated values are closer than anticipated in view of the well known variations in nutrient content due to source of food items, cooking methods, season, and length of storage and processing time, particularly of fruits and vegetables (23-27). Geographic location may be a particularly important source of varia-

tion in mineral content (26, 28-30). Whether the differences are due to variety, cultivation practices, or soil is not known.

For the major minerals, comparisons showed the greatest differences for calcium and phosphorus, explainable in part by the method of analysis. Murphy *et al.* reported (31) that analyses of Type A school lunches for calcium by atomic absorption spectroscopy yielded data 14 per cent higher than values based on analyses by emission spectroscopy. However, the nature and composition of the food in the homogenate may also influence the results of analyses. Published data (32, 33) agreed well with results of mineral analyses from chemical, spectrometric, and atomic absorption procedures, except for calcium, magnesium, iron, and copper where a greater variation was found for some materials. Recently published data of Watt *et al.* (34) show that copper values obtained by emission spectroscopy are more apt to be low, compared with other methods than for other elements studied to date. Differences in sodium may be explained by the addition of salt to foods during cooking and by the use of margarine. In addition, variations in the sodium in such food items as dairy prod-

Table 4: Analyzed and calculated composition of daily diets containing 25 and 35 per cent calories from fat: Ten-day averages for minerals and vitamins

constituent	25 per cent calories from fat*				35 per cent calories from fat†			
	analyzed‡	calculated#	per cent difference¶	analyzed per 1,000 kcal	analyzed‡	calculated#	per cent difference¶	analyzed per 1,000 kcal
Minerals								
calcium (mg.)	450 ± 21	742 ± 15	-39.4	219	724 ± 53	1,061 ± 57	-31.8	254
phosphorus (mg.)	895 ± 15	1,207 ± 10	-25.8	436	1,357 ± 59	1,725 ± 50	-21.3	475
iron (mg.)	13.6 ± 0.9	14.5 ± 0.5	- 6.2	6.6	18.6 ± 2.1	18.1 ± 0.8	+ 2.8	6.5
sodium (mg.)	2,784 ± 190	2,258 ± 147	+23.3	1,357	4,007 ± 248	3,325 ± 181	+20.5	1,404
potassium (mg.)	3,992 ± 115	3,603 ± 136	+10.8	1,945	4,985 ± 190	4,543 ± 177	+ 9.7	1,747
Vitamins								
vitamin A (i.u.)								
total	8,298 ± 805	8,046 ± 815	+ 3.1	4,044	12,426 ± 972	10,749 ± 1,018	+15.6	4,354
preformed								
vitamin A	2,635 ± 222	—	—	1,284	3,133 ± 383	—	—	1,098
beta-carotene	5,663 ± 777	—	—	2,760	9,293 ± 1,041	—	—	3,256
thiamin (mg.)	1.65 ± 0.13	1.45 ± 0.08	+13.8	0.80	2.09 ± 0.16	1.87 ± 0.11	+11.8	0.73
riboflavin (mg.)	1.48 ± 0.05	1.53 ± 0.03	- 3.3	0.72	1.98 ± 0.09	2.13 ± 0.08	- 7.0	0.69
niacin (mg.)**	23.9 ± 1.2	21.1 ± 0.8	+13.3	11.6	31.5 ± 1.6	27.3 ± 0.91	+15.4	11.0
ascorbic acid (mg.)	144 ± 15	183 ± 20	-21.3	70	181 ± 18	224 ± 25	-19.2	63

*2,000-kcal meals analyzed.

†2,800-kcal meals analyzed.

‡Average results of laboratory analyses of homogenates for ten menus.

#Average results of data calculated from Agriculture Handbook No. 8 for ten menus.

¶analyzed - calculated
calculated × 100.

||Mean ± standard error.

**Values for niacin represent preformed vitamin without equivalents from tryptophan.

ucts (35) added to the 35-per-cent-fat-calorie diets may explain further variations. Values for calcium and magnesium in fluid milk analyzed by atomic absorption, reported by Feeley *et al.* (35), were lower than some values reported by others.

Except for ascorbic acid, values for vitamins obtained by analysis and calculation were not too different. It is not known whether the losses of ascorbic acid occurred during cooking, homogenizing, storage prior to homogenizing, or analysis, or whether geographic or seasonal variations were responsible. Although cooking methods alone may explain variation in ascorbic acid content (36), the differences are not considered of sufficient magnitude to invalidate the calculated data.

Table 5 shows values for additional minerals and vitamins that were determined but not calculated prior to analysis. We are aware of some serious problems encountered in analyzing trace minerals, particularly chromium (37). Wide variations in aluminum content of meals have been reported (38). Losses from cooking and processing, although known to occur (39, 40), constitute important missing information in our study.

Menus were designed to meet the 1968 Recommended Dietary Allowances (41) for the reference man as calculated from the nutrients in Agriculture Handbook No. 8. As expected, the analyzed nutrients in the 2,800-kcal level of the 35-per-cent-fat-calorie diet more nearly approximated the allowances for the major nutrients than did the 2,000-kcal diet with 25 per cent fat calories. Magnesium, zinc, copper, fola-

cin, pantothenic acid, and biotin in both the 25- and 35-per-cent-fat-calorie diets as analyzed were lower than either recommended levels or those in average American diets. However, when analyzed values were recalculated on a per-1,000-kcal basis (Tables 3, 4, and 5) and on the basis of recommended caloric levels for adult men and women (Table 6), there were only small differences, except for calcium and vitamin E activity, between the diets containing 25 or 35 per cent calories from fat. The additional calcium in the 35-per-cent-fat-calorie diet was apparently due to the greater use of dairy products; the additional vitamin E was due to more margarine. Tocopherol contents, as predicted from other available data (42), were lower than the 1968 allowance of 30 i.u. per day for alpha- (80 per cent) and other (20 per cent) tocopherols prevailing before the study was planned. The allowances for tocopherol were recently revised (8) to 12 to 15 i.u. instead of 30 i.u. By the new standard, Table 6 shows that the tocopherol contents of our diets, as well as those of Bieri and Evans (42), were equal to or exceeded the new allowances (1 mg. d-alpha-tocopherol = 1.5 i.u.).

Few analyses of complete meals have been reported in the literature. The papers of Murphy *et al.* (29-31, 43, 44) reporting the extensive analyses of Type A school lunches showed that goals for most of the nutrients were met by the lunches, although fat content was higher than desirable for some schools. Nutritive value of the lunches from laboratory analyses were compared with goals, but no effort was made to com-

Table 5: Composition of daily diets containing 25 and 35 per cent calories from fat: Ten-day averages of determined data for additional minerals and vitamins not in Table 1 of Agriculture Handbook No. 8

constituents	25 per cent calories from fat*			35 per cent calories from fat†		
	analyzed, total	per kilogram	analyzed, per 1,000 kcal	analyzed, total	per kilogram	analyzed, per 1,000 kcal
Minerals						
magnesium (mg.)	199 ± 9‡	110	97	277 ± 16	123	97
zinc (mg.)	7.30 ± 0.44	4.02	3.56	10.87 ± 0.67	4.83	3.81
aluminum (mg.)	4.95 ± 1.3	2.73	2.41	6.15 ± 1.68	2.74	2.15
copper (mg.)	0.735 ± 0.053	0.405	0.358	0.995 ± 0.081	0.443	0.349
manganese (mg.)	3.14 ± 0.42	1.73	1.53	3.69 ± 0.56	1.64	1.29
chromium (mg.)	0.777 ± 0.115	0.428	0.379	1.171 ± 0.174	0.521	0.410
barium (mg.)	0.331 ± 0.024	0.182	0.161	0.399 ± 0.022	0.177	0.140
strontium (mg.)	1.54 ± 0.08	0.85	0.750	2.44 ± 0.12	1.09	0.85
boron (mg.)	1.31 ± 0.09	0.72	0.638	1.68 ± 0.13	0.75	0.59
Vitamins						
vitamin B ₆ (mg.)	2.31 ± 0.13	1.27	1.13	3.20 ± 0.20	1.42	1.12
vitamin B ₁₂ (mcg.)	5.55 ± 0.44	3.06	2.70	7.32 ± 0.95	3.26	2.56
pantothenic acid (mg.)	2.33 ± 0.30	1.28	1.14	3.11 ± 0.67	1.38	1.09
folacin (mg.)	0.173 ± 0.008	0.095	0.084	0.239 ± 0.014	0.105	0.084
biotin (mcg.)	30.4 ± 2.3	16.8	14.8	40.0 ± 3.0	17.8	14.0
tocopherol (mg.)						
alpha	7.6 ± 0.8	4.2	3.7	12.9 ± 0.9	5.7	4.5
gamma	14.2 ± 0.9	7.8	6.9	26.9 ± 2.1	12.0	9.4
delta	4.0 ± 0.2	2.2	1.9	5.6 ± 0.3	2.5	2.0
total	25.8 ± 1.5	14.2	12.6	45.4 ± 2.9	20.2	15.9

*2,000-kcal meals analyzed.

†2,800-kcal meals analyzed.

‡Mean ± standard error.

Table 6: Analyzed nutrient composition of diets compared with 1974 Recommended Dietary Allowances for adult men and women

constituents	allowances for adults*		analyzed values†			
	men	women	25 per cent fat calories		35 per cent fat calories	
			men	women	men	women
food energy (kcal)	2,400	1,800	2,400	1,800	2,400	1,800
protein (gm.)	56	46	96.2	72.2	100.4	75.3
minerals						
calcium (mg.)	800	800	575	431	670	502
phosphorus (mg.)	800	800	1,144	858	1,255	942
magnesium (mg.)	350	300	254	191	256	192
iron (mg.)	10	10	17.4	13.0	17.2	12.9
zinc (mg.)	15	15	9.3	7.0	10.1	7.5
copper (mg.)‡	2.5	2.5	0.939	0.705	0.920	0.690
vitamins						
vitamin A activity (i.u.)	5,000	4,000	10,604	7,956	11,495	8,623
thiamin (mg.)	1.2	1.0	2.11	1.58	1.93	1.45
riboflavin (mg.)	1.5	1.1	1.89	1.42	1.83	1.37
niacin (mg.)	16	12	30.5	22.9	29.1	21.9
ascorbic acid (mg.)	45	45	184	138	167	126
vitamin B ₆ (mg.)	2.0	2.0	2.9	2.2	3.0	2.2
vitamin B ₁₂ (mcg.)	3.0	3.0	7.1	5.3	6.8	5.1
pantothenic acid (mg.)‡	5-10	5-10	3.0	2.2	2.9	2.2
folacin (mcg.)	400	400	221	166	221	166
biotin (mcg.)‡	150-300	150-300	38.8	29.1	37.0	27.8
vitamin E activity (i.u.)#	15	12	—	—	—	—
alpha-tocopherol (mg.)			9.7	7.3	11.9	9.0
total tocopherol (mg.)			33.0	24.7	42.0	31.5

*Age 51 plus; allowances for this age were chosen because average age of subjects was about fifty years.

†For diets with 25 per cent calories from fat, values adjusted to 2,400 and 1,800 kcal from 1,878 and 2,594 kcal. in analyzed diets.

‡Average amounts in daily diets; no requirements established.

#80% alpha-tocopherol; 20% other tocopherols; 1 mg. d-alpha-tocopherol = 1.5 i.u. |

Table 7: Comparison of ten-day averages for calculated and analyzed composition of diets by paired t-test analysis

nutrient	25 per cent calories from fat				35 per cent calories from fat			
	mean difference*	standard error of difference	t value	P	mean difference*	standard error of difference	t value	P
calories (kcal)	- 25.2	20.86	1.21	NS	- 23.2	50.80	0.46	NS
protein (gm.)	4.3	1.11	3.86	0.005	4.0	2.30	1.74	NS
carbohydrate (gm)	33.1	4.26	7.77	0.001	33.8	6.92	4.88	0.001
fat (gm.)	4.8	3.33	1.44	NS	14.2	2.46	5.76	0.001
cholesterol (mg.)	23.0	6.72	3.42	0.01	80.9	9.68	8.36	0.001
fiber (gm.)	0.9	0.27	3.37	0.01	0.3	0.49	0.62	NS
ash (gm.)	1.2	0.43	2.77	0.025	1.3	1.0	1.30	NS
calcium (mg.)	292.1	25.30	11.54	0.001	337.1	37.98	8.87	0.001
phosphorus (mg.)	311.3	20.08	15.51	0.001	367.8	44.74	8.22	0.001
iron (mg.)	1.0	0.63	1.60	NS	- 0.5	1.82	0.28	NS
sodium (mg.)	-525.3	172.34	3.05	0.025	- 682.5	216.7	3.15	0.025
potassium (mg.)	-398.3	107.51	3.62	0.01	- 442.8	99.20	4.46	0.005
vitamin A (I.U.)	-252.3	712.03	0.35	NS	-1,677.4	1,096.7	1.53	NS
thiamin (mg.)	- 0.2	0.07	2.89	0.025	- 0.22	0.11	1.95	NS
riboflavin (mg.)	0.05	0.06	0.84	NS	0.15	0.073	2.05	NS
niacin (mg.)	- 2.8	0.83	3.38	0.01	- 4.12	- 2.25	1.83	NS
ascorbic acid (mg.)	38.6	11.56	3.34	0.01	42.5	11.74	3.62	0.01

*Mean difference = mean of calculated minus mean of analyzed values for a ten-day period.

pare analyzed with calculated values.

Some studies have reported dietary analyses for specific nutrients for comparison with calculated content of diets. Mosen *et al.* found (45) analyzed values for iron to be highly correlated with calculated values. Manola *et al.* compared (46, 47) analyzed and calculated values for a total diet. Their analyzed values for sodium and phosphorus were higher, and for magnesium and calcium lower, than calculated values; no differences were found for potassium and nitrogen. Although statistically significant differences were found, they concluded that close approximation could be obtained with published values for all analyzed elements except phosphorus and magnesium. They concluded also (46) that, with the above exceptions, published tables offer a closer estimate of dietary values than previously reported.

The data in Table 7 confirm the conclusion of Manola and Jones (46) that only a few nutrients present a problem in calculating nutrient intakes. Although many differences found in this study were statistically significant by the paired *t*-test, little importance can be attached to them except for calcium. Differences between values for carbohydrate are probably related to the additive variations in proximate composition, since carbohydrate was calculated by difference.

Table 8 shows the variation in composition of the same menus served on Day 4 for four cycles during the 35-per-cent-fat-calorie period. The largest variation was in iron content. Of the remaining sixteen components, ten varied more than 10 per cent. However, reproducibility of the analyses may be reflected in the comparison of values for the analyzed composite with the average of the four individual menus for Day 4. All except four of the sixteen components varied about 5 per cent or less.

Comment

Obviously, more information is needed before calculated dietary intakes can be relied on exclusively to provide data on specific amounts of the many nutrients in foods eaten by participants in a research study. Uses and limitations of such tables have already been discussed (48). However, calculations must always be used to provide some estimations of intake prior to a study, because some information from analyses can be obtained only during or after completion of the study. Certain foods can be prepared, analyzed, and/or frozen for later consumption, but fresh fruits and vegetables, milk, eggs, and so forth cannot, if maximum palatability of meals is to be maintained. Although highly desirable, the cost of analyses of individual foods, as well as of meals before and after preparation or processing, is prohibitive.

Only after better methods of analysis and more data are available for some nutrients can nutrients not included in Agriculture Handbook No. 8 be calculated with confidence. The updating of the Handbook, as a result of the Nutrient Data Bank (49), will reflect current processing methods and fortification.

Our purpose in chemically analyzing the meals was not solely to compare analyzed with calculated data. A thorough job would have required individual analyses of all food items. To repeat, our primary purpose was to learn, as accurately as possible, the nutrient composition of what our subjects ate. As a bonus, we gained added confidence in our calculations. The questions that remain about the differences in results that may have been produced by inadequate methods show the massive effort needed by the food industry, in cooperation with government agencies, universities, agricultural experiment stations, and all food and nutritional scientists, to answer vital questions about

Table 8: Results of analyses of Day 4 for each of four cycles of diets containing 35 per cent calories from fat, 2,800 kcal: Ranges and deviations from means

nutrients in diets	mean of four cycles of Day 4	ranges in four cycles of Day 4	per cent deviation from average of four days	analyzed composite for Day 4	per cent deviation of average of four cycles vs. composite
food energy (kcal)*	2,895 [†] ± 77	2,791 - 2,976	6.4	2,859	1.2 [‡]
total protein (gm.)	115.0 ± 2.5	112.1 - 118.2	5.3	113.9	1.0
carbohydrate (gm.)#	333.2 ± 10.5	318.0 - 341.9	7.2	327.5	1.6
total fat (gm.)	89.7 ± 3.2	87.5 - 94.4	7.7	94.3	- 5.1
cholesterol (mg.)	224 ± 28	201 - 265	28.6	234	- 4.5
fiber (gm.)	7.1 ± 0.6	6.6 - 8.0	19.7	7.1	0
ash (gm.)	26.1 ± 1.6	23.9 - 27.6	14.2	26.1	0
calcium (mg.)	798 ± 8.9	784 - 824	5.0	842	- 5.5
phosphorus (mg.)	1,403 ± 87	1,288 - 1,499	15.0	1,388	1.1
iron (mg.)	17.4 ± 6.8	13.3 - 27.2	79.9	18.3	- 5.2
sodium (mg.)	5,834 ± 404	5,444 - 6,240	13.6	5,707	2.2
potassium (mg.)	5,243 ± 467	4,544 - 5,526	18.7	5,025	4.2
vitamin A (I.U.)‡	15,968 ± 1,808	13,957 - 18,329	27.4	17,204	- 7.7
thiamin (mg.)	2.85 ± 0.12	2.72 - 2.99	9.5	2.61	8.4
riboflavin (mg.)	2.53 ± 0.16	2.38 - 2.68	11.8	2.41	5.1
niacin (mg.)	35.2 ± 1.7	33.1 - 36.9	10.8	32.7	7.1
ascorbic acid (mg.)**	173 ± 9	166 - 184	10.3	193	-10.9

*Analyzed by bomb calorimetry.

†Mean ± standard deviation.

‡deviation in analyzed values
composite × 100.

#100 - (total fat + protein + ash + fiber + moisture) (carbohydrate by difference).

‡Preformed vitamin A plus beta-carotene.

**Reduced plus dehydroascorbic acid.

optimal nutrient intakes for human beings.

Although the calcium comparisons are of concern, it was gratifying to find results of the nutrient analyses were as close as they were to calculated data.

The message to dietitians, nutritionists, and others involved in formulating fat- and cholesterol-controlled diets is that these diets can be derived from foods readily available on the open market. Except for a few nutrients, such diets can be nutritionally adequate for those most in need of them. Whether the low levels of some nutrients observed on chemical analysis are low because of inadequate analytical methods is not known. More detailed information on the reasons for the differences in the calcium content, in particular, is needed.

Since the preparation of this manuscript, Head *et al.* reported (50) lower calcium values for analyzed type A lunches than for calculated values. As they suggested, if these differences are real, some consideration might be given to the use of a correction factor applied when calculated values are used exclusively for this nutrient.

Summary

In a dietary study conducted to evaluate the effects of low-fat, low-cholesterol diets on the reduction of blood lipids in man, meals were analyzed for proximate composition, ten vitamins, fourteen minerals and trace minerals, fatty acids, tocopherols, and cholesterol. Validity of the calculated nutrient composition of the diets was assessed by comparing cal-

culated with determined values. Comparisons were also made of the determined values with 1974 Recommended Dietary Allowances. Analyzed values and those calculated from Agriculture Handbook No. 8 were exceptionally close, except for calcium. By analysis, on a per-kilogram basis, the 35-per-cent-fat-calorie diet more nearly met the Recommended Dietary Allowances for most of the nutrients than did the 25-per-cent-fat-calorie diet. However, on a per-1,000-kcal basis, there were few differences between nutrients in diets with the two levels of fat calories.

References

- (1) NATL DIET-HEART STUDY RESEARCH GROUP: The National Diet-Heart Study Final Report. Am. Heart Assoc. Monograph 18. Circulation 37: Suppl. 1, 1968.
- (2) BROWN, H.B.: The National Diet-Heart Study—implications for dietitians and nutritionists. J. Am. Dietet. A. 52: 279, 1968.
- (3) Report of Inter-Society Commission for Heart Disease Resources. Primary prevention of the atherosclerotic diseases. Circulation 42: 55, 1970.
- (4) FOOD & NUTR. BD., NATL. ACAD. SCI.—NATL. RESEARCH COUNCIL AND COUNCIL ON FOODS & NUTR., AM. MED. ASSOC.: Diet and coronary heart diseases. J. Am. Dietet. A. 61: 379, 1972.
- (5) COMM. ON NUTR., AM. HEART ASSOC.: Diet and coronary heart disease. NUTR. TODAY 9: 26 (May/June), 1974.
- (6) FRIEND, B.: Nutritional review. In USDA National Food Situation No. 146, Nov. 1973.
- (7) WATT, B.K., AND MERRILL, A.L.: Composition of Foods—Raw, Processed. Prepared. Rev. USDA Agric. Handbook No. 8, 1963.

Diet 200 - 265 mg chol per 100-150 cal Phase III

- (8) FOOD & NUTR. BD.: Recommended Dietary Allowances, Eighth Revised Edition, 1974. Washington, D.C.: Natl. Acad. Sci., 1974.
- (9) IACONO, J.M.: Unpublished data.
- (10) BROWN, H.B.: Food patterns that lower blood lipids in man. *J. Am. Dietet. A.* 58: 303, 1971.
- (11) CARPENTER, D.L., AND SLOVER, H.T.: Lipid composition of selected margarines. *J. Am. Oil Chem. Soc.* 50: 372, 1973.
- (12) HORWITZ, W., ED.: Official Methods of Analysis. 11th ed. Washington, D.C.: Assoc. Off. Anal. Chem., 1970.
- (13) WRIGHT, L.D., AND SKEGGS, H.R.: Determination of biotin with *Lactobacillus arabinosus*. *Proc. Soc. Exp. Biol. Med.* 56: 95, 1944.
- (14) NEILANDS, J.B., AND STRONG, F.M.: The enzymatic liberation of pantothenic acid. *Arch. Biochem.* 19: 287, 1948.
- (15) Vitamin B₁₂ activity assay. U.S. Pharmacopeia XVII: 864, 1964.
- (16) ATKIN, L., SCHULTZ, A.A., WILLIAMS, W.L., AND FREY, C.N.: Yeast microbiological methods for determination of vitamins. Pyridoxine. *Indust. Engin. Chem.* 15: 141, 1943.
- (17) ROBINSON, W.B., AND STOTZ, E.: The indophenol-xylene extraction method for ascorbic acid and modification for interfering substances. *J. Biol. Chem.* 160: 217, 1945.
- (18) LEHMANN, J., AND SLOVER, H.T.: Determination of plasma tocopherols by gas liquid chromatography. *Lipids* 6: 35, 1971.
- (19) CONSUMER & FOOD ECON. INST., AGRIC. RESEARCH SERV.: Food composition data on punched cards and magnetic tape. USDA Data Set 8-1-1, Expansion 1. March 1972.
- (20) FEELEY, R.M., CRINER, P.E., AND WATT, B.K.: Cholesterol content of foods. *J. Am. Dietet. A.* 61: 134, 1972.
- (21) LACROIX, D.C., MATTINGLY, W.A., WONG, N.P., AND ALFORD, J.A.: Cholesterol, fat, and protein in dairy products. *J. Am. Dietet. A.* 62: 275, 1973.
- (22) FRIEND, B. (Consumer & Food Econ. Inst.): Personal communication.
- (23) BOOHER, L.E., HARTZLER, E.R., AND HEWSTON, E.M.: A Compilation of the Vitamin Values of Foods in Relation to Processing and Other Variants. USDA Circ. No. 638, 1942.
- (24) HEWSTON, E.M., DAWSON, E.H., ALEXANDER, L.M., AND ORENT-KEILES, E.: Vitamin and Mineral Content of Certain Foods as Affected by Home Preparation. USDA Misc. Publ. No. 628, 1948.
- (25) SWEENEY, J.P., CHAPMAN, V.J., MARTIN, M.E., AND DAWSON, E.H.: Quality of frozen fruit from retail markets. *Food Technol.* 16: 138, 1962.
- (26) ZOOK, E.G., AND LEHMANN, J.: Mineral composition of fruits. 2. Nitrogen, calcium, magnesium, phosphorus, potassium, aluminum, boron, copper, iron, manganese, and sodium. *J. Am. Dietet. A.* 52: 225, 1968.
- (27) ZOOK, E.G.: Mineral composition in fruits. 3. Total solids, ash, nitrogen, and minerals of six dried fruits. *J. Am. Dietet. A.* 53: 588, 1968.
- (28) HOPKINS, H., AND EISEN, J.: Mineral elements in fresh vegetables from different geographic areas. *J. Agric. Food Chem.* 7: 633, 1959.
- (29) MURPHY, E.W., WATT, B.K., AND PAGE, L.: Regional variations in vitamin and trace element content of Type A school lunches. In Hemphill, D., ed.: Trace Substances in Environmental Health. Columbia, Mo.: Univ. of Missouri Press, 1971.
- (30) MURPHY, E.W., PAGE, L., AND WATT, B.K.: Trace minerals in Type A school lunches. *J. Am. Dietet. A.* 58: 115, 1971.
- (31) MURPHY, E.W., PAGE, L., AND WATT, B.K.: Major mineral elements in Type A school lunches. *J. Am. Dietet. A.* 57: 239, 1970.
- (32) CHRISTENSEN, R.E., BECKMAN, R.M., AND BIRDSALL, J.J.: Some mineral elements of commercial species and herbs as determined by direct reading emission spectroscopy. *J. Assoc. Off. Anal. Chem.* 51: 1003, 1968.
- (33) CHRISTENSEN, R.E., COON, F.B., AND DERSE, P.H.: Computer application for direct concentration printout of plant tissue analysis by emission spectroscopy. Presented at Pittsburgh Conf. on Anal. Chem. and Appl. Spectroscopy, March 1967 (issued as Reprint No. 74, Jarrell-Ash Co.).
- (34) WATT, B.K., MURPHY, E.W., AND GERHARDT, S.E.: Fruits and vegetables: USDA research for tables of food composition. In White, P.L., and Selvey, N.: Nutritional Qualities of Fresh Fruits and Vegetables. Mt. Kisco, N.Y.: Futura Pub. Co., 1974, chap. 2.
- (35) FEELEY, R.M., CRINER, P.E., MURPHY, E.W., AND TOEPFER, E.W.: Major mineral elements in dairy products. *J. Am. Dietet. A.* 61: 505, 1972.
- (36) SWEENEY, J.P., GILPIN, G.L., STANLEY, M.G., AND MARTIN, M.E.: Effect of cooking methods on broccoli. 1. Ascorbic acid and carotene. *J. Am. Dietet. A.* 35: 354, 1959.
- (37) WOLF, W., MERTZ, W., AND MASIRONI, R.: Determination of chromium in refined and unrefined sugars by oxygen plasma ashing flameless atomic absorption. *Agric. Food Chem.* 22: 1037, 1974.
- (38) ZOOK, E.G., AND LEHMANN, J.: Total diet study: Content of ten minerals—aluminum, calcium, phosphorus, sodium, potassium, boron, copper, iron, manganese, and magnesium. *J. Assoc. Off. Agric. Chem.* 48: 850, 1965.
- (39) ORR, M.L.: Pantothenic Acid, Vitamin B₆, and Vitamin B₁₂ in Foods. USDA Home Econ. Research Rept. 36, 1969.
- (40) ORR, M.L., AND WATT, B.K.: Losses of vitamins and trace minerals resulting from processing and preservation of foods. *Am. J. Clin. Nutr.* 25: 648, 1972.
- (41) FOOD & NUTR. BD.: Recommended Dietary Allowances. Seventh Revised Edition, 1968. Natl. Acad. Sci. Pub. No. 1694, 1968.
- (42) BIERI, J.G., AND EVANS, R.P.: Tocopherols and fatty acids in American diets. *J. Am. Dietet. A.* 62: 147, 1973.
- (43) MURPHY, E.W., KOONS, P.C., AND PAGE, L.: Vitamin content of Type A school lunches. *J. Am. Dietet. A.* 55: 372, 1969.
- (44) MURPHY, E.W., PAGE, L., AND KOONS, P.C.: Lipid components of Type A school lunches. *J. Am. Dietet. A.* 56: 504, 1970.
- (45) MONSEN, E.R., KUHN, J.N., AND FINCH, C.A.: Iron status of menstruating women. *Am. J. Clin. Nutr.* 20: 842, 1967.
- (46) MANOLA, R., AND JONES, J.E.: The content of constant diets. A comparison between analyzed and calculated values. *Am. J. Clin. Nutr.* 18: 339, 1966.
- (47) MANOLA, R., FLORA, R.E., AND JONES, J.E.: A simple method for estimating dietary magnesium. *Am. J. Clin. Nutr.* 20: 627, 1967.
- (48) MURPHY, E.W., WATT, B.K., AND RIZEK, R.L.: Tables of food composition: Availability, uses and limitations. *Food Technol.* 27: 40 (Jan.), 1973.
- (49) WATT, B.K., GERHARDT, S.E., MURPHY, E.W., AND BURRUM, R.R.: Food composition tables for the 70's. *J. Am. Dietet. A.* 64: 257, 1974.
- (50) HEAD, M.K., WELKS, R.J., AND GIBBS, E.: Major nutrients in the Type A lunch. 1. Analyzed and calculated values of meals served. *J. Am. Dietet. A.* 63: 620, 1973.



Nutrition imbalance and angiotoxins as dietary risk factors in coronary heart disease^{1, 2}

Fred A. Kummerow

ABSTRACT Imbalancing nutritionally adequate diets with an excessive amount of fat calories and cholesterol has obscured the fact that intimal thickening occurs spontaneously in time on low-fat cholesterol-free diets during the aging process, and that intimal thickening can be accelerated by dietary angiotoxic "risk factors." Electron microscopy of arterial tissue from animal models identified degenerated smooth muscle cells in the fetus from sows kept on low-fat cholesterol-free diets. After birth, the degenerated smooth muscle cells increased in number with age. The presence of angiotoxic "risk factors" such as oxidized cholesterol and vitamin D₃ (cholecalciferol) in the diet of such animal models increased the frequency of smooth muscle cell death in their arteries. Two types of pathology could be developed in the thoracic aorta by continuous or short term feeding of 12.5 times more vitamin D than normally present in commercial rations: 1) a diffuse fibroelastic intimal thickening in the thoracic aorta (arteriosclerosis) with no evidence of lipid deposition by continuous feeding of vitamin D or 2) an intimal thickening in the thoracic aorta and intimal thickening with foam cells and extracellular lipid deposits (atherosclerosis) in the coronary arteries after a short period of supplemental vitamin D followed by 3 to 4 months of supplement-free diets. These two types of arterial damage were identical to that in the plugs of thoracic aorta obtained as a by-product of elective coronary bypass surgery. Although all of the possible sources of oxidized cholesterol in the diet have as yet not been identified, laboratory studies have identified oxidized cholesterol as an angiotoxic factor. Since population groups that consume less vitamin D-supplemented foods, less deep fat fried cholesterol-containing foods, and less hydrogenated fats have a lower incidence of coronary heart disease than Americans, it seems judicious for food processors to reduce these previously unconsidered risk factors to a minimum. This could be done by eliminating vitamin D₂ and D₃ from all vitamin supplements, from all food and cereal products and from the diet of livestock 1 month before they were killed so that the intake of vitamin D is no larger than the 400 IU/quart in milk which is necessary to prevent rickets in children. Deep fat fryers, which are kept at almost 200 C for 24 hr/day, could perhaps be replaced with microwave ovens in fast food chain outlets. Processors could hydrogenate vegetable oils to a minimum *trans* fatty acid content and rearrange this fat with polyunsaturated fats to produce high polyunsaturated fats *trans*-free margarines and shortenings. *Am. J. Clin. Nutr.* 32: 58-83, 1979.

The recommendations in regard to diet that were made in 1970 in the Report of Inter-Society Commission for Heart Disease Resources (1) have recently been incorporated into the first report entitled Dietary Goals for the United States by the United States Senate Select Committee on Nutrition and Human Needs (2). Seven recommendations were listed in this report: 1) Increase consumption of fruits and vegetables and whole grains, 2) Decrease consumption of meat and increase consumption of poultry

and fish. 3) Decrease consumption of foods high in fat and partially substitute polyunsaturated fat for saturated fat. 4) Substitute non-fat for whole milk. 5) Decrease consumption of butterfat, eggs, and other high cholesterol

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sources. 6) Decrease consumption of sugar and food high in sugar content. 7) Decrease consumption of salt and food high in salt content.

These goals were projected to decrease fat consumption from 42 to 30% and increase carbohydrate consumption from 46 to 58% with no change in protein consumption. Furthermore, cholesterol consumption was projected to decrease from 600 to 300 mg per capita a day. It may be possible to decrease fat consumption by eliminating from the diet high fat foods, such as fried foods, and increase grain consumption. However, it is impossible to lower cholesterol intake to 300 mg/day and keep the protein level at 12% because vegetable protein food items in quantity large enough to supplant cholesterol-containing protein sources, such as meat (primarily beef), milk, cheese and eggs, are not available for purchase in the United States. Poultry and fish contain as much cholesterol as beef. The limitations that are imposed on these recommendations by agriculture and technology and individual variations have not been fully considered. Furthermore, the subtle changes that occur in the coronary arteries and the heart muscle, during the normal aging process, have not been elucidated thoroughly enough to warrant adopting of the radical change in diet implied under point 5). However, technology could be altered to improve the nutrition of the foods available to all Americans. These alterations in technology are discussed in this paper.

A high protein diet is necessary for population groups on high fat diets

The complete protein that is furnished by meat, milk, cheese, and eggs is necessary to the *in vivo* synthesis of the various apoproteins that carry the excessive level of fat (lipid) in the blood as serum lipoproteins in population groups on high fat diets. The liver incorporates the preformed cholesterol in meat, milk, cheese, and eggs into lipoproteins or synthesizes the equivalent of six times more cholesterol into lipoproteins each day (3) than the 300 mg of dietary cholesterol recommended as a daily maximum in the Dietary Goals (2). The approximately 1800 mg of cholesterol that is made available by the liver every day is believed to be necessary

to the normal structure and function of every cell membrane in the body. Goldstein and Brown (3) believe that most of the specialized cells in the body have either lost the ability to synthesize cholesterol or synthesize cholesterol too slowly to meet the structural or metabolic need.

The Dietary Goals do not emphasize the need for restricting total calorie intake. A diet excess in calories from either carbohydrate or fat would only result in the synthesis of more fat and cholesterol *in vivo* than would a diet of meat, milk, cheese, eggs, fruits, vegetables, and cereals in an amount that meets nutritional need (4). The recommendations that were listed under the Dietary Goals were based on the hypothesis (3) that the amount of cholesterol in the low density lipoprotein (LDL) of human serum seems to reflect susceptibility to coronary heart disease (CHD). As food products of animal origin, such as meat, milk, cheese, and eggs, contain cholesterol and as preformed cholesterol is absorbed from the intestinal tract, it was assumed that a decreased consumption or elimination of some of these food items from the diet would result in a lower concentration of cholesterol in the serum (5, 6).

Americans have consumed approximately 600 mg of cholesterol per day since 1909, the date that food consumption data first became available (Table 1). This level of cholesterol consumption is higher than for some population groups with less CHD than Americans (1). On the other hand, some population groups (7), such as rural Romanians, have 10 to 20 times less CHD than Americans and yet consume 300 mg more cholesterol per day than Americans (Table 2). For example, rural Romanians consume approximately 900 mg of cholesterol from their major calorie sources; Americans consume approximately 600 mg of cholesterol per day from these sources. Yet, rural Romanians have a CHD rate of only 0.3/1000 per year as compared to 6/1000 per year for Americans. If population groups exist that consume more preformed cholesterol and yet have a lower serum cholesterol level and a lower rate of CHD than Americans, there must be other factors than cholesterol in the diet of Americans that increase the serum cholesterol level and the rate of CHD.

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TABLE 1
Annual per capita consumption of
animal food products

Year	Meat		Poultry		Fish		Eggs		Dairy products		Animal fat		Total pounds of linoleic acid ^a	Total cholesterol ^b
	Pounds	Pounds of linoleic acid ^a	Pounds	Pounds of linoleic acid ^a	Pounds	Pounds of linoleic acid ^a	Pounds	Pounds of linoleic acid ^a	Quarts	Pounds of linoleic acid ^a	Pounds	Pounds of linoleic acid ^a		
1909	146.6	0.70	15.9	0.38	13.9	0.47	35.1	0.63	178	0.18	12.4	0.43	2.8	545
1960	146.9	0.71	34.6	0.83	13.2	0.45	42.5	0.76	238	0.24	7.5	0.26	3.3	614
1961	145.9	0.70	37.8	0.90	13.7	0.47	41.7	0.75	235	0.24	7.4	0.26	3.3	563
1962	147.1	0.71	37.4	0.90	13.6	0.46	41.4	0.75	235	0.24	7.3	0.26	3.3	609
1963	152.0	0.73	37.9	0.91	13.7	0.47	40.4	0.73	234	0.23	6.9	0.24	3.3	610
1964	155.7	0.75	38.9	0.93	13.5	0.46	40.4	0.73	235	0.24	6.9	0.24	3.4	616
1965	148.3	0.71	41.3	0.99	13.8	0.47	39.8	0.72	234	0.23	6.4	0.22	3.3	602
1966	151.4	0.73	44.3	1.1	13.9	0.47	39.7	0.71	234	0.23	5.7	0.20	3.4	607
1967	158.3	0.76	46.2	1.1	13.6	0.46	40.6	0.73	230	0.23	5.5	0.20	3.5	623
1968	162.4	0.78	45.8	1.1	14.0	0.48	40.1	0.73	231	0.23	5.7	0.20	3.5	628
1969	161.4	0.77	47.8	1.1	14.2	0.48	39.3	0.71	230	0.23	5.4	0.19	3.5	633
1970	164.6	0.79	50.1	1.2	14.8	0.50	39.5	0.71	226	0.23	5.3	0.19	3.6	628
1971	170.0	0.82	50.3	1.2	14.4	0.49	39.9	0.72	228	0.23	5.1	0.18	3.6	639
1972	166.5	0.80	52.5	1.3	15.3	0.52	39.0	0.70	228	0.23	4.9	0.17	3.7	631
1973	154.6	0.74	50.5	1.2	15.6	0.53	37.2	0.67	229	0.23	4.8	0.17	3.5	600

^a Calculations of pounds of linoleic acid based upon assumption that amount of linoleic acid in certain foods has not changed over the past 60 years. (Fat Content and Composition of Animal Products, The National Research Council, December, 1974). ^b Mg/day as calculated from the total per capita intake of each food item and its cholesterol content (*J. Am. Oil Chem. Soc.* 27: 414, 1950. (*Am. J. Clin. Nutr.* 30: 664, 1977.)

were listed in the Dietary Goals for the United States decrease serum cholesterol levels and decrease the rate of CHD? Data obtained with animal models indicate that decreasing protein, vitamins, and mineral intake (which would result from a decrease in the consumption of meat, milk, cheese, and eggs) stunted growth, increased serum cholesterol levels (8), and increased the incidence of atherosclerosis the cause of 90% of all coronary heart disease (9). Swine supplemented with either animal or vegetable fat had a higher serum cholesterol level and a higher incidence of atherosclerosis than swine on the basal unsupplemented diet. Furthermore, Rhesus monkeys fed a diet of commonly available food items in an amount 200 cal/day higher than the amount considered prudent (10) had higher serum cholesterol levels and more atherosclerosis than those on the nutritionally optimum diet for this animal model (Table 3). In the American diet, high calorie food items, such as potato chips and french fries, serve the same role as fat added to the diet of experimental models. That is, they imbalance the diet of animal models and thus increase serum cholesterol levels and the incidence of atherosclerosis.

Technical difficulties involved in implementing the dietary goals

That better nutrition would lead to better health is beyond dispute. What is under dispute is how to reach this goal. Nutritionists would commend the recommendation to increase the consumption of fruits and vegetables and of whole grains.

A recommendation to increase the consumption of fruits, vegetables, and grains was made because these food items furnish fiber to the diet and fiber reduces the amount of fat absorption from the intestinal tract in a diet high in fat calories such as the American diet (11, 12). These food items also supply vitamins and minerals to the diet and vegetables and fruits are low in fat content. It would be desirable to consume more whole grains. However, milling processes will have to be changed before acceptable whole grain products or their milled products are available to human consumption (13-17). The byproducts will have to be freed of fat in order to stabilize them against oxidation and then either sold separately or recombined with the main milled fraction.

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TABLE 2
Food consump
compared with

Item
Bread ^d
Sugar (total)
Sugar (as soft drink)
Hydrogenated Vegetable fat (shortening)
Unhydrogenat vegetable oil
Margarine ^e
Butter
Lard
Milk
Beef
Poultry
Fish
Eggs (no.)
Average intake from above
Average protei
Average fat int
Average calori
Average serum rate of myoc

^a Consumpt
and Human
Pavel. ^b Cal
of Foods. Unit
2% lard. ^c V
hydrogenated.
United States.
414, 1950. ^d
Institute of Pu

grain is consumed as bread made from de-hulled and degermed grain in order to enhance its keeping quality. To increase the consumption of bread made from whole grain would require a degree of cooperation between the grain processor, the Food and Drug Administration and the baker that does not exist today.

A decreased consumption of red meat (primarily beef), and an increased consumption of poultry and fish, was recommended because beef fat contains less polyunsaturated fat than poultry or fish. Disappearance data available from the United States Department of Agriculture (Table 1) indicates that both beef and poultry consumption has increased while fish consumption has remained station-

ary at 15 pounds/capita per year since 1909. Calculations based on disappearance data indicates that meat consumption has increased by 8 pounds since 1909. As chicken consumption has tripled and chicken fat contains more linoleic acid than beef fat, the actual consumption of linoleic acid from animal protein sources has increased from 2.8 to 3.5 pounds/capita per year. More fish could be made available through fish farms, but the marketing technology has been too risky to attract the capital investment that would be required (18).

The recommendation that the consumption of saturated fat from animal fat sources should be decreased and the polyunsaturated fat from poultry and fish should be increased

TABLE 2
Food consumption in the United States as compared with rural Romania

Item	United States			Rural Romania		
	Per capita/yr ^a	Cholesterol per capita/day ^b	Linoleic acid per capita/day ^c	Per capita/yr ^a	Cholesterol per capita/day	Linoleic acid per capita/day
Bread ^d	75 lb	2	0.2	281 lb	8	0.7
Sugar (total)	102 lb			32 lb		
Sugar (as soft drinks)	23 lb			0		
Hydrogenated Vegetable fat (shortening)	14 lb		1.2	0		0
Unhydrogenated vegetable oil ^e	14 lb		9.2	20 lb		13.1
Margarine ^f	10 lb		1.8	2 lb		0.4
Butter	6 lb	21	0.2	12 lb	42	0.7
Lard	5 lb	7	0.6	8 lb	11	1.0
Milk	229 qt	87	1.3	101 qt	38	0.6
Beef	154 lb	239	0.8	68 lb ^g	105	0.4
Poultry	50 lb	43	2.2	22 lb ^g	19	1.0
Fish	15 lb	11	0.04	7 lb ^g	5	0.02
Eggs (no.)	279	172	0.4	1141	703	1.5
Average intake per capita/day from above items		582 mg	17.9 g		931 mg	19.4 g
Average protein intake/capita ^h			69.1 g			73.9 g
Average fat intake/capita ^h			138.4 g			105.3 g
Average calorie intake/capita ^h			2088			2159
Average serum cholesterol level			203 mg ml ⁱ			160 mg ^j
rate of myocardial infarction			6/1000			0.3/1000

^a Consumption data on the United States from Dietary Goals for the United States Select Committee on Nutrition and Human Needs, United States Senate, February, 1977. Consumption data for Romania from Dr. Pitea Pavel. ^b Calculated from data in J. Am. Oil Chem. Soc. 27: 414, 1950. ^c Calculated from data in Composition of Foods. United States Department of Agriculture Handbook no. 8, Dec., 1963. ^d Calculated for bread containing 2% lard. ^e Values calculated for corn oil. ^f Calculated for margarine composed of all vegetable fat, principally hydrogenated. ^g Assuming the proportions of beef, poultry, and fish consumed in Romania are the same as in the United States. ^h Calculated from data in H. J. Heinz Company, Nutritional Data. ⁱ J. Am. Oil Chem. Soc. 27: 414, 1950. ^j Results of survey in seven villages in Transylvania, Romania in 1962, courtesy of Dr. Pitea Pavel, Institute of Public Health and Medical Researches, Cluj, Romania.

Total cholesterol^b

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TABLE 3
Results of feeding rhesus monkeys excess calories^a

Item	Nutrient					American diet ^b					Prudent diet ^c				
	% Protein	% Fat	% C18:2	Cholesterol mg/100 g	Amt.	Protein	Fat	C18:2	Cholesterol g	Amt.	Protein	Fat	C18:2	Cholesterol	
Dry milk	35.6	1.0	3	468	11	3.92	0.11	0.30	46.8	10	3.56	0.10	0.02	0	
Eggs	12.8	11.5	19	95	10	1.28	1.15	0.07	15.8	0	0	0	0	5.3	
Beef	17.4	23.0	2	75	15	2.61	3.45	0.05	3.2	5	0.87	1.15	0.02	0	
Beef fat	0.0	100	2	60	2.4	0	2.40	0.63	11.7	0	0	0	0.27	5.0	
Pork	16.4	25	9	108	7.0	1.15	1.75	0.14	1.48	3.0	0.49	0.75	0.08	0	
Pork fat	0.0	100	9	60	1.6	0	1.60	0.04	5.0	6.0	1.28	0.42	0.48	10.0	
Salmon	21.4	7.0	20	60	3.0	0.64	0.21	0.16	6.7	18.0	3.64	2.27	0.36	8.9	
Chicken	20.2	12.6	22	90	6.0	1.21	0.76	0.30	0.62	0	0	0	0	0	
Turkey	20.1	20.2	22	320	2	0	0.08	0.02	1.5	0	0	0	0	0	
Liver	19.8	4.2	38	160	2.4	0.57	0.77	0.06	0	3.0	0.59	0.02	0.06	0	
Cheese	23.9	32.3	3	0	0	0	0	0.06	0	36.0	3.06	0.72	0.02	0	
Cottage cheese	19.8	0.8	3	0	0	0	0	0.04	0	20.0	0.40	0.02	4.8	0	
Bread	8.5	2.0	9	0	20.0	0.14	0.04	1.8	0	20.0	0.14	0.04	1.8	0	
Potato	2.0	0.1	24	0	10.0	0.12	0.02	2.08	0	10.0	0.12	0.02	0	0	
Carrots	0.7	0.2	18	0	4.0	0.32	0.03	0.24	0	20.0	0.24	0.04	0	0	
Lettuce	1.2	0.2	18	0	20.0	0.06	0.08	0.04	0	20.0	0.06	0.08	0	0	
Cereal	7.9	0.7	52	0	8.0	0.51	0.66	0.24	4.0	0	0	0	0	0	
Banana	1.2	0.2	3	20	24.0	0.21	0.04	0.81	0	24.0	0.21	0.04	0	0	
Apple	0.3	0.4	0	0	18.0	0	0	0.40	0	9.0	0	0	0	0	
Pound cake	6.4	8.2	0	0	1.8	0	1.80	0.04	0	0	0	0	0	0	
Orange juice	0.9	0.2	0	0	4.4	0.02	3.52	0.40	0	0	0	0	0	0	
Sugar	0	0	45	0	0	0	0	0.22	0	3.0	0.02	2.4	0.96	0	
Cottonseed oil	0	100	9	0	0	0	0	0.18	0	7.0	0	7.0	3.50	0	
Saturated	0.6	80	9	0	3	0.03	0.65	0.17	1.67	0	0	0	0	0	
Margarine	0.6	80	32	0	0	16.94	28.56	3.56	6.0	0	16.29	11.21	5.73	49.2	
PUFA Margarine	0.6	100	50	0	0	0	0	0.22	0	0	0.02	2.4	0.96	0	
Corn oil	0.6	81	3	185	9	0.05	7.29	0.18	4.86	7.0	0	7.0	3.50	0	
Butter	0	100	9	120	2	0	2.0	0.18	1.67	0	0	0	0	0	
Lard	0	100	9	50	3	0.03	1.95	0.17	6.0	0	0	0	0	0	
Bacon	9	65	9	0	0	0	0	0.17	6.0	0	0	0	0	0	
Totals				109.33											

^a Wissler, R. The Myocardium: Failure and Infarction, edited by E. Braunwald. New York: HP Publishing Co., 1974. ^b American diet: cal/day, 560; serum cholesterol, 383 mg; total serum lipids, 1029 mg; aorta involvement, 54%; coronary atherosclerosis, 27%. Prudent diet: calories/day, 360; serum cholesterol, 199 mg; total serum lipids, 549 mg; aorta involvement, 9%; coronary atherosclerosis, 6%. ^c Identical to Nutrient Requirements of Laboratory Animals, no. 10, 2nd ed. Washington, D.C.: National Academy of Sciences, 1972.

was based on serum cholesterol tested as a standard diet consisting of skim milk and unsaturated fat. The serum cholesterol contained both low linoleic and cholesterol. When a similar fat disappeared from the serum cholesterol. An increase in diet also decreased (Fig. 2). For low energy cholesterol energy to produce. How well is expressed in the absorbed from the body for growth. Whole human

FIG. 1. The 34) and high (E J. Clin. Nutr. 8:

was based on the influences of such fats on serum cholesterol levels when they were tested as a single fat source in a liquid formula diet consisting of the test fat (19), sugar, and skim milk powder (Fig. 1). Fats of high polyunsaturated (linoleic) fatty acid content lowered the serum cholesterol level while fats of low linoleic acid content increased serum cholesterol levels. However, the diet of man contains both saturated and unsaturated fats. When a simulated mixture of fats based on fat disappearance data (20) was tested, the serum cholesterol level was not significantly different from an animal model fed corn oil. An increase in the protein level of the (21, 22) diet also depressed serum cholesterol levels (Fig. 2). For example, chickens fed a diet of low energy to protein ratio had lower serum cholesterol levels than chickens fed a high energy to protein ratio (22).

How well protein can be utilized generally is expressed as biological value, which represents the percentage of the protein nitrogen absorbed from food retained by the human body for growth and maintenance (23-25). Whole human milk is, expectedly, at the top

of the list, with a biological value of 95. Whole egg is nearly equivalent (biological value = 94) and is used as the standard when other foods are considered. Whole cow's milk has a value of 90. Some other values are shown in Table 4. Egg white, the principle ingredient in commercial egg substitute (26), has a biological value of only 83, considerably below that of whole egg. Soybean meal, wheat germ, and whole rice each have values of 75, while dried peas and beans are far down the list with a value of just 40.

Protein foods have also been considered in terms of digestibility by humans (27). Again, the best protein for human utilization and digestibility is from egg and milk. True digestibility of these proteins for adults is 97%, compared, for example, with 84% for polished rice, 79% for whole wheat, and 78% for soybeans. The digestibility of a mixed diet appears to fall (from about 92 to 85%) as large amounts of fruits or vegetables are added to the diet. Generally, the digestibility for whole-grained cereals and vegetables may be in the range of 85%, and refined cereals in the range of 90% (28). Furthermore, proteins

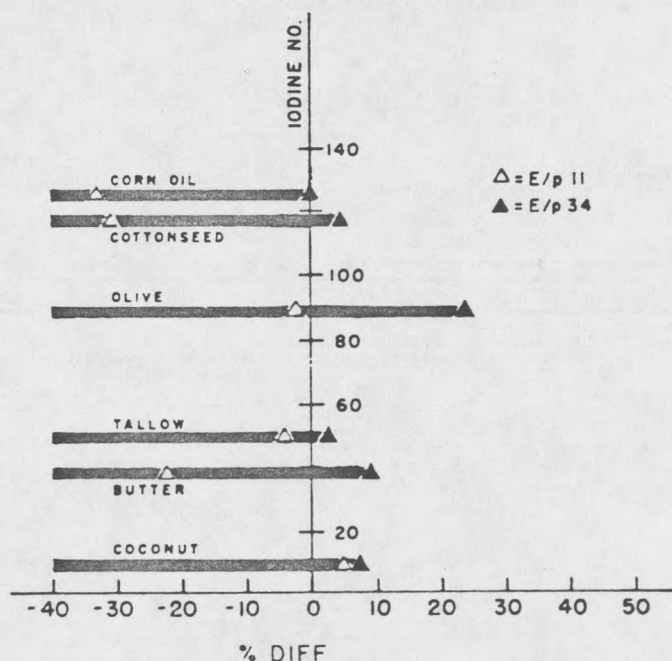


FIG. 1. The relationship of the iodine value of various fats to the serum cholesterol level in chicks fed low (E/P 34) and high (E/P 11) protein diets with deviations in mg/100 ml from chicks fed corn oil at an E/P ratio of 34. (Am. J. Clin. Nutr. 8: 62, 1960).

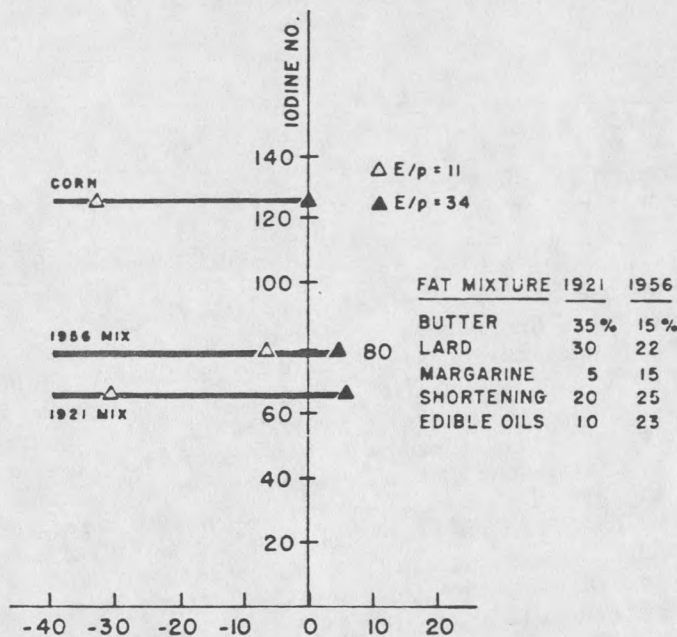


FIG. 2. The relationship of dietary protein level of the serum cholesterol level in mg/100 ml in chicks fed two composite fat mixtures as compared with chicks fed corn oil. At the higher protein level ($E/P = 11$), the simulated fat mixture based on consumption data in 1921 provided for a lower serum cholesterol value than the mixture based on consumption data in 1956, even though the former contained more animal fat. As in the National Diet Heart Study, the *trans* fatty acids in the higher percentage of margarine and shortening in the 1956 simulated mixture prevented as high a decrease in serum cholesterol levels as the 1921 mixture. (Am. J. Clin. Nutr. 8: 62, 1960).

decrease in ability to be utilized as the amount in the diet approaches the minimal amount needed (29, 30).

In the American diet meat, milk, cheese, and eggs furnish the essential amino acids that are necessary for the synthesis of tissue and the serum apoproteins which carry the serum lipid as serum lipoprotein. The lipoproteins have been divided into five classes: chylomicrons, very low density lipoproteins (VLDL), LDL, high density lipoproteins (HDL), and very high density lipoproteins. The percentage of lipid components gradually decreases, from 90% in the VLDL to approximately 50% in the HDL fraction or class (Table 5). Each lipoprotein class is heterogeneous with respect to its apoprotein constituents (Table 6). ApoA refers to the apoproteins that are primarily, but not exclusively, found in HDL. ApoB is the major apoprotein of LDL, but also comprises about 35% of the protein in VLDL. ApoC represents a group of proteins originally described in VLDL, but which are also present in HDL (31).

The apoproteins that have been sequenced to date (Table 6) contain varying numbers of the essential amino acids interspersed among the amino acids that can be synthesized *in vivo*. Their arrangement may be important to their lipid carrying capacity (32). Approximately 50% of the amino acids in the apoproteins cannot be synthesized *in vivo* and must, therefore, be furnished in the daily diet. An unbalanced dietary amino acid level can increase serum cholesterol levels (33). For example, chicks kept for 7 days on a diet containing synthetic amino acids plus 1.38, 1.74, or 2.82% arginine had serum cholesterol levels of 120, 103, and 125 mg/100 ml, respectively. Similar differences in serum cholesterol levels were noted after the deletion or excess addition of serine, alanine, leucine, lysine, proline, or methionine. The type of animal model also seems important; a complete protein such as a high level of casein seems more atherogenic to rabbits than soybean protein (34). The serum cholesterol level may, therefore, be as dependant on the amount and quality of dietary protein as on

the sources of cholesterol that are used in experimental diets. The several sources listed in the table are the "consumption in fat" nor do they have control over the amount of fat such as margarine, butter, and french fries. Commercial experiments are required to most restaurants, a part of the diet, burgers, the popular and increasing in

TABLE 4
Comparison of

Food

Egg, whole
Milk, cow's, v
Rice, whole
Rice, polished
Rice, experime
measureme
Soybean mea
Wheat, whole
Soybean curd
Soybean prot
Peanut protei
Peas, dried

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biological val
egg/milk/net
1954. (PA
System). ^g

TABLE 5
Composition

VLDL
LDL
HDL
VHDL^b

^a Unester.

the sources and dietary level of fat and cholesterol that have been used in previous experimental designs.

The seven recommendations that were listed in the Dietary Goals do not mention to the consumer the names of the foods "high in fat" nor does the consumer have any control over the composition of culinary fats, such as margarines, shortenings, and frying fats. Foods high in fat, such as potato chips and french fries, were not in nationwide commercial existence in 1909. Today, potato chips are routinely served with sandwiches in most restaurants and french fries are as much a part of the fast-food chains as the hamburgers they serve. That high fat foods are popular and profitable is shown by the rapid increase in doughnut and pizza shops. All of

the fried food items provide an overabundant amount of calories from saturated fats.

The major share of these food items are fried in an especially prepared heat stable hydrogenated vegetable fat which contains less than one percent linoleic acid (polyunsaturated fats (PUFA)). Although animal fats have been considered the main source of saturated fats, they contain more PUFA than frying fats of which more than 600 million pounds are consumed each year, or more than all of the corn oil produced in the United States. The intake of visible animal fats (butter, tallow and lard) has decreased from 21.9 in 1950 to 10.2 pounds/capita per year in 1975. On the other hand, the intake of vegetable fat has increased from 24.0 to 43.1 pounds/capita per year during the same time

TABLE 4
Comparison of biological value and digestibility of protein sources

Food	Biological value ^a	Digestibility ^b	NPU ^c	NPU egg/ NPU food	Safe level ^d	
					g/kg/day	g/day
Egg, whole	0.94 ^e	0.97 ^f	0.91	1.00	0.58	37
Milk, cow's, whole	0.90 ^e	0.97 ^f	0.87	1.05	0.61	39
Rice, whole	0.75 ^e					
Rice, polished		0.84 ^f				
Rice, experimental measurements					1.1	70
Soybean meal	0.75 ^e	0.78	0.58	1.57	0.91	58
Wheat, whole	0.67 ^e	0.79 ^f	0.53	1.72	1.00	64
Soybean curd	0.65 ^e	0.96 ^f	0.62	1.47	0.85	54
Soybean protein	0.64 ^e	0.88 ^f -0.95 ^f	0.56	1.63	0.95	60
Peanut protein	0.59 ^e	0.95 ^f	0.56	1.63	0.95	60
Peas, dried	0.40 ^e	0.85 ^f	0.34	2.68	1.55	99

^a Biological value = N retained by body/N absorbed. ^b Digestibility = N absorbed/N intake. ^c NPU = biological value × digestibility. ^d Safe level of protein = safe level of egg/milk protein × net protein utilization of egg/milk/net protein utilization of other food. ^e Wooster, H.A., Jr., Nutritional Data, Pittsburgh: H.J. Heinz Co., 1954. ^f PAG Bulletin, vol. V, no. 3, September, 1975. (Protein-Calorie Advisory Group of United Nations System). ^g Chiu, W-C. L. Thesis, University of Illinois, Urbana, Illinois, 1950.

TABLE 5
Composition of lipoproteins

	Total		Lipid ^a			
	Protein	Lipid	Triglyceride	Phospholipid	Cholesterol	Cholesterol ester
VLDL	10%	90%	50%	20%	8.5%	8.5%
LDL	25	75	10	30	10	50
HDL	50	50	10	50	5	25
VHDL ^b	60	40	12	76	1	8

^a Unesterified fatty acid. ^b Very high density lipoproteins.

TABLE 6
Amino acids in human serum lipoprotein

Essential amino acids	Apo A-I ^a	Apo A-II ^b	Apo B ^c	Apo C-I ^d	Apo C-II ^e	Apo C-III ^f	Arginine-rich ^g	Thin-line ^h
Isoleucine	0	2	48	3	1.0	0	13	37.2
Leucine	38	16	107	6	10.0	5	109	56.2
Lysine	19	18	71	9	5.0	6	48	58.8
Methionine	3	2	15	1	2	2	24	6.3
Phenylalanine	6	8	45	3	3	4	14	29.1
Threonine	10	12	59	3	10	5	38	44.8
Tryptophane	4			1		3	28	3.2
Valine	13	12	45	2	5	6	68	50.2
Total	93	70	390	28	36	31	342	285.8
Non essential amino acids								
Alanine	18	10	56	3	9	10	108	50.7
Arginine	15		27	3	1	2	106	17.6
Aspartic Acid	20	6	96	5	6	7	48	83.5
Glutamic Acid	49	32	109	9	18	10	233	99.4
Glycine	10	6	43	1	3	3	58	33.8
Histidine	5		18		0	1	13	10.7
Half-cystine	0	2	7		5	2	0	9.7
Proline	10	8	42	1	5	2	27	66.9
Serine	15	12	78	7	11	11	54	35.3
Tyrosine	7	8	30		6	2	14	25.9
Total	149	84	506	29	59	48	661	433.5
Grand total	245	154	896	57	95	79	1003	719.3
	Major				Minor			
Chylomicrons	ApoB, ApoC-I, ApoC-II, ApoC-III				ApoA-I, ApoA-II			
VLDL ⁱ	ApoB, ApoC-I, ApoC-II, ApoC-III, arginine-rich protein				Thin-line protein, ApoA-I, ApoA-II			
LDL	ApoB				ApoC-I, ApoC-II, ApoC-III, thin-line protein, arginine-rich protein			
HDL	ApoA-I, ApoA-II							
VHDL ⁱ	ApoA-I, ApoA-II							

^a Baker, H. N., R. L. Jackson and A. M. Gotto, Jr. Isolation and characterization of the cyanogen bromide fragments from the high-density apolipoprotein glutamine I. *Biochemistry* 12: 3866, 1973. ^b Jackson, R. L., J. D. Morrisett, H. J. Pownall and A. M. Gotto, Jr. Human high density lipoprotein, apolipoprotein glutamine II. *J. Biol. Chem.* 248: 5218, 1973. ^c Jackson, R. L., O. D. Taunton, R. Segura, J. G. Gallagher, H. F. Hoff and A. M. Gotto, Jr. Comparative studies on plasma low density lipoproteins from pig and man. *Comp. Biochem. Physiol.* 53b: 245, 1976. ^d Jackson, R. L., J. T. Sparrow, H. N. Baker, J. D. Morrisett, O. D. Taunton and A. M. Gotto, Jr. The primary structure of apolipoprotein-serine. *J. Biol. Chem.* 249: 5308, 1974. ^e Brown, W. V., R. I. Levy and D. S. Frederickson. Further characterization of apolipoproteins from human plasma very low density lipoproteins. *J. Biol. Chem.* 245: 6588, 1970. ^f Morrisett, J. D., J. S. K. David, H. J. Pownall and A. M. Gotto, Jr. Interaction of an apolipoprotein (ApoLP-alanine) with phosphatidylcholine. *Biochemistry* 12: 1290, 1973. ^g Shore, V. G. and B. Shore. Heterogeneity of human plasma very low density lipoproteins. Separation of species differing in protein components. *Biochemistry* 12: 502, 1973. ^h McConathy, W. J., and P. Alaupovic. Isolation and partial characterization of apolipoprotein D: a new protein moiety of the human plasma lipoprotein system. *FEBS Lett.* 37: 178, 1973. ⁱ VHDL, very high density lipoproteins.

period (35). Approximately two-thirds of those 43 pounds of vegetable fat was hydrogenated and sold as shortening or margarine. On the basis of per capita intake and percentage composition, the present 11.2 pounds/capita consumption of margarine furnishes more saturated fatty acids (palmitic and stearic acids) than butter or 2.03 and 1.95

pounds/capita. Furthermore, shortenings at 17.3 pounds/capita furnish to the American diet more saturated fat than meat or 3.68 and 3.59 pounds/capita.

Nutritionists would applaud including more fluid milk in the diet, as a glass of milk provides protein, minerals, and vitamins. A glass of soft drink supplies only unneeded

TABLE 7
Comparison
unsaturated
soft and h

	Source
Hard fat	
Milk fat	
Human milk	
Human butter	
Soft fat	
Safflower oil	
Cottonseed oil	
Corn oil	
Palm oil	

^a Dairy
Westport, C

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Nonfat milk
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TABLE 7
Comparison of total saturated and unsaturated fatty acids, in soft and hard fats^a

Source of fat	Saturated		Unsaturated	
	-C ₁₆	C ₁₆₊₁₈	C18:1	C18:2
	%			
Hard fat				
Milk fat	20	37	36	5
Human milk fat	17	35	37	8
Human body fat	4	32	52	9
Soft fat				
Safflower oil	1	12	20	67
Cottonseed oil	1	23	29	45
Corn oil	1	12	45	42
Palm oil	2	46	43	9

^a Dairy Lipids and Lipid Metabolism, (Chapt. 6), Westport, Conn.: Avi Publishing Company, Inc., 1968.

calories at a higher cost than a glass of milk. Nonfat milk contains less fat than whole milk or less than 1 and 3.5%, respectively. However, the difference in calories between a glass of nonfat milk and a glass of whole milk is 80 calories, or the amount of fat equivalent to four large potato chips. From a nutritional point of view, it would be more judicious to drink the whole milk and eat four less potato chips at the noon luncheon. Milk fat contains only 3% more saturated fatty acids less than C₁₆ in chain length and approximately the same amount of C₁₆ and C₁₈ saturated fatty acids as human milk fat (Table 7). The fat in the whole milk supplies the essential fat soluble vitamins, vitamins A, D, and E. The fat in potato chips supplies nothing but unneeded calories.

The greatest error in the Dietary Goals for the United States rests in the recommendation to decrease the consumption of "butter, eggs, and other high cholesterol sources" because it links heart disease with eating our best sources of protein, which are eggs, meat, and dairy products. In order for the protein intake to remain at its present level of 12% of the total diet, a source of vegetable protein food items equivalent to approximately 30 pounds of complete protein per capita would have to become available to replace meat and eggs in the diet. Less than 2 pounds/capita of soybean protein is presently being consumed as a part of already acceptable food items, such as bakery and meat products. Unless entirely new food products can be developed

to take the place of meat and eggs, there is little likelihood that the consumption of vegetable protein intake can be increased fifteen times. The production of all nuts, including peanuts, is also only 2 pounds/capita per year. Furthermore, the climate is too severe to grow pecan, almond or walnut trees on a commercial scale on the Midwest farmland that now supplies the corn and soybean for animal protein production. Therefore, there is no acceptable way, at present to convert the protein in corn and soybeans into food items that are suitable to the American palate.

Butter is a fat source and not a protein source as shown by the fat, protein and cholesterol composition of these basic food items (Table 8). Butter contains less than 5% linoleic acid. However, Americans are not deficient in linoleic acid (PUFA) (36). The percentage of linoleic acid in the erythrocytes (red blood cells) of patients in Bulgaria, a country with a lower coronary heart disease rate than the United States, indicated no significant difference from patients in American hospitals (Table 9), nor from Americans with no apparent signs of heart disease. The linoleic acid content of erythrocytes can be increased by including a source of PUFA in the diet or decreased by including a saturated fat in the diet (37).

To reduce salt consumption could mean using the salt shaker at the table less often. However, the major source of excess salt is provided by potato chips, salted peanuts, pizzas, salted snack foods, and salt preserved vegetables, such as sauerkraut and pickles.

The furor over the banning of saccharin in noncalorie soft drinks should alert the Select

TABLE 8
Fat, protein, and cholesterol in 100 g of butter, eggs, cheese, meat, and milk

Food item	Fat ^a	Protein	Cholesterol ^b
			µg
Butter	81.0	0.6	185
Eggs (whole)	11.5	12.8	468
Cheese (cheddar)	32.3	23.9	160
Meat (chuck)	16.0	18.6	125
Milk (whole)	3.9	3.5	13

^a Wooster, H. A., and F. C. Blanck. Nutritional Data, H. J. Heinz and Co. 1950. ^b Lange, W. Cholesterol, phytosterol and tocopherol content of food products and animal tissue. J. Am. Oil Chem. Soc. 27: 414, 1950.

TABLE 9
Comparison of the mixed fatty acid compositions of lipids extracted from the red blood cells of Americans, Finns, Bulgarians, and rural Romanians

Fatty acid	Americans	Finns	Bulgarians ^a	Rural Romanians ^b
		%		
Palmitic	29.9 ± 2.9	25.22 ± 4.2	34.5 ± 3.0	31.8 ± 1.8
Stearic	14.2 ± 0.2	11.54 ± 0.74	12.0 ± 0.5	19.8 ± 0.9
Oleic	24.5 ± 1.9	13.22 ± 1.6	30.6 ± 2.0	17.7 ± 0.3
Linoleic	13.7 ± 0.2	10.9 ± 1.8	11.3 ± 2.1	13.0 ± 0.6
Arachidonic	9.8 ± 0.5	8.48 ± 0.7	7.9 ± 2.7	12.2 ± 0.1
Average <i>trans</i> acids	2.0	2.6	Trace	0.0

^a Courtesy of Dr. Peter Ilinov, Institute of Nutrition, Sofia, Bulgaria, who is presently redoing samples on a column packed with 15% OV-275 coated on 100/120 mesh Chromosorb P, AW-DMCS (Supelco, Inc.). ^b Samples from Romania obtained through the courtesy of Drs. M. Cucuianu and T. Popescu, Clinica Medicala I. Cluj, Str. Clinicilor 3, Cluj, Romania.

Committee of the Senate on Nutrition that Americans have a "sweet tooth" and that it is difficult to decrease sugar consumption. The data cited by the committee (2) indicates that household consumption of sugar has actually decreased from 52.1 pounds/capita in 1909 to 24.7 in 1971 and a further decrease in household use is unlikely. The greatest increase in sugar consumption was due to soft drinks which increased from 3.5 pounds in 1909 to 22.8 pounds in 1971. Diet conscious consumers are aware that a 12-ounce bottle of soft drink furnishes 120 cal and so they are now spending a total of almost 2 billion dollars for sugar-free soft drinks. It is ironic to note such consumers drink the sugar-free soft drinks while munching potato chips or some other high calorie food. They are not aware that the tasty snack food items supply more calories than a soft drink would have.

The recommendations to decrease sugar consumption and to increase starch consumption is based on the observation that sugar is absorbed more rapidly from the intestinal tract than starch and if in surfeit supply will be converted to fat *in vivo*. However, starch (carbohydrate) foods are as readily converted to fat as sugar, if not used as muscle energy or body heat (38-40).

If sugar is used judiciously as part of a nutritionally balanced diet, it should not be considered a "risk factor" any more than the fat in such a diet. In 1909, the household consumption of sugar was greater than in 1971 because sugar was used in the now lost art of home preservation of fruits, jams and jellies. Sugar has a vital function in food

technology as it serves as a preservative for fruit and therefore helps add to the supply of vitamin C and other water soluble vitamins. The preservation of these fruits without sugar would require more refrigeration and energy expenditures than is presently available or will be available in the future. In fact, the use of sugar as a means of preserving fruit juices should be expanded so that such juices could become economical enough to replace artificial fruit juices and soft drinks. Sugar in combination with fat has also made possible a large array of desserts which add to the pleasure of eating a well balanced meal and assures that human food has more esthetic interest than dog and cat food. Optimum nutrition can involve more than a minimum of essential nutrients. It should involve the taste buds, aroma, and the appearance of food as well. There is no reason for a well-balanced diet to be unappetizing and to take heroic efforts to consume. The American supermarket abounds in nutritious foods and food items which please the palate. A choice based on essential nutrients and consumed in amounts sufficient to maintain correct weight will help to maintain optimum health.

Is susceptibility to heart disease inherited?

Studies on the hyperlipidemia in individuals from families that have had a greater than normal incidence of CHD has both confused and aided the search for the cause of CHD. It has confused the search, because such studies focused attention on the high serum cholesterol levels in these individuals

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(41-45). The high serum cholesterol levels could be duplicated in animal models that had been fed a massive amount of cholesterol over an extended period of time (46-50). However, such observations have resulted in confusing optimum nutrition with hyperlipidemia. The 1 to 2% of crystalline cholesterol that was necessary to induce atherosclerosis in an animal model in a reasonable period of time was equivalent to the amount of cholesterol in 80 to 120 eggs per day. Cholesterol in the form of egg yolk has also been used (51, 52). These experimental conditions have been projected to a recommendation that no eggs or a minimum amount of eggs should be used in the American diet (53). This is confusing experimental manipulation with the optimum nutrition that eggs provide to the diet.

Three recent, independent studies (36, 54, 55) have shown that including two whole eggs per day in the diet had no significant effect on the serum cholesterol value of normal human subjects. Furthermore, an amount of cholesterol equivalent to 40 eggs per day did not increase the serum cholesterol level of swine significantly (56).

Estimates of gene frequency have indicated that only 0.6 to 1.0% of individuals tested in the United States were hyperlipidemic (56). A study of the mutation that is responsible for such hyperlipidemia has aided in the search for the cause of CHD. It is believed (57-60) that normal subjects maintain a plasma LDL-cholesterol level of about 120 mg/dl and are generally protected against myocardial infarction for about 60 years. Subjects with the heterozygous form of familial hypercholesterolemia must maintain a plasma LDL-cholesterol level that is elevated 2- to 3-fold, and in general develop myocardial infarctions 20 years earlier than do normal subjects. Subjects with the homozygous form of familial hypercholesterolemia are believed to maintain plasma LDL-cholesterol levels that are at least six-fold higher than normal, and as a result they develop myocardial infarctions usually before age 20.

It has recently been suggested (60) that a high HDL-cholesterol level may be protective against CHD. However, the serum cholesterol and HDL level of human subjects with proven coronary blockage, as shown by angiography, were as variable as "normal" sub-

jects of similar age, sex, blood pressure, and total serum cholesterol levels (61). An LDL-cholesterol level below 120 mg/100 ml may be desirable, but it does not provide complete protection against CHD as seven out of 55 patients that were subjected to coronary bypass surgery (62) had a low serum cholesterol level. Coronary artery blockage is, therefore, more complex than measurements of HDL and LDL serum cholesterol levels would indicate.

LDL lipoprotein infiltration does not seem necessary to the initiation of arteriosclerosis or atherosclerosis

The methodology developed by Imai et al. (63, 64) and applied by Kamio et al. (65-68), and Taura et al. (69-70) has made it possible to study, in animal models kept on a low-fat cholesterol-free diet, the sequence of events that contribute to arteriosclerosis (intimal thickening) and atherosclerosis (lipid deposits in the thickened intima). They found, in studies on the ultrastructure, with the aid of a transmission electron microscope, degenerated smooth muscle cells in the arterial tissue from the fetus of sows kept on low fat cholesterol-free diets. After birth, the frequency of degenerated smooth muscle cells increased with age. The gradual intimal thickening which resulted from degenerated smooth muscle cell accumulation seemed to occur without any apparent disruption of the endothelium. The protective effect of the endothelium against serum lipid infiltration was demonstrated by mechanical denudation of the aorta of weanling swine (70). Seven days after denudation, prominent intimal thickening was observed; the intima contained large numbers of modified and degenerated smooth muscle cells, and collagen fibers in the interstitium of the intima. No such changes were apparent in the aorta of weanling swine which had not been subjected to denudation.

The rate at which degenerated smooth muscle cells accumulated in the intima could also be accelerated by including angiotoxic factors such as oxidized sterols in the diet. Raised lesions were found in the thoracic aorta of weanling piglets after only one month of feeding 25,000 IU of vitamin D₃

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per pound of feed. Electron microscopy (EM) indicated that the grossly normal areas of the aorta from weanling swine that had been fed 25,000 IU of vitamin D₃ per pound of basal ration for 3 months had a higher frequency of degenerated smooth muscle cells than the grossly normal areas of swine fed the initial D-unsupplemented commercial ration (Fig. 3A). A comparison of the EM of such thoracic aorta tissue with the EM of the plugs of thoracic aorta that were obtained as a by-product of elective coronary bypass surgery revealed identical changes in human thoracic aorta tissue (Fig. 3B). Sections of the thoracic aorta obtained from weanling piglets that had been fed 12,500 IU vitamin D₃ per pound of ration (approximately 12.5 times more than in the commercial ration) for 3 months also contained the same type of lipid-free intima thickening as the plug of thoracic aorta obtained as a by-product of coronary bypass surgery (Fig. 3A). However, they developed extensive lipid deposits and coronary occlusion by the time they were six months of age (71). The EM of the thoracic aorta from 3-year-old female swine (Fig. 4A) that had been kept on the low-fat cholesterol-free unsupplemented ration was identical to the EM of the lipid-laden plug of thoracic aorta from a 60-year-old male subjected to elective coronary bypass surgery (Fig. 4B). Coronary artery tissue is not available as a by-product of elective coronary bypass surgery and, therefore, a direct comparison between the EM of swine and human coronary tissue is not possible. However, the results that were obtained after a short period of vitamin D supplementation of weanling swine indicated that atherosclerosis could have been present in the coronary arteries of patients whose thoracic aorta exhibited only arteriosclerosis (69). Although supplementation with vitamin D does not seem to influence lipid metabolism (72, 73), it has been reported to increase the concentration of acid mucopolysaccharides in arterial (74) and kidney tissue (75). The exact role of vitamin D in the formation of raised lesions in the intima (68) deserves further study (76).

It is possible that the frequency of smooth muscle cell death can be accelerated in the human fetus before birth by the presence of angiotoxic risk factors in the diet. Pregnant women are routinely advised by their obstre-

tricians to supplement their diet with vitamin D, although the 400 IU/quart of milk is sufficient to provide all the vitamin D that is required. A vitamin capsule contains 400 IU vitamin D and if one capsule per day is prescribed, the pregnant woman adds an extra 400 IU of vitamin D over and above the approximately 2435 IU which is the average per capita intake per day from vitamin D-enriched foods or six times the National Research Council requirements. Approximately 10,000 pounds of vitamin D₂ and D₃ per year are added to animal feeds, and thus consumed indirectly in meat or eggs, or added directly into baby foods, imitation dairy products, sweet sauces, beverages, prepared breakfast cereals, margarine, macaroni, noodles, farina, and flour (77). Bread and rolls contain 250 to 750 IU/pound. In addition, considerable amounts of vitamin D is present naturally in various foods; for example, 2700 IU/pound were found in fish (78), other sea foods probably contain similar levels of vitamin D.

As vitamin D is a fat soluble vitamin, it is stored and accumulates in all body tissues. For example, human muscle tissue contained more vitamin D than swine fed a commercial ration which contained 14 times more than the National Research Council requirements (78). Furthermore, the intake of vitamin D is three times higher in babies 6 to 11 months of age than in adults (77). The baby is usually supplemented with 2 drops of vitamin D (200 IU/drop) which adds to the vitamin D provided by the lactating mother. However, because the mother weighs more than the baby, the vitamin D is more diluted in her than in her baby. The dose per kilo of body weight is approximately 20 times higher for the baby than for the mother. Such a level of vitamin D may accelerate the frequency of degenerated smooth muscle cell death in the vitamin D sensitive intima of the coronary arteries. Later, the teenager who eats foods that contain oxidized cholesterol may further accelerate the rate at which degenerated smooth muscle cells form in the intima.

Imai et al. (79) has shown an oxidized sterol, 25-hydroxy cholesterol, to accelerate smooth muscle degeneration. This derivative of oxidized cholesterol was found in supposedly pure crystalline cholesterol. Impurities were concentrated from several lots of cholesterol by recrystallizing cholesterol from




FIG. 3.
foam cells
90 mg/100
or extracel



FIG. 3A. Thoracic aorta of 6-month-old swine fed 25,000 IU of vitamin D₃ per pound ration for 3 months. (No foam cells or extracellular lipids, intimal thickening with calcification of the media, $\times 6200$). Serum cholesterol level, 90 mg/100 ml. B, thoracic aorta of a 40-year-old man subjected to elective coronary bypass surgery. (No foam cells or extracellular lipid, intimal thickening only, $\times 14,000$). Serum cholesterol level, 312 mg/100 ml.

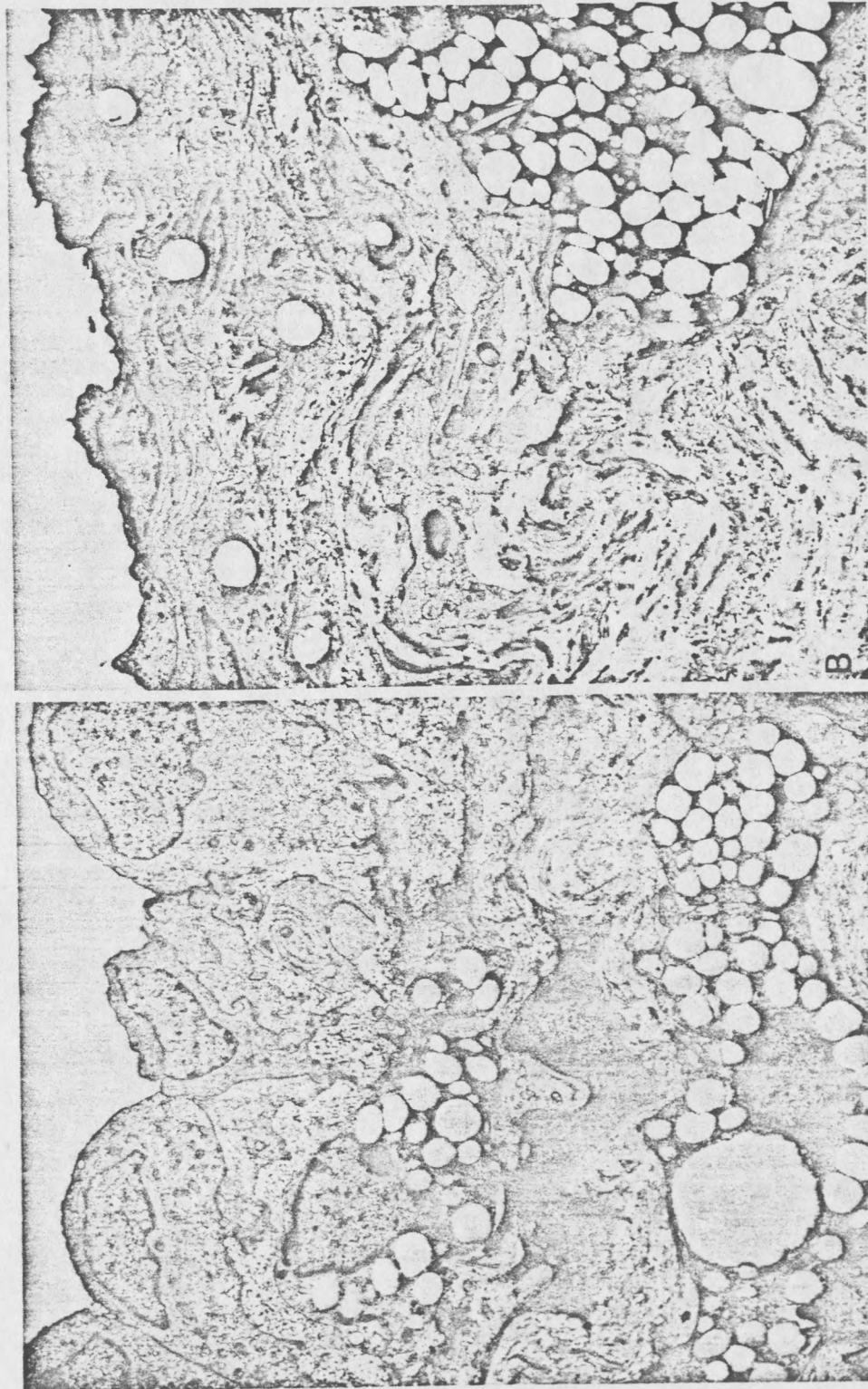


FIG. 4A. Thoracic aorta of 3-year-old female swine (foam cells with electron opaque lipid inclusions, $\times 6000$). Serum cholesterol level, 130 mg/100 ml. B, thoracic aorta of 60-year-old male subjected to elective coronary bypass surgery (foam cells with electron opaque lipid inclusions, $\times 6000$). Serum cholesterol level, 272 mg/100 ml.

methanol solvent, and even under vacuum the products are cholesterol. The frequency of cholesterol and induced cholesterol 24 hr after surgery.

Although it is difficult to be obtained, cholesterol is used to dehydrate "used" frying fat as much as fat in our pounds of each year, oxidized cholesterol or even fried in beef Donald's, but they contain 80% of fries (80% of fries) and may contain teenagers and commercial cholesterol content of such foods in many.

The advanced coronary artery disease by the dysfunction of the inner system. On the other hand, it has been shown that of the intima diffusion coefficient (81). A significant intima by generated inadequate media. The effect of having stress affects oxygen nutrition.

Lipid deposits in aorta tissue in vitro (82, 83) and aorta tissue influences studies with

methanol solution, retaining the mother liquor, and evaporating the residue to dryness under vacuum. This concentrate contained the products of spontaneous oxidation of cholesterol. The concentrate increased the frequency of dead aortic smooth muscle cells and induced focal intimal edema in rabbits 24 hr after gavage at 250 mg/dk.

Although the experimental data still need to be obtained, it is conceivable that oxidized cholesterol is present in any fat that has been used to deep fry chicken or fish; such "used" frying fats have been found to contain as much as 214 mg of cholesterol per 100 g of fat in our laboratory. As over 600 million pounds of fat are used in frying operations each year, the potential exists for forming oxidized cholesterol in the frying of fish, poultry or even in potatoes if the potatoes are fried in beef tallow. Data provided by McDonald's, Inc. for french fries indicate that they contain 12 mg of cholesterol per 100 g of fries (80). The smooth muscle cell degeneration accelerated by vitamin D in infancy may continue with oxidized cholesterol in the teenagers who are exposed to easily available commercially fried foods. The oxidized cholesterol content and the high calorie content of such foods could be reduced by preparing them in microwave ovens.

The advanced stages of atherosclerosis in the coronary artery may also be accelerated by the dynamics of metabolism and by lipid infiltration once the endothelium is breached. The inner arterial wall contains no vascular system. Oxygen must come from diffusion and it has been suggested that the thickness of the intima wall is very close to the limiting diffusion distance for oxygen in intimal tissue (81). A small change in the thickness of the intima by an increase in the number of degenerated smooth muscle cells may lead to an inadequate oxygen supply in the intima and media. These tissues are in constant danger of having a deficient oxygen supply and any stress affecting the supply will affect the oxygen nutrition, i.e., the energy supply to them.

Lipid droplets have been found to accumulate in hypoxic cells both in vivo and in vitro (82, 83). Metabolic studies with swine aorta tissue (84) indicates that oxygen supply influences the rate of lipid synthesis. In vitro studies with incubated swine aorta indicated

that less radioactively labeled acetyl coenzyme A was converted to lipid when the oxygen supply was optimum. Therefore, one can visualize a metabolic change as the intima gradually thickens. The acetyl coenzyme A that is not used as an energy source is synthesized into lipid in situ. Lipid begins to accumulate in the interstitial spaces and adds further to the thickness and inertness of the intima. The endothelium cells may be first stretched thin (70), then a gap develops, and at this point may allow lipoprotein to infiltrate into the intima adding further to intimal thickening. The thickening may then become great enough to rupture the endothelium layer completely and cause the "rubber tire-like patch" that pathologists have noted on autopsy. Whether lipoproteins that contain *trans* fatty acids infiltrate more easily or change the characteristic of the cell membranes in the presence of oxidized cholesterol is also unknown, but worthy of study.

The kind of unsaturated fatty acids in heart mitochondria may influence heart composition and function

Although the aorta and coronary vessels have been studied extensively as key factors in CHD, the lipid composition of the heart muscle itself may be crucial to its function (85-88). The working unit of the heart muscle, the mitochondria, contain a high proportion of PUFA. Furthermore, dietary fats influence this composition (Table 10). For example, the phospholipid in mitochondria isolated from weanling rats fed corn oil contained a much higher percentage of PUFA than those fed hydrogenated fat after only 1 week on the diet. There was considerable incorporation of dietary elaidic acid into all three phospholipid fractions. This *trans* isomer represented nearly half of the total octadecenoic acid.

The mitochondria from the hearts of rats fed hydrogenated fats oxidized fatty acids at a slower rate than those from rats fed corn oil. Heart mitochondria isolated from rats which had been fed corn oil had a higher rate of oxygen uptake and a higher rate of energy (ATP) synthesis than the heart mitochondria isolated from those fed hydrogenated fats (89). Similar results were obtained when the

FIG. 4A. Thoracic aorta of 3-year-old female swine (foam cells with electron opaque lipid inclusions, $\times 6000$). Serum cholesterol level, 130 mg/100 ml. B, thoracic aorta of 60-year-old male subjected to elective coronary bypass surgery (foam cells with electron opaque lipid inclusions, $\times 6000$). Serum cholesterol level, 272 mg/100 ml.

TABLE 10
Relative composition^a of total fatty acids of separated phospholipids
in pooled rat heart mitochondria from rats fed
corn oil or hydrogenated fat^b

One week	Corn oil ^c			Hydrogenated fat ^c		
	CL	PE	PC	CL	PE	PC
			%			
16:0 ^d	1.4	10.6	19.7	12.4	20.1	20.1
16:1 ω 7	1.2	1.0	2.7	2.9	1.5	3.6
18:0	2.8	22.3	24.5	7.5	23.2	23.2
18:1- <i>cis</i> ω 9	14.0	19.2	15.5	21.1	18.5	19.2
18:1- <i>trans</i> ω 9				21.5	18.5	17.0
18:2 ω 6	68.3	16.2	21.2	29.2	1.7	4.6
18:3 ω 3	0.3	0.1	0.1			
20:1 ω 11	2.1	0.5	1.6	0.8		
20:3	1.7	0.8	0.5	2.0	2.3	3.5
20:4 ω 6	2.6	14.8	11.4	0.1	6.4	5.5
22:1 ω 9	0.2	0.1	0.1			
22:6	5.0	14.3	5.2	2.5	8.9	3.2

^a Values are expressed as areas percent of the total peak areas. Results are the average of triplicate gas chromatography analysis. ^b Hsu, C. M. L., and F. A. Kummerow. Influence of elaidate and erucate on heart mitochondria. *Lipids* 12: 486. 1977. ^c CL, cardiolipin; PE, phosphatidylethanolamine; PC, phosphatidylcholine. ^d Carbon atom chain length: number of double bonds.

heart mitochondria isolated from swine were incubated with oleyl or elaidyl carnitine (90). Furthermore, elaidyl carnitine inhibited the oxidation of oleyl carnitine.

As it has been shown that the fatty acids flow into the mitochondria at an increased rate during oxygen deficit, the ability to use fatty acids as an energy source may become crucial (91). In a "heart attack" the heart beat can increase to as high as 280 beats/min which puts a tremendous strain on energy needs. When additional ATP was added to isolated heart mitochondria the rate of oxidation increased four times (92). As the fatty acids furnish the ATP, the ability of the various fatty acids to furnish ATP may be crucial to the tremendous energy demand at 280 beats/min. It is understandable why the heart muscle would stop functioning from sheer exhaustion due to a lack of ATP. Whether oleic acid is a better source of energy than elaidic acid in the intact heart at such a critical time would seem to deserve thorough testing and further research.

Changes in food processing procedures and food consumption that would improve nutrition

Changes in food processing procedures are difficult to implement as items of food and

drink become tradition that few would question. Basic milling technology of wheat has not changed since biblical times, and it would cost millions of dollars to install the fat extraction equipment that would be necessary to the stabilization of wheat germ. The fat free germs could be recombined with the flour to provide more nutritious bread. This bread could also be fortified with 5% dried yeast as the bread in Romania and could in addition be fortified with the B-complex vitamins that have become commercially available since World War II, the time at which thiamin, riboflavin, and niacin were added to flour. Flour could also be fortified with trace minerals. Bread baked from such a flour and fortified with milk solids has been biologically tested and has been found to be far superior in biological value to the present commercially available bread (93).

Wheat germ oil is a rich source of vitamin E and could be sold as a salad oil or blended with shortenings so that BHT and BHA need no longer be used as fat stabilizers. These stabilizers against oxidation are not allowed as additives in Western Europe because of their toxicity and could be replaced by an economical source of vitamin E such as in unrefined wheat germ oil.

To date, shortenings, frying fats, and margarines have been formulated purely on the

basis of technical conditioning of fat instead of "rearrangement" vegetable shortening" use lard instead whenever economic expensive than contain emulsifier glycerides, or a citric acid and contains antioxidant additives is used on fat absorptive been tested to c

The technology proved heat stable frying operation frying fats. The enough fat to a fresh frying oil. PUFA that polyfoaming that the fats depressed g fed a diet of l. The present firm readily. However, certain *trans* fatty acids provide trans index (98) and smooth spreader without consistency of color are more stable and therefore a fat.

Over 30 million trying to convert to others; in freedom from l.tain various am is no indicator 11). One margin than another almost identical garine furnish fat intake, the contains linole something margarine provide

basis of technology. It has taken years of conditioning of the consumer to accept shortening made from "saturated" vegetable fat instead of "saturated" animal fat. The "rearrangement" of lard has improved the "shortening" value of lard so that it is equal to vegetable shortening. Commercial bakers use lard instead of vegetable shortening whenever economy is needed, as lard is less expensive than vegetable shortenings. Both contain emulsifiers such as mono- and diglycerides, or acylated fatty acids of tartaric or citric acid and silicones. In addition, bread contains antistaling agents. None of these additives is used in Europe. Their influence on fat absorption and atherosclerosis has not been tested to date.

The technology of frying fats has so improved heat stability that many deep fat frying operations no longer need to discard frying fats. The product to be fried absorbs enough fat to allow a continual addition of fresh frying oil. Previous frying oils contained PUFA that polymerized and caused so much foaming that they had to be discarded. Such fats depressed growth in rats which had been fed a diet of low nutritional value (94-97). The present frying oils do not polymerize readily. However, they, like margarine, contain *trans* fatty acids. In margarine these fatty acids provide triglycerides of various solid fat index (98) and allow for the fabrication of a smooth spread that can be kept in the refrigerator without taking on the hard waxy consistency of cold butter. The *trans* fatty acids are more stable to heat than the *cis* fatty acids and therefore add to the heat stability of the fat.

Over 30 million dollars a year is spent on trying to convince physicians and their patients that particular margarines are superior to others; in fact, by implication, promises freedom from heart disease. Margarines contain various amounts of linoleic acid but price is no indicator of linoleic acid content (Table 11). One margarine can cost three times more than another brand and yet both can be almost identical in linoleic acid content. Margarine furnishes only 10% towards the total fat intake, the other 90% of the fat intake also contains linoleic acid. To claim that there is something magical about the 10% that margarine provides is a delusion. When margar-

ine was tested against butter under clinically controlled conditions the difference in serum cholesterol values was insignificant. (Promise Margarine Clinical Studies, Lever Brothers Co., 1972. Personal communications.)

A margarine can be prepared in two ways: by substituting elaidic (mp 43.7 C) instead of oleic acid into the triglyceride or by using a mixture of triglycerides of various melting points. Margarine D that was used in the National Heart Study (5) is an example of the former and margarine B and C examples of the latter (Table 12). Margarine D contained 38% *trans* (elaidic) acid and 12% linoleic acid of questionable essential fatty acid content. Margarine B and C contained only 15% *trans* fatty acids and 62% linoleic acid, yet it had a melting point only 2 C lower than margarine D.

The serum cholesterol level of subjects fed diet B and C in the National Diet Heart Study averaged 20 mg/100 ml less than those fed diet D (Table 13). It is possible that the high *trans* fatty acid content of the margarine used in diet D may have cancelled out the serum cholesterol lowering influence of the 12% linoleic acid in this culinary fat. When swine were fed a hydrogenated fat which contained 50% *trans* fatty acids, their serum cholesterol level was 14 mg/100ml higher than those fed animal fat even though the animal fat contained 25% more saturated fatty acids. The serum lipoproteins isolated from weaning swine fed a basal diet that had been supplemented with animal or hydrogenated vegetable fat for 6 months did not differ significantly in composition (56). The aortas from such fat supplemented animals did not differ significantly in the degree of atherosclerotic involvement. However, mature swine that had been supplemented with hydrogenated fat for 8 months had a greater number of lesions than swine supplemented with animal fat (99).

The report for the Intersociety Commission for Heart Disease Resources listed data that showed a direct correlation between percent of calories from saturated fat and CHD death and serum cholesterol levels and CHD death. Such a correlation can also be made between hydrogenated fat consumption and CHD death. Hydrogenated fats are not available in quantity in Spain, Italy, Yugoslavia, Greece,

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TABLE 11
Fatty acid analysis of margarines, July 9, 1976

Composition	Kraft Parkay (stick)	Kraft Squeeze Parkay	Kraft Miracle Margarine	IGA* Table Right	IGA Soft	Area %		Imperial Soft	Mazola Stick	Chiffon Soft	Fleischmann's (stick)	Fleischmann's Soft
						Blue Bonnet	Imperial (stick)					
Cost/pound	0.39	0.66	0.55	0.38	0.49	0.47	0.49	0.67	0.65	0.66	0.69	0.76
IGA, Urbana	0.46	0.67	0.49			0.56	0.65	0.71	0.67	0.68	0.72	0.79
Eisner, Urbana	0.04						0.08	0.10	0.17	0.04	0.06	0.04
12:0	0.10						0.27	0.28	0.14	0.09	0.06	0.04
16:0	11.38	11.65	11.60	0.05	0.08	0.11	16.92	16.62	11.86	10.82	11.50	11.29
16:1 ω 7 c	0.13	0.14		0.18	0.18	0.20	0.11	0.21	0.19	0.19	0.24	0.22
18:0	7.05	4.79	6.59	7.38	6.91	7.55	5.33	5.06	6.78	6.65	6.72	6.49
18:1 ω 9 c	35.22	30.38	29.30	26.20	27.02	35.47	28.53	26.56	27.82	32.04	25.24	24.76
18:1 ω 9 t	31.41	6.88	14.26	21.70	13.19	30.77	15.29	13.70	20.07	15.57	22.03	11.36
18:2 ω 6 c	9.78	39.69	33.01	28.84	34.67	9.37	23.25	24.82	30.33	28.83	32.08	43.98
18:2 ω 6 t ₁ c ₁	4.07	2.49	2.49	1.05	1.80	3.75	6.95	8.59	1.68	3.41	1.24	0.73
18:3 ω 3	0.50	2.87	2.75	3.78	4.13	0.66	2.02	2.22	0.54	1.89	0.60	0.83
Others	0.32	0.66		0.28	0.68	0.61	1.25	1.84	0.42	0.47	0.29	0.30
Total trans (GLC)	35.48	9.87	16.75	22.75	14.99	34.50	22.24	22.29	21.75	18.98	23.56	12.41
Total trans (IR-7)	35.00	15.50	31.50	26.75	20.50	37.75	30.75	26.50	27.70	25.50	28.50	16.50
Total trans (IR-HEMHRF)	35.50	14.25	20.80	25.00	16.50	33.00	22.50	24.25	21.50	23.50		

* A house brand margarine which has 65% of the market. *Trans* fatty acids act like saturated fatty acids in the body and should be considered as saturated fatty acids even though they are "unsaturated" in structure. J. Am. Oil Chem. Soc. 54: 279, 1977.

Bulgaria, or 15 European Health Orga from CHD th Poland, and fats are cons of the *trans* d cytes from Finns, Bulgacans indicat USA and F than those fr (Table 9). F differ as tho plied by anir

TABLE 12^a
Composition of

M	
Total fat (%)	
Liquid safflo	
Hydrogenate	
Hydrogenate	
Wiley meltin	
% Solids	50 F
	70 F
	80 F
	92 F
	104 F

Fatty acid com
Myristic
Palmitic
Stearic
Oleic
Linoleic
Linolenic
P/S ratio
Trans Acids
Essential Fa

^a Courtesy c

TABLE 13
Influence of cu
human subject

Saturated
Monounsatur
Polyunsaturat
Trans
Serum cholest

^a National I

Bulgaria, or Romania which, according to a 15 European Nation Study by the World Health Organization, have lower death rates from CHD than England, Germany, Sweden, Poland, and Finland in which hydrogenated fats are consumed in quantity. A comparison of the *trans* fatty acid content of the erythrocytes from blood samples obtained from Finns, Bulgarians, Romanians, and Americans indicates that blood samples from the USA and Finland contain more *trans* acids than those from Bulgaria and rural Romania (Table 9). Furthermore, the *trans* acids may differ as those from rural Romania were supplied by animal rather than hydrogenated fat.

The rate of CHD in these countries according to data published by World Health Organization is higher for Finland and lower for Romania and Bulgaria. The people of Finland consume 60% of their total fat intake as hydrogenated fat and 40% from dairy and animal fats that have been considered the only saturated fat consumed in Finland to date (100). On the basis of these data, it seems as valid to compare hydrogenated fat consumption with CHD as to compare cholesterol or animal fat consumption with CHD.

In the attempt to provide cholesterol free substitutes for cream, meat, and eggs, food processors have provided "dairy creamers"

TABLE 12^a
Composition of margarines used in National Diet Heart Study

Margarines used in national diet heart study	B	C	D
Total fat (%)	50	80	80
Liquid safflower	40	64	
Hydrogenated soybean (62 IV)	10	16	
Hydrogenated soybean/cottonseed			80
Wiley melting point, C		35.0	37.4
% Solids			
50 F (approx)	10		30
70 F (approx)	7		19
80 F (approx)	6		14
92 F (approx)	3		5
104 F (approx)	0		0
Fatty acid composition (%)			
Myristic	t		t
Palmitic	9		12
Stearic	6		9
Oleic	22		66
Linoleic	62		12
Linolenic	0.3		0.3
P/S ratio	4.0		0.6
Trans Acids (%)	12-15		38 ± 3
Essential Fatty acids	60 ± 3		?

^a Courtesy of Anderson, Clayton and Company Foods Division, Sherman, Texas.

TABLE 13
Influence of culinary fats on serum cholesterol levels in human subjects and mature swine

Culinary fat	Human subjects ^a		Mature swine ^b	
	B and C	D	Beef tallow	Hydrogenated fat
			%	
Saturated	16	22	58	33
Monounsaturated	22	66	33	58
Polyunsaturated	62	12	9	9
<i>Trans</i>	15	38	0	50
Serum cholesterol (mg/100 ml)	-20	0	-14	0

^a National Diet Heart Study, Circulation 37: (Suppl. 1) 303, 1968. ^b F. A. Kummerow, unpublished results.

made from hydrogenated coconut oil, rubbery meat substitutes from soybean protein, and emulsions of egg white and corn oil as substitutes for whole eggs. None of these substitutes has been tested for their biological value, against the food items they are supposed to replace, or for their ability to lower serum cholesterol levels. In fact, an egg substitute is nutritionally inferior to whole eggs (26). A search for angiotoxin factors may bear more fruit than a search for cholesterol-free substitutes.

The literal intoxication with soft drinks may be as detrimental to good nutrition as an excessive amount of fat (101). The 10 billion dollars/year of soft drinks have taken on the role of a nutrient rather than a thirst quencher in the American diet. Furthermore, 35% of the total carbon dioxide capacity in the United States is used by the soft drink industry (102). Some of this carbon dioxide is a by-product of industrial processes such as the manufacture of liquid ammonia. However, much of the carbon dioxide could be saved for essential uses and thus conserve energy. Admittedly much of the water in the United States is not as desirable to drink as a soft drink. On the other hand, the potable water in many sections of the country could be improved with the expenditure of funds for better sources of water.

The recommendation to increase carbohydrate consumption at the expenses of fat does not take into account that the optimum percentage of fat in the diet depends on more than an intake of fat calories. An excess

intake of calories from carbohydrate increases fat synthesis in the body while an excess intake of calories from fat depresses fat synthesis (103). The most massive induction of fatty acid synthesis occurs in animals re-fed glucose or fructose after a preceding fast (104). The control mechanisms in fatty acid synthesis is still under study in both in vivo and in vitro systems. A high carbohydrate diet does not automatically mean that less fat will be deposited in the tissue.

If surfeit calories from carbohydrates are consumed they will be synthesized into palmitic, stearic, and oleic acid. These are the ω -9 series of non-essential rather than the PUFA ω -6 essential series of fatty acids. It would therefore seem judicious to consume a diet of 40% fat rather than 30% fat, if the missing fat calories are replaced by even more calories from carbohydrates. A survey of seven villages in Romania (Table 14) indicated no significant difference in serum cholesterol level of the villagers consuming excess calories from 38% as compared to those consuming 28% of calories from fat.

As vegetable protein sources, such as soybeans, contain as high a percentage of calories from fat as from protein, population groups on largely vegetable protein sources do not consume significant amounts of additional calories from commercially prepared food items that contain either hydrogenated vegetable or animal fats. The replacement of animal protein with dried soybeans in the American diet would only further increase the percentage of fat in the diet which has

TABLE 14
Results of survey in seven villages in Transylvania, Romania in 1962^{a, b}

Village	No. of Persons	Cal/day	Lipid	Serum	Total
			in diet	cholesterol	serum lipid
			%	mg/100 ml	
Marisel	412	2895	20.0	137	721
Avram Iamu	94	2990	23.0	140	804
Finigal	130	3023	25.0	157	
Corna	87	3623	28.0	187	850
Sintana	171	3883	34.0	176	761
Vlaka	130	4137	38.0	188	
Culenesti_Oas	102	3051	20.7	135	709

^a Margarine introduced in 1968; present consumption in urban areas = 2 to 3 kilo/year/capita. These villages are presently being resurveyed. The results to date indicate an increase of 40 mg/100 ml in serum cholesterol values. Courtesy of Dr. Pitea Pavel, Institute of Public Health and Medical Researches, Cluj-Napoca, Romania. ^b Moga, A. Investigations concerning serum cholesterol levels in areas with endemic thyrotoxic dystrophy. *Revue Sci. Med.* 7: 69, 1962.

TABLE 15
Comparison of essential amino acids yield 70 g of protein

Essential amino acid	Yield 70 g of protein
Isoleucine	
Leucine	
Lysine	
Methionine	
Phenylalanine	
Threonine	
Tryptophane	
Valine	
Amount required	
tein ^c	
Cal/70 g protein ^d	
Biological value ^e	
Amount based on	
that must be consumed	
70 g protein	
Calories in amount	
summed to yield	
Approximate cost	
must be consumed	
protein ^f	

^c Rose, W. C., J., and H. J. Souness. Acid Content of Food. Agriculture, 1957. Research Service, H. H. Mitchell. *T. Rev.* 16: 249, 1946 of grocery stores in


already increased year (35) since isolated from soybeans or industrial process more economical acceptable to calories from through pigs, turkeys or chickens. In this may represent a repetitive way of cost per American meat, eggs, and on the other hand, this process of agriculture (10) that is larger than industries compared economical for g of complete milk than 70 beans, nuts, c

TABLE 15
Comparison of essential amino acid content amount that must be consumed to yield 70 g of protein and caloric value of various foods

Essential amino acids	Required ^a	Eggs, whole 2 medium (96 g)	Hamburger ^c (4.0 oz)	Whole milk ^d (1 pint)	Dried beans (4.0 oz raw)	Raw pecans (4.0 oz)	White bread 4 slices (50 g)
		g					
Isoleucine	0.70	0.81	0.96	0.98	1.39	0.63	0.17
Leucine	1.10	0.99	1.50	1.58	2.10	0.88	0.74
Lysine	0.80	0.80	1.60	1.28	1.82	0.50	0.17
Methionine	1.10	0.39	0.45	0.40	0.25	0.17	0.18
Phenylalanine	1.10	0.63	0.75	0.78	1.35	0.64	0.31
Threonine	0.50	0.64	0.81	0.72	1.06	0.44	0.17
Tryptophane	0.25	0.22	0.21	0.22	0.23	0.16	0.08
Valine	0.80	0.96	1.01	1.08	1.48	0.60	0.17
Amount required to yield 70 g protein ^e		12 eggs	11.1 oz	4 pints	11.5 oz	26.1 oz	36 slices
Cal/70 g protein ^f		933	1384	1361	1106	5181	1750
Biological value ^g		96%	76%	90%	38%	60%	67%
Amount based on biological value that must be consumed to yield 70 g protein		12 eggs	14.6 oz	4.6 pints	30.1 oz	44.3 oz	54 slices
Calories in amount that must be consumed to yield 70 g protein ⁱ		933	1829	1514	2852	8637	2625
Approximate cost for amount that must be consumed to yield 70 g protein ^j		\$0.70	\$0.82	\$1.24	\$1.17	\$2.89	\$1.08

^a Rose, W. C., R. L. Wixom, H. B. Lockhardt and G. F. Lambert. *J. Biol. Chem.* 217: 987, 1955. ^b Everson, G. J., and H. J. Sounders. Composition and nutritive importance of eggs. *J. Am. Dietet. Assoc.* 33: 1244, 1957. ^c Amino Acid Content of Foods. Home Economics Research Report no. 4. Washington, D. C.: United States Department of Agriculture, 1957. ^d Composition of Foods. Agricultural Handbook no. 8-1. Washington D.C.: Agricultural Research Service, 1976. ^e The Heinz Handbook of Nutrition. H. J. Heinz Company, 1959. ^f Block, R. J., and H. H. Mitchell. The correlation of the amino acid composition of proteins with their nutritive value. *Nutr. Abstr. Rev.* 16: 249, 1946-1947. ^g Amount necessary, adjusting for biological value. ^h Based on a nonrandom survey of grocery stores in Champaign, Ill. during July, 1977.

already increased by 1 pound/capita every year (35) since 1955. The protein can be isolated from vegetable protein sources such as soybeans or alfalfa through well-developed industrial processes. However, it has been more economical and more organoleptically acceptable to date to remove the excess fat calories from soybeans and pass them through pigs, beef cattle, milk cows, turkeys, or chickens. In a highly industrialized society, this may represent an unsophisticated, primitive way of converting one ton of plant protein per American to animal protein such as meat, eggs, and dairy products. On the other hand, this process involves 94% of American agriculture (105) and an industrial activity that is larger than the automotive and steel industries combined (106). It is simply more economical for an American to consume 70 g of complete protein from eggs, meat, or milk than 70 g of incomplete protein from beans, nuts, or bread, or \$0.70, \$0.82, and

\$1.24, and \$1.17, \$2.89, and \$1.08, respectively (Table 15). However, it is essential to balance these protein sources against the empty calories from fat and sugar so as to minimize their conversion to lipids *in vivo* and yet provide enough calories to maximize protein utilization (107) and to concentrate more research effort on the angiotoxic risk factors in the diet. Although other angiotoxic factors may remain to be considered, vitamin D, oxidized cholesterol, and *trans* fatty acids can all be more easily removed from the diet than meat, milk, or eggs. The role of *trans* fatty acids as a risk factor may be difficult to clarify as it is masked by the level of dietary PUFA (56, 89, 90, 99, 101, 106, 108). 

Addendum

The articles by Glueck and Connor and Reiser in the May issue of this *Journal* (31: 727, 1978 and 31: 865, 1978, respectively) both focus on the possible relationship between the serum cholesterol level and CHD. Glueck

Total serum lipid

7721
804

850
761

709

villages are
rol values.
^b Moga,
Sci. Med.

and Connor believe that the increased CHD in ethnic groups that shift from a low-fat to a high-fat, such as the Japanese to the American diet, is due to increased serum cholesterol levels. This thesis does not take into consideration that the underlying pathology is already established as shown by the death rate from cerebral arterial damage which is 2½ times greater and at an earlier age in Japanese residing in Japan than in Americans. The pathology, as established by hundreds of papers in the Japanese literature, is due to arteriosclerosis and not atherosclerosis.

The low-fat diet of the Japanese and their continual intake of high amounts of vitamin D in the total intake of fish products precludes the development of atherosclerosis. The underlying pathology that results in arterial damage is gradually established during "aging;" its development can be accelerated by angiotoxic factors. Whether vitamin D and oxidized cholesterol are the only angiotoxic factors in the diet remains to be determined. In perspective, the reality of the American diet must be faced. It is the most economical, most nutritious diet ever available to man. The principles upon which it is based as promulgated by the Committees on Nutrition, National Research Council, National Academy of Sciences are used by the pet food industry to keep pets healthy and by the animal industry to produce healthy animals at economical costs. To ignore these principles in human nutrition by making recommendations which would not allow the optimum use of the essential nutrients that are available in the marketplace does not provide for good economics or optimum health.

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References

- Report of Inter-Society Commission for Heart Disease Resources. *Circulation* 42: A-55, 1970.
- Dietary Goals for the U.S. Senate Select Committee on Nutrition and Human Needs. Washington, D.C.: United States Government Printing Office, 1977.
- GOLDSTEIN, J. L., AND M. S. BROWN. The low density lipoprotein pathway and its relation to atherosclerosis. *Ann. Rev. Biochem.* 46: 897, 1977.
- Recommended Dietary Allowance. Food and Nutrition Board, (18th rev. ed.). National Research Council Washington, D.C.: National Academy of Sciences, 1974.
- National Diet Heart Study. *Circulation* 37: (Suppl. 1) 1968.
- Diet and coronary heart disease. *J. Am. Med. Assoc.* 222: 1647, 1972.
- Myocardial Infarction Community registers. Regional Office for Europe. Public Health in Europe 5. Copenhagen: World Health Organization, 1976.
- KOKATNUR, M., N. T. RAND, F. A. KUMMEROW AND H. M. SCOTT. Effect of dietary protein and fat on changes of serum cholesterol in mature birds. *J. Nutr.* 64: 177, 1958.
- American Heart Association. Report, Committee on Nutrition, as Approved by Board of Directors, June 5, 1965, *Food Engineering* 37: 54, 1965.
- WISSLER, R. Development of the atherosclerotic plaque. In: *The Myocardium: Failure and Infarction*, edited by E. Braunwald. New York: H. P. Publishing Co., 1974, p. 155.
- TROWELL, H. Ischemic heart disease and dietary fiber. *Am. J. Clin. Nutr.* 25: 926, 1972.
- KRITCHEVSKY, D., SHIRLEY A. TEPPER AND J. A. STORY. Symposium: Nutritional perspective and atherosclerosis. Nonnutritive fiber and lipid metabolism. *J. Food Sci.* 40: 8, 1975.
- INGLETT, G. E. *Wheat: Production and Utilization*. Westport, Conn.: Avi Publishing Co., Inc.: 1974, p. 193.
- KENT, N. L. *Technology of Cereals with Special Reference to Wheat*, (2nd ed.). Oxford: Pergamon Press, Ltd., 1971.
- POMERANZ, Y. *Wheat: Chemistry and Technology*, (2nd ed.). St. Paul, Minn.: American Association of Cereal Chemists, Inc., 1971, p. 61.
- DIMLER, R. J., R. S. HARRIS AND H. VON LOESECKE. *Wheat: Nutritional Evaluation of Food Processing*. New York: John Wiley and Sons, 1960, p. 198.
- FELLERS, D. A., A. P. MOSSMAN, P. H. JOHNSTON AND E. L. WHEELER. Mechanical debranning of whole-kernel wheat. III. Composition, cooking characteristics and storage stability. *Cereal Chem.* 53: 308, 1976.
- WEATHERLY, A. H., AND B. M. G. COGGER. Fish culture: problems and prospects. *Science* 197: 427, 1977.
- AHRENS, E. H., JR., W. INSULL, JR., R. BLOMSTRAND, J. HIRSCH, T. T. TSALTAS AND M. L. PETERSON. The influence of dietary fats on serum-lipid levels in man. *Lancet* 1: 943, 1957.
- KUMMEROW, F. A., A. UENO, T. NISHIDA AND M. KOKATNUR. Unsaturated fatty acids and plasma lipids. *Am. J. Clin. Nutr.* 8: 62, 1960.
- KOKATNUR, M. G., AND F. A. KUMMEROW. The relationship of corn oil and animal fats to serum cholesterol values at various dietary protein levels. *J. Am. Oil. Chem. Soc.* 36: 248, 1969.
- KOKATNUR, M., N. T. RAND AND F. A. KUMMEROW. Effect of the energy to protein ratio on serum and carcass cholesterol levels in chicks. *Cir. Res.* 6: 424, 1958.
- SOMMER, A., AND M. S. LOEWENSTEIN. Nutritional status and mortality: a prospective validation of the QUAC stick. *Am. J. Clin. Nutr.* 28: 287, 1975.
- GREULICH, W. W. Growth of children of the same race under different environmental conditions. *Science* 127: 515, 1958.
- BEATON, G. H., AND L. D. SWISS. Evaluation of the nutritional quality of food supplies: prediction of "desirable" or "safe" protein: calorie ratios. *Am. J. Clin. Nutr.* 27: 485, 1974.
- NAVIDI, M. K., AND F. A. KUMMEROW. Nutritional value of egg beaters compared with "farm fresh eggs." *Pediatrics* 53: 565, 1974.
- PAG Bulletin, Protein-Calorie Advisory Group of United Nations System 30: 3, 1975.
- SOUTHGATE, D. A. T., AND J. V. G. A. DURNIN. Calorie conversion factors. An experimental re-assessment of the factors used in the calculation of the energy 517, 1970.
- INOUE, B., protein rec and rice p energy intake
- YOUNG, V. RAND AND of man: within the of intakes 534, 1975.
- JACKSON, GOTTO, J. Physiol. R
- JACKSON, J. P. SEGF HOFF AND apolipop- Biol. Chem
- KOKATNUR acid imba Nutr. 75:
- CARROLL, posium: n sis. Effect plasma ch rosis. *J. F United St J. Am. Oil*
- KUMMEROW P. ILINOV influence terol leve 30: 664, 1
- YEH, S.-J ROW. Eff cholesterol Exptl. Bic
- HARROW, chemistry p. 298.
- KUMMEROW and the h
- KRITCHEVSKY and Sons
- MCGILL, mechanis 108, 1974
- GOLDSTEIN E. L. BIE hyperlipi Assoc. Ai
- FREDRICK hyperlysc inherited New Yor
- GOLDSTEIN E. L. BIE idemia in Invest. 52
- FREDRICK phenotyp 1965.
- ESTEP, G

- the energy value of human diets. *Brit. J. Nutr.* 23: 517, 1970.
29. INOUE, B., Y. FUJITA AND Y. NIYAMA. Studies on protein requirements of young men fed egg protein and rice protein with excess and maintenance energy intakes. *J. Nutr.* 103: 1673, 1973.
 30. YOUNG, V. R., L. FAJARDO, E. MURRAY, W. M. RAND AND W. S. SCRIMSHAW. Protein requirements of man: comparative nitrogen balance response within the submaintenance-to maintenance range of intakes of wheat and beef proteins. *J. Nutr.* 105: 534, 1975.
 31. JACKSON, R. L., J. D. MORRISSETT AND A. M. GOTTO, JR. Lipoprotein structure and metabolism. *Physiol. Rev.* 56: 259, 1976.
 32. JACKSON, R. L., J. D. MORRISSETT, J. T. SPARROW, J. P. SEGREST, H. J. POWNALL, L. C. SMITH, H. F. HOFF AND A. M. GOTTO, JR. The interaction of apolipoprotein-serine with phosphatidylcholine. *J. Biol. Chem.* 249: 5314, 1974.
 33. KOKATNUR, M. G., AND F. A. KUMMEROW. Amino acid imbalance and cholesterol levels in chicks. *J. Nutr.* 75: 319, 1961.
 34. CARROLL, K. K., AND R. M. G. HAMILTON. Symposium: nutritional perspectives and atherosclerosis. Effects of dietary protein and carbohydrates on plasma cholesterol levels in relation to atherosclerosis. *J. Food Sci.* 40: 18, 1975.
 35. United States fats, oils consumption at record level. *J. Am. Oil Chem. Soc.* 54: 614a, 1977.
 36. KUMMEROW, F. A., Y. KIM, J. HULL, J. POLLARD, P. ILINOV, D. L. DOROSSIEV AND J. VALEK. The influence of egg consumption on the serum cholesterol level in human subjects. *Am. J. Clin. Nutr.* 30: 664, 1977.
 37. YEH, S.-J. C., T. MIZUGUCHI AND F. A. KUMMEROW. Effect of dietary fat on the release rate of cholesterol from swine erythrocytes. *Proc. Soc. Exptl. Biol. Med.* 156: 236, 1974.
 38. HARROW, B. AND A. MAZUR. *Textbook of Biochemistry.* Philadelphia: W. B. Saunders Co., 1962, p. 298.
 39. KUMMEROW, F. A. Metabolism aspects of nutrition and the heart. *Ill. Med. J.* November, 1964.
 40. KRITCHEVSKY, D. *Cholesterol.* New York: J. Wiley and Sons, 1958, p. 57.
 41. MCGILL, H. C., JR., AND G. E. MOTT. Genetic mechanisms in atherosclerosis. *Advan. Cardiol.* 13: 108, 1974.
 42. GOLDSTEIN, J. L., W. R. HAZZARD, H. G. SCHROTT, E. L. BIERMAN AND A. G. MOTUSKY. Genetics of hyperlipidemia in coronary heart disease. *Trans Assoc. Am. Phys.* 85: 120, 1972.
 43. FREDRICKSON, D. S., AND R. I. LEVY. Familial hyperlipoproteinemias. In: *The metabolic basis of inherited disease*, (3rd ed.) edited by J. B. Stanbury. New York: McGraw-Hill, 1972, p. 575.
 44. GOLDSTEIN, J. L., W. R. HAZZARD, H. G. SCHROTT, E. L. BIERMAN AND A. G. MOTUSKY. Hyperlipidemia in coronary heart disease. I. Lipid levels in 500 survivors of myocardial infarction. *J. Clin. Invest.* 52: 1533, 1973.
 45. FREDRICKSON, D. S., AND R. S. LEES. A system for phenotyping hyperlipidemia. *Circulation* 31: 321, 1965.
 46. ESTEP, G. D., R. C. FANHUY AND F. M. FERGUSON. The effect of age and heredity upon serum cholesterol levels in chickens. *Poultry Sci.* 48: 1980, 1969.
 47. ADEL, H. N., Q. B. DEMING AND L. BRUN. Genetic hypercholesterolemia in rats. *Circulation* 40: (Suppl. 3) 1, 1969.
 48. GREENBURG, L. D., AND H. D. MOON. Blood lipid studies in a rhesus monkey without essential hypercholesterolemia. *Federation Proc.* 20: 256, 1961.
 49. LOFLAND, H. B., JR., F. B. CLARKSON, R. W. ST. CLAIR AND N. D. M. LEHNER. Studies on the regulation of plasma cholesterol levels in squirrel monkeys of two genotypes. *J. Lipid Res.* 13: 39, 1972.
 50. RATCLIFFE, H. L., AND H. LUGINBUHL. The domestic pig: a model for experimental atherosclerosis. *Atherosclerosis* 13: 133, 1971.
 51. CONNOR, W. E., D. B. STONE AND R. E. HODGES. The interrelated effects of dietary cholesterol and fat upon human serum lipid levels. *J. Clin. Invest.* 43: 1691, 1964.
 52. CONNOR, W. E., AND D. S. LIN. The intestinal absorption of dietary cholesterol by hypercholesterolemic (type II) and normocholesterolemic humans. *J. Clin. Invest.* 53: 1062, 1974.
 53. AHRENS, E. H. JR., T. C. CHALMERS AND W. E. CONNOR. Report of the Diet Heart Review Panel. Mass Field Trials of the Diet Heart Question. New York: The American Heart Association, Inc. Monograph no. 28, 1969.
 54. SLATER, G., J. MEAD, G. DHOPESHWARKAR AND R. B. ALFIN-SLATER. Plasma cholesterol and triglycerides in men with added eggs in the diet. *Nutr. Rep. Internat.* 14: 249, 1976.
 55. PORTER, M. W., W. YAMANAKA, S. D. CARLSON AND M. A. FLYNN. Effect of dietary egg on serum cholesterol and triglyceride of human males. *Am. J. Clin. Nutr.* 30: 490, 1977.
 56. JACKSON, R. L., J. D. MORRISSETT, H. J. POWNALL, A. M. GOTTO, JR., A. KAMIO, H. IMAI, R. TRACY AND F. A. KUMMEROW. Influence of dietary trans-fatty acids on swine lipoprotein composition and structure. *J. Lipid Res.* 18: 182, 1977.
 57. GOLDSTEIN, J. L., W. R. HAZZARD, H. G. SCHROTT, E. L. BIERMAN AND A. G. MOTUSKY. Hyperlipidemia in coronary heart disease. I. Lipid levels in 500 survivors of myocardial infarction. *J. Clin. Invest.* 52: 1533, 1973.
 58. GOLDSTEIN, H. L., AND M. S. BROWN. Binding and degradation of low-density lipoproteins by cultured human fibroblasts. *J. Biol. Chem.* 249: 5153, 1974.
 59. ANDERSON, R. G. W., J. L. GOLDSTEIN AND M. S. BROWN. Localization of low density lipoprotein receptors on plasma membrane of normal human fibroblasts and their absence in cells from a familial hypercholesterolemia homozygote. *Proc. Natl. Acad. Sci.* 73: 2434, 1976.
 60. CASTELLI, W. P., J. T. DOYLE, T. GORDON, C. G. HAMES, M. C. HJORTLAND, S. B. HULLEY, A. KAGAN AND W. J. ZUKEL. HDL cholesterol and other lipids in coronary heart disease. The cooperative lipoprotein phenotyping study. *Circulation* 55: 767, 1977.
 61. TAURA, S., M. TAURA, K. TOKUYASU, A. KAMIO AND F. A. KUMMEROW. Ultrastructure of aortic intima obtained as a by-product of coronary bypass surgery. *Artery* 3: 529, 1977.

62. KAMIO, A., F. A. KUMMEROW, S. TAURA, K. TOKUYASU, J. C. CLEVELAND AND S. TAKEBAYASHI. Ultrastructure of human aorta. I. Cellular composition of diffuse intimal thickening. *Med. Bull. Fukuoka Univ.* 4: 15, 1977.
63. IMAI, H., S. K. LEE, S. J. PASTORI AND W. A. THOMAS. Degeneration of arterial smooth muscle cells: Ultrastructural study of smooth muscle cell death in control and cholesterol-fed animals. *Virchows Arch. Pathol. Anat.* 350: 183, 1970.
64. IMAI, H., AND W. A. THOMAS. Cerebral atherosclerosis in swine. Role of necrosis in progression of diet induced lesions from proliferative to atheromatous stage. *Exptl. Molec. Pathol.* 8: 830, 1968.
65. KAMIO, A., B. H. S. CHO, F. A. KUMMEROW AND S. TAKEBAYASHI. Light and electron microscopic studies of diffuse intimal thickening in young and aging swine aortae. *Med. Bull. Fukuoka Univ.* 3: 377, 1976.
66. KAMIO, A., F. A. KUMMEROW, S. TAURA, K. TOKUYASU, J. C. CLEVELAND AND S. TAKEBAYASHI. Ultrastructure of human aorta. II. A mode of lipid formation in atherosclerosis. *Med. Bull. Fukuoka Univ.* 4: 29, 1977.
67. KAMIO, A., W. Y. HUANG, H. IMAI AND F. A. KUMMEROW. Mitotic structures of aortic smooth muscle cells in swine and in culture: Paired cisternae. *J. Electron Microsc.* 26: 29, 1977.
68. KAMIO, A., F. A. KUMMEROW AND H. IMAI. Degeneration of aortic smooth muscle cells in swine fed excess vitamin D₃. *Arch. Pathol. Lab. Med.* 101: 378, 1977.
69. TAURA, S., M. TAURA, K. TOKUYASU, A. KAMIO, F. A. KUMMEROW AND J. C. CLEVELAND. Human arterio- and atherosclerosis: Identical to that in a 6 and 36 month old swine fed a corn soy diet free of cholesterol and saturated fat. *Artery* 4: 100, 1978.
70. TAURA, M., S. TAURA, F. A. KUMMEROW, A. KAMIO AND S. TAKEBAYASHI. Mitotic structure of aortic intimal cells induced by mechanical injury in swine. *Acta Pathol. Jap.* 28: 555, 1978.
71. TAURA, S., M. TAURA, H. IMAI AND F. A. KUMMEROW. Coronary atherosclerosis in normocholesterolemic swine artery. *Arter. Wall* 4: 395, 1978.
72. HUANG, W. Y., A. KAMIO, S.-J. YEH AND F. A. KUMMEROW. The influence of vitamin D on plasma and tissue lipids and atherosclerosis in swine. *Artery* 3: 439, 1977.
73. HUANG, W. Y., AND F. A. KUMMEROW. Cholesterol and fatty acid synthesis in swine. *Lipids* 11: 34, 1976.
74. EISENSTEIN, R., AND W. A. GROFF. Experimental hypervitaminosis D: hypercalcemia, hypermucoproteinemia, and metastatic calcification. *Proc. Soc. Exptl. Biol. Med.* 94: 441, 1957.
75. ROSZKIEWICZ, J., AND S. ZAWISTOWSKI. Histological studies on the kidneys of white rats under conditions of overdosage of vitamins D₂ and D₃. *Folia Morphol.* 27: 301, 1968.
76. KAMIO, A., W. Y. HUANG, B. H. S. CHO, H. IMAI AND F. A. KUMMEROW. Aortic intimal changes in aging swine. *Arter. Wall* 4: 27, 1977.
77. Scientific literature reviews on generally recognized as safe (GRAS) food ingredients—vitamin D. Washington D.C.: Natl. Tech. Information Serv., U.S. Dept. of Commerce, July, 1974.
78. KUMMEROW, F. A., B. H. S. CHO, W. Y.-T. HUANG, H. IMAI, M. H. DEUTSCH AND W. M. HOOPER. Additive risk factors in atherosclerosis. *Am. J. Clin. Nutr.* 29: 579, 1976.
79. IMAI, H., N. T. WERTHESSEN, C. B. TAYLOR AND K. T. LEE. Angiotoxicity and arteriosclerosis due to contaminants of USP-grade cholesterol. *Arch. Pathol. Lab. Med.* 100: 565, 1976.
80. Nutritional analysis of food served at McDonald's restaurants. WARF Institute, Inc. Madison, Wisc.: McDonald's Systems, Inc., 1977.
81. WHEREAT, A. F. Recent advances in experimental and molecular pathology. Atherosclerosis and metabolic disorder in the arterial wall. *Exptl. Molec. Pathol.* 7: 233, 1967.
82. GORDON, G. B., M. A. BARCZA AND M. E. BUSH. Lipid accumulation in hypoxic tissue culture cells. *Am. J. Pathol.* 88: 663, 1977.
83. BRIGGS, R. G., AND J. L. GLENN. Lipid accumulation in cells derived from porcine aorta and grown under anaerobic conditions. *Lipids* 11: 791, 1976.
84. HUANG, W. Y., AND F. A. KUMMEROW. Esterification of *cis* and *trans* fatty acids by swine aortic smooth muscle cells during aerobic and hypoxic incubations. *Proc. Soc. Exptl. Biol. Med.* In press.
85. MCGILL, H. C., JR. The lesion. In: *Atherosclerosis III. Proceedings of the Third International Symposium on Atherosclerosis*, edited by G. G. Schettler and A. Weizel. Berlin: Springer, 1974, pp. 27-38.
86. DAUD, A. S., K. E. FRITZ, J. JARMOLYCH AND J. M. AUGUSTIN. Use of aortic medial explants in the study of atherosclerosis. *Exptl. Molec. Pathol.* 18:177, 1973.
87. MCGILL, H. C., JR., J. C. GEER AND J. P. STRONG. Metabolism of Lipids as Related to Atherosclerosis, edited by F. A. Kummerow. Springfield, Ill.: Charles C Thomas, 1965, p. 36.
88. WATTS, H. F. Pathogenesis of human coronary artery atherosclerosis: Demonstration of serum lipoprotein in the lesions and of localized intimal enzyme defects by histochemistry (P). *Circulation* 24: 1066, 1961.
89. Hsu, C. M. L., AND F. A. KUMMEROW. Influence of elaidate and erucate on heart mitochondria. *Lipids* 12: 486, 1977.
90. LAWSON, L. D., AND F. A. KUMMEROW. Oxidation of 9-trans-octadecanoyl-carnitine in heart mitochondria. *Biochim. Biophys. Acta* In press.
91. KOBAYASHI, D., AND J. R. NEELY. Maximum rates of glycolysis in heart muscle. *Federation Proc.* 36: 545, 1977.
92. COMTE, J., D. GAUTHERON, F. PEYPOUZ AND G. MICHEL. Lipid composition and endogenous respiration of pig heart mitochondria. *Lipids* 6: 882, 1976.
93. HOWARD, H. W., W. J. MONSON, C. D. BAUER AND R. J. BLOCK. The nutritive value of bread flour proteins as affected by practical supplementation with lactalbumin, nonfat dry milk solids, soybean proteins, wheat gluten and lysine. *J. Nutr.* 63: 151, 1957.
94. Safety of used frying fats. *Nutr. Rev.* 26: 210, 1968.
95. THOMPSON, J. A., M. M. PAULOSE, B. R. REDDY, R. G. KRISHNAMURTHY AND S. S. CHANG. A lim-

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96. PERKINS
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- ited survey of fats and oils commercially used for deep fat frying. *Food Technol.* 21: 87A, 1967.
96. PERKINS, E. G., AND L. A. VAN AKKEREN. Heated fats. IV. Chemical changes in fats subjected to deep fat frying processes: cottonseed oil. *J. Am. Oil Chem. Soc.* 42: 782, 1965.
 97. HEMANS, C., F. A. KUMMEROW AND E. G. PERKINS. Influence of protein and vitamin levels on the nutritional value of heated fats for rats. *J. Nutr.* 103: 1655, 1973.
 98. ZALEWSKI, S., AND F. A. KUMMEROW. Rapeseed oil in a two-component margarine base stock. *J. Am. Oil Chem. Soc.* 45: 87, 1968.
 99. KUMMEROW, F. A., T. MIZUGUCHI, T. ARIMAS, B. H. S. CHO AND W. Y. HUANG. The influence of three sources of dietary fats and cholesterol on lipid composition of swine serum lipids and aorta tissue. *Artery* 4: 360, 1978.
 100. World Health Statistics. Vital Statistics and Causes of Death. Geneva: World Health Organization, 1977.
 101. KUMMEROW, F. A. Current studies on relation of fat to health. *J. Am. Oil Chem. Soc.* 51: 255, 1974.
 102. Key chemicals: carbon dioxide. *Chem. Engineer. News* 55: 11, 1977.
 103. NISHIKORI, K., N. IRTANI AND S. NUMA. Levels of acetyl coenzyme A carboxylase and its effectors in rat liver after short-term fat-free refeeding. *FEBS Lett.* 32: 19, 1973.
 104. BRUNENGRABER, H., M. BOUTRY AND J. M. LOWENSTEIN. Fatty acid and 3- β -hydroxysterol synthesis in the perfused rat liver. *J. Biol. Chem.* 248: 2656, 1973.
 105. BRINK, R. A., J. W. DENSMORE AND G. A. HILL. Soil deterioration and the growing world demand for food. *Science* 197: 625, 1977.
 106. KUMMEROW, F. A. Symposium: nutritional perspective and atherosclerosis. *Lipids in atherosclerosis. J. Food Sci.* 40: 12, 1975.
 107. HEGSTED, D. M. Protein-calorie malnutrition. *Am. Sci.* 66: 61, 1978.
 108. SGOUTAS, D., AND F. A. KUMMEROW. Incorporation of *trans*-fatty acids into tissue lipids. *Am. J. Clin. Nutr.* 23: 1111, 1970.

Nutritional implications of dietary fiber¹

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ABSTRACT When dietary fiber intakes are increased by supplementing diets with bran and whole wheat products, then fecal fat, nitrogen, energy, and mineral excretion rise. These changes suggest that fiber may be altering normal digestive and absorptive function. Recent studies have confirmed this and have also shown that fiber of different composition and from contrasting sources produces different physiological effects. The gel-forming polysaccharides such as guar gum and pectin alter the pattern of glucose absorption and are hypocholesterolemic; fiber from cereals is not hypocholesterolemic but exerts a pronounced effect on the large gut. Dietary fiber is largely digested in the colon by the microflora and so influences colonic function, fecal weight, and composition. The significance of the changes in fat, nitrogen, and energy output remains to be evaluated, but the impairment of mineral absorption—particularly of calcium, zinc, and iron—by fiber gives cause for concern. Fiber must now be considered with other dietary constituents in all nutritional studies. *Am. J. Clin. Nutr.* 31: S21-S29, 1978.

In addition to the increasingly well-documented effects fiber has on colonic function, it is now clear that fiber is a nutrient of importance in its own right. The amount and type of fiber included in the diet may have significant nutritional implications. Table 1 lists the areas in which published evidence suggests that fiber alters the digestion absorption or subsequent metabolism of various nutrients. At the present time, one would be unwise to study the nutritional consequences of any group of substances in the diet without including some reference to their interaction with dietary fiber. With the well-publicized suggestion that fiber deficiency is a major public health problem, expansion of research in this field is inevitable. It is therefore important to establish clear priorities. Although it is relatively easy to show that fiber has an effect on a particular aspect of nutrition and nutrient metabolism, it is more difficult and more important to measure the significance of these changes, particularly in the long term.

Traditional concepts of nutrition are of no help in considerations on the role of dietary fiber. Fiber cannot be termed an essential nutrient, and it is not easy to ascertain man's daily requirement for so varied a substance as fiber. Studies with elemental diets demonstrate that man can live satisfactorily for 6 months on a fiber-free diet (1), the only notable change being a decrease in stool bulk and frequency.

Whether a long-term requirement exists for fiber in man remains to be answered.

Fat

Fiber alters lipid absorption. When whole wheat products such as bran are fed, fecal fat excretion rises. Figure 1 shows fecal fatty acid excretion measured by gas liquid chromatography in six medical students during the 3rd week of a metabolic study in which they ate either a controlled diet or a diet to which whole wheat products were added (2). Fiber intake on the controlled diet was 17 g/day and was increased to 45 g/day by exchanging white for wholemeal bread and All Bran for cornflakes, and by adding a bran biscuit and 30 g of bran. A small change in fatty acid intake from 86 to 96 g/day occurred, but fecal fatty acid output increased significantly from 1.7 to 2.7 g/day. Fat excretion measured by the Van de Kamer technique (3) rose from 3 to 4.5 g/day with the additional fiber.

This increase in fecal fat is not one that would be considered significant in terms of overall digestibility and absorption of fat, nor does it represent a pathological rise in fecal fat excretion in gastroenterological terms. It is, however, a well-described phenomenon.

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TABLE 1
Nutritional implications of fiber in
man with selected bibliography

Suggested nutritional implications	Relevant bibliography
Short-term requirements for fiber	(1)
Long-term deficiency states	Widely suggested but as yet unproven
Effect on growth	Unknown for man
Absorption and metabolism	
Carbohydrate	(17-27)
Protein	(2, 4-6, 9, 12)
Lipid	(2, 4-8)
Effect on appetite, satiety, and obesity	See other papers in this symposium
Bile acid and cholesterol metabolism	See other papers in this symposium
Vitamins	(39-44)
Mineral absorption	
Calcium	(45-62)
Iron	(73-77)
Zinc	(47, 63-72)

McCance and Glaser (4) and McCance and Walsham (5) noted fat absorption to be between only 54 and 62% complete from various varieties of whole wheat bread and only slightly greater from oatmeal. These figures are well below normal fat absorption coefficients observed when traditional "Western" style food is eaten.

Does this change represent a true alteration in the digestive and absorptive processes for fat in the gut? Or is a proportion of the fat associated with fiber in such a way as to be unavailable for digestion? Alternatively, does this represent an increase in "endogenous" lipid excretion as suggested by Walker (6)? Detailed analyses of the fecal fatty acids in our studies have suggested that at least part of the fecal fat constitutes fatty acids associated with fiber in such a way that these acids pass through the gut undigested (H. S. Wiggins, personal communication). Kay and Truswell (7) have shown that 15 g/day of pectin can double fecal fat excretion, and Jenkins et al. (8) have shown guar gum also increases fecal fat although both the pectin and guar as fed are lipid free. This suggests that the actual process of lipid absorption can be altered in some way by fiber although the mechanism remains unclear. Few studies have been done that throw much light on this finding, especially ones in which fiber other than that from whole wheat or cereal products is used. More information is needed

particularly about the effect of fiber from commonly eaten fruits and vegetables.

At the moment we can tentatively conclude that the overall balance of fat absorption is not seriously altered by fiber but that small intestinal events leading to lipid absorption are affected. In addition, some fatty acids may be associated with fiber in such a way as to lead to their malabsorption.

Nitrogen

An increase in nitrogen as well as fat excretion occurs when whole wheat products are fed (Fig. 2) (2, 4-6). This increase in fecal nitrogen is readily detectable but not nutritionally significant in Western industrialized communities where high protein intakes are usual. It may of course be more important in societies such as those in rural Africa where dietary fiber intakes are considerably higher.

The source of this fecal nitrogen remains to be established. Two suggestions are that it represents either an increase in endogenous nitrogen excretion (5, 6) or is due to the presence of unavailable protein complexes (9) that pass through the gut undigested. These complexes are formed during cooking or other pretreatment of food before eating and result from the degradation, condensation,

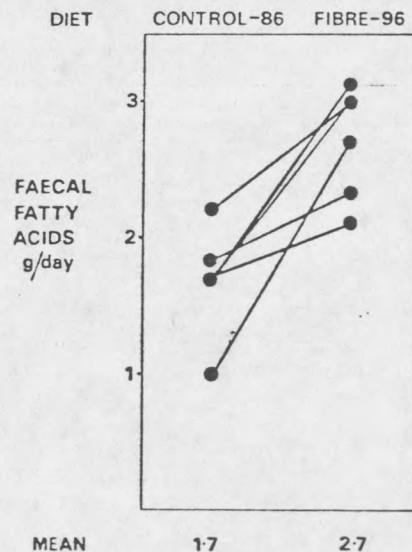


FIG. 1. Fecal fatty acid excretion in g/day (measured by gas liquid chromatography) in six medical students during the 3rd week of metabolic diets of standard "Western" type food or the same diet with whole meal bread, All-Bran, and bran biscuits (2).

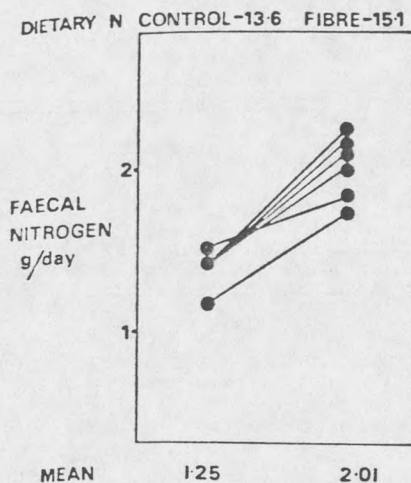


FIG. 2. Fecal nitrogen excretion in g/day from same subjects as in Figure 1.

and polymerization of carbohydrates and amino acids (10). Van Soest (11) has suggested that up to a third of the total protein in some breakfast cereals may be bound up in these complexes. They are known as Maillard complexes and interfere with lignin measurement.

Fiber may also interfere with the normal digestive and absorptive pathways of protein. It has recently been reported that *in vitro* activity of trypsin and chymotrypsin can be inhibited by various plant fibers (12).

A further possible explanation for increased fecal nitrogen excretion is that it represents an increase in fecal microbial flora. Although experiments using careful quantitation of fecal microbial flora with high-fiber diets have so far failed to show significant changes in the flora (13-16), the precision of the techniques used in these studies does not rule out the possibility of increased numbers of microflora in the feces of fiber-supplemented diets.

Carbohydrate

It must have come as a surprise to many people in 1972 to find Trowell suggesting that diabetes was a disorder related to our fiber-deficient diet (17); however, the relatively low fasting blood sugar levels in rural African peoples (18) and the rarity of this condition in communities consuming high-fiber diets have led several people to take this suggestion seriously. It is now clear that dietary fiber can

alter the process of carbohydrate absorption in man. Jenkins et al. (19) have shown that adding 14.5 g of guar gum to a liquid test meal containing glucose or 10 g of pectin to a solid test meal containing bread, butter, marmalade, milk, and tea, or a combination of these substances, leads to a significant flattening of the blood glucose response to these test meals and that this is accompanied by a significant lowering of serum insulin levels measured simultaneously. A similar but less striking change in blood glucose after a test meal containing bran was shown by Jeffreys (20), while bagasse and wood cellulose tended if anything to have the opposite effect. This work has been extended by Jenkins et al. (21) to diabetic subjects. The response in blood glucose to a solid test meal containing both 10 g of pectin and 16 g of guar gum was considerably reduced in both insulin-dependent and noninsulin-dependent diabetics; similar changes in the serum insulin responses were observed in the latter group.

These experimental changes are upheld when diabetics are fed diets containing increased amounts of dietary fiber (22, 23). Although in these dietary studies the intakes of fat, protein, and carbohydrate have all been altered in addition to the change in fiber intake, it is clear that insulin requirements can be reduced and fluctuations in blood glucose minimized by these regimens. The problem of the etiology of diabetes remains, but this work provides a rational basis for an alternative form of treatment.

The way in which these changes in glucose absorption are brought about remains to be established. Jenkins et al. (19) have suggested several ways this could occur. First, glucose absorption rates could be altered through the gel-forming properties of the water-soluble fractions of fiber altering the rate of diffusion of glucose in the upper small intestine. Glucose absorption would, therefore, be slower and would be spread out along a greater length of intestine, thus enabling tissue uptake to keep pace with absorption after initial stimulation of insulin release. These authors (19) demonstrated no increase in the evolution of breath hydrogen derived from the colon so that overall glucose malabsorption does not occur. Second, fiber may effect the release of gastrointestinal hormones and modify pancreatic secretory and digestive

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processes. Alternatively, changes may occur in gastric emptying or in small intestine transit time. Mouth-to-cecum transit time, as judged by the evolution of breath hydrogen, may be delayed by these forms of fiber (24). The likely mechanism probably relates to local interactions of these various foodstuffs in the small intestine rather than to long-term changes in the body's metabolic response to a glucose load although the evidence is somewhat conflicting on this point (25-27). Whatever the mechanism, however, the observation that carbohydrate absorption is altered by fiber remains a valid one.

Fiber digestion

In a consideration of the nutritional implications of dietary fiber, it is important to remember not only that fiber affects the absorption of other nutrients but also that it is itself digested in the human gut. Problems with methodology have considerably hindered research in this field, but digestion of fiber has been shown from several sources including bran (28), mixed diets (29), assorted vegetables (30), cellulose (31), and possibly pectin (32). Southgate (33) pointed out that human capacity to digest fiber varies widely, but in general at least half the fiber is digested in the gut, most probably by the colonic microflora. The breakdown products of fiber include short-chain fatty acids, hydrogen, methane, and carbon dioxide.

The implications of this are clearly important and as yet incompletely understood. One key aspect that has received some attention is the role of fiber in fecal bulking. With evidence now accumulating that short-chain fatty acids are absorbed from the human large bowel (34, 35) as they are from the animal colon, it seems likely that the ability of a particular fiber to resist digestion would be closely related to its fecal bulking capacity. Other important alterations in colonic physiology are also likely to occur.

The factors controlling the breakdown of fiber in the human large bowel are uninvestigated, but, if any parallels can be drawn with the nonruminant animal, the composition of the fiber, its degree of lignification, mean transit time through the gut, and the intestinal microflora are all likely to be important (36-38).

Vitamins

Few investigators have turned their attention to the possible effect of fiber on vitamin metabolism and absorption in the gut. With the known capacity of fiber to adsorb organic compounds and the general association of malnutrition and vitamin deficiency with staple diets containing large amounts of fiber, this seems a reasonable area for research. *In vitro* studies have shown that vegetable material is able to adsorb folic acid and its monoglutamates (39), but no impairment of folic absorption from fiber-rich cereal products occurs when fed in test meals to man (40). Indeed it would appear that high-fiber diets stimulate intestinal microbial synthesis of some B vitamins (41, 42). Rats fed very high supplements of pectin or cellulose have been shown to have impaired vitamin B₁₂ absorption, but it is doubtful if these sort of studies will have many parallels for man (43). Investigations of the interactions of fiber with vitamins is partly complicated by the presence of significant quantities of B vitamins bound within whole wheat products (44).

Mineral absorption

Calcium

It has long been known that subjects fed a high proportion of whole wheat products go into negative calcium balance. Figure 3 shows the calcium balance data for four medical students doing a metabolic study in which we were reexamining this question. Initially a diet containing 88 g of fat, 13 g of protein, 396 g of carbohydrate, 960 mg of calcium, and 22 g of dietary fiber was fed. Fiber intake was then increased by substituting 200 g of wholemeal bread for white bread, 25 g of All Bran for cornflakes, and 50 g of bran biscuits for Nice biscuits, and adding 30 g of Allinsons Bran Plus. These additions increased dietary fiber intake to 53 g/day, and, as a result of calcium having been added to the biscuits, calcium intake increased to 1302 mg/day. Despite the increase in calcium intake, the subjects all went into negative calcium balance during the 3rd week of the diet period.

This observation was previously noted and studied in some depth 30 years ago by McCance, Widdowson, and others (5, 45, 46) and more recently by Rheinhold et al. (47,

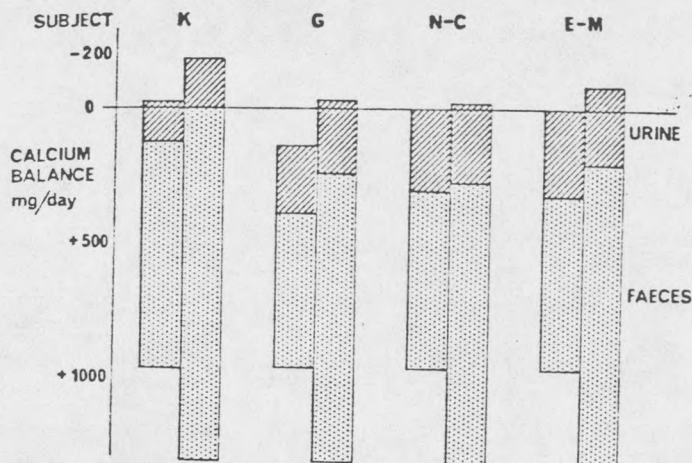


FIG. 3. Calcium balances in four medical students during the 3rd week of metabolic diets. *Left column* for each subject shows fecal and urinary calcium excretion in mg/day during control Western type diet. *Right column* for each subject shows calcium excretion during the same diet in which whole wheat foods had been substituted for equivalent refined foods. Calcium excretion above the balance line represents negative calcium balance.

turned their attention to the effect of fiber on vitamin B₁₂ in the gut. With the aim to adsorb organic acids, the general association of calcium deficiency with substantial amounts of fiber, the need for research. In that vegetable materials, folic acid and its absence, no impairment of calcium-rich cereal products, it is clear that high-fiber diets, microbial synthesis (42). Rats fed very high levels of cellulose have no effect on vitamin B₁₂ utilization if these sort of diets. The effects of fiber on calcium balance in man (43). The effects of fiber with calcium, mediated by the presence of B vitamins products (44).

That subjects fed a high-fiber diet of whole wheat products gave a negative calcium balance. Figure 3 shows the results for four medical students in which whole wheat products were substituted for equivalent refined foods. Initially a diet of 13 g of protein, 100 mg of calcium, and 100 mg of fiber was fed. Fiber intake was substituted 200 g of whole wheat bread, 25 g of Allinson's bran biscuits, and 30 g of Allinson's bran biscuits. The increased dietary fiber, as a result of the substitution of the biscuits, increased calcium intake to 1302 mg/day. The negative calcium balance of the diet period was previously noted and reported 10 years ago by Walker et al. (5, 45, 46) and by Hold et al. (47).

45) Their work perhaps more than any other evidence has cast doubt on the value of wholemeal bread in our diet and has been cited as one of the few possible hazards to increasing dietary fiber intake from whole grain sources. It has even been suggested that whole wheat products may be responsible for the development of osteomalacia (49), and recently these foods have been implicated in the development of rickets in chuppati-eating immigrants in Britain (50, 51).

The association of calcium malabsorption with wholegrain products is traditionally ascribed to the phytic acid content of these foods (52, 53). While it is undoubtedly true that phytic acid impairs calcium absorption (46, 47), it now seems distinctly possible that fiber itself may also have a role. Some of the questions we need to answer in relation to fiber and mineral balance are these:

1) Is overall balance affected by fiber, or is the change one in route of excretion?

2) Can changes be induced by commonly eaten fiber sources and if so at what levels of intake?

3) Which component of fiber is responsible for the changes in absorption (e.g., cellulose, lignin, noncellulosic polysaccharides, or other cell wall constituents), and what are the mechanisms of this change?

4) Do long-term adaptive effects occur with high-fiber diets?

5) What interactions occur between fiber and other minerals and dietary constituents?

6) Is the change in mineral balance significant in biological terms?

It is possible that fiber itself, independent of phytate, interferes with mineral absorption and metabolism. Review of the botanical literature quickly leads one to the belief that not only do cell wall substances themselves bind calcium and other cations but also that these cations play an essential role in the development of the plant cell wall and in plant growth. Detailed calcium-binding studies have been reported with polysaccharides from cell walls (54), and in vitro work suggests that fiber may indeed bind calcium (55), possibly in relation to the uronic acid component of the cell wall structures (56). Calcium binding directly to fiber may be part of the explanation for negative mineral balances in subjects fed high-fiber diets (48). At the present time little is known about the effect of feeding fiber from sources other than cereals on mineral balance in man; work in this area is clearly needed.

Equally important is the suggestion that these changes in calcium balance are in fact only short term and that over a course of weeks and months man is able to adapt to the alterations in intestinal physiology and return to normal calcium balance. Walker et al. (57) were among the first to suggest that, if subjects were studied for long periods (over 4 weeks), calcium balance would return to normal. A number of factors in Walker's study makes interpretation difficult, particularly

the large fall in calcium intake when subjects were transferred from their normal diet to the study diets (i.e., a change in calcium intake from 1000 to 500 mg/day). Interestingly, the subject who normally took the lowest calcium intake showed no change in calcium balance with whole meal bread. Campbell et al. (58) were unable to show adaptation in calcium balance to whole wheat diets in two subjects whom they studied over a period of 5 to 6 weeks. Their two subjects remained in negative calcium balance throughout the experiment, and both showed a fall in serum calcium levels.

Serum calcium levels have not fallen in other studies in which the effect of supplementing a Western-type diet with bran or whole wheat products has been observed over periods of 3 to 19 weeks (59-61). A significant exception to this pattern, however, was seen in a study of 27 elderly patients whose diets were supplemented with 10 or 20 g of bran for 6 weeks (62). These findings clearly need further study.

Studies in which magnesium absorption have been measured at the same time as calcium absorption tend to show the two cations behaving similarly when whole wheat diets are fed.

Zinc

The possibility that man might become zinc deficient has been taken seriously only in recent years; that fiber may play a part in the development of this deficiency is a new concept. Initial impetus to this work came originally from Prasad et al. (63-66) who described a zinc-deficiency syndrome in Iranian children characterized by iron deficiency anaemia, hepatosplenomegaly, hypogonadism, dwarfism, and geophagia. This was later shown to be associated with impaired zinc absorption, and it is now clear that growth can be improved in young people by adding zinc to their diet (67). Equally startling has been the demonstration by Hambidge and Walravens (68) that zinc deficiency occurs even among the children of middle- and upper-income families in the United States. The role of diet in promoting or impairing zinc absorption therefore becomes crucial.

Rhinehold et al. (47, 48) showed that diets high in whole wheat products lead to negative zinc balance and initially suggested that this

was due to the phytic acid content of these foods analogous to the calcium malabsorption-phytic acid story. More recently he has shown that zinc binds quite strongly, perhaps more strongly than calcium, to dephytinized fiber in vitro (55, 69). In fact the removal of phytate from bran or whole meal bread or the addition of calcium to the preparation seems to increase zinc binding in vitro. Zinc has also been shown to bind in vitro to starch and pure cellulose.

In contrast to their findings for calcium absorption, Campbell et al. (58) suggest that adaptation to impaired zinc absorption does in fact occur after a period of weeks. However, few long-term studies have been done. Preliminary evidence on the properties of other fiber preparations has yielded conflicting results (70, 71).

Further interest in this aspect of mineral absorption has been stimulated by the suggestion that the ratio of zinc to copper absorbed could be important in the development of atherosclerosis through an effect on cholesterol metabolism (72). Many questions relating to zinc absorption and fiber remain unanswered.

Iron

Iron absorption is known to vary considerably depending on the type and composition of the diet being eaten. Its availability from whole wheat products is particularly poor (73). Balance studies show that iron absorption is impaired from diets containing wholemeal bread (74) and from test meals containing rice and vegetables (75). Other investigators have shown decreased serum iron levels after 3 or 5 weeks of diets containing bran and whole wheat products (62, 76). Once again phytic acid has been incriminated as the main factor diminishing iron absorption in these circumstances and in particular the form in which the iron is bound to phytate. In the rat the monoferric phytate present in bran was readily available for absorption when isolated from the bran while other bran-iron-phytate complexes were not (77). It is also possible that iron binds directly to plant cell wall material other than phytic acid (55), so we need to study iron absorption specifically from the point of view of dietary fiber.

The interaction in the gut of cell wall materials with mineral absorption represents a

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Possible disadvantage of high-fiber diets and perhaps particularly so when Western-style diets are supplemented with high-fiber preparations. One area where caution would seem to be needed is in supplementing the diet of old people with whole wheat products to improve bowel habits when their dietary intakes of iron and calcium may be barely sufficient for their normal requirements. It is difficult to disentangle the relative roles of phytic acid and dietary fiber in altering mineral absorption, but current evidence suggests that fiber may have an independent role. Further information is needed particularly on the mineral-binding properties of dietary fiber as present in commonly eaten fruits and vegetables. Equally important to bear in mind will be the possibility that in the long term the human system is able in some way to adapt to the diminished availability of minerals as a result of these dietary changes and that we may ultimately have seemed overcautious about this aspect of dietary fiber metabolism.

Conclusions

Sufficient preliminary evidence is available to suggest that fiber has important nutritional implications in addition to its already well-known gastroenterological effects. Fiber appears to affect the rate and route of absorption and metabolism of dietary fat, carbohydrate, and protein as well as altering sterol metabolism and mineral balance.

The important question of the significance of these changes remains to be answered. We need further information before the role of dietary fiber can be assessed. For example we need to know more about the amount of fiber eaten by various populations, its type, and its physiological effects. Gathering this information is unlikely to prove easy as the essence of the fiber story is one of long-term changes in diet leading to altered patterns in human disease. Furthermore we need to recognize that the nutritional significance of a fiber-rich or fiber-depleted diet will depend on other constituents of the diet, which vary greatly in different parts of the world. Fiber cannot be seen in isolation from the rest of the diet.

It is wisest to assume at the moment that the absorption and metabolism of many if not all nutrients are affected by fiber. Some

of these changes appear to be nutritionally beneficial although the role of fiber in mineral absorption gives grounds for concern. If the effect of fiber on colonic function proves to be important in relieving constipation and preventing diverticular disease and large bowel cancer, the need to increase fiber intakes will be clear. This alone means that the nutritional implications need to be evaluated. However, it is now possible to conceive that some of these nutritional changes may be of equal importance in their own right. □

The author is indebted to Dr. Philip James for helpful discussions during the preparation of this paper and to Will Branch, Helen Houston, Dr. David Jenkins, and Dr. Hugh Wiggins for collaboration in studies on dietary fiber.

References

1. WINITZ, M., D. A. SEEDMAN AND J. GRAFF. Studies in metabolic nutrition employing chemically defined diets. I. Extended feeding of normal human adult males. *Am. J. Clin. Nutr.* 23: 525, 1970.
2. CUMMINGS, J. H., M. J. HILL, D. J. A. JENKINS, J. R. PEARSON AND H. S. WIGGINS. Changes in fecal composition and colonic function due to cereal fiber. *Am. J. Clin. Nutr.* 29: 1468, 1976.
3. JOVER, A., AND R. S. GORDON. Procedure for quantitative analysis of feces with special reference to fecal fatty acids. *J. Lab. Clin. Med.* 59: 878, 1962.
4. MCCANCE, R. A., AND E. M. GLASER. The energy value of oatmeal and the digestibility and absorption of its proteins, fats and calcium. *Brit. J. Nutr.* 2: 221, 1948.
5. MCCANCE, R. A., AND C. M. WALSHAM. The digestibility and absorption of the calories, protein, purines, fat and calcium in wholemeal wheaten bread. *Brit. J. Nutr.* 2: 26, 1948.
6. WALKER, A. R. P. Effect of high crude fibre intake on transit time and the absorption of nutrients in South African Negro school children. *Am. J. Clin. Nutr.* 28: 1161, 1975.
7. KAY, R. M., AND S. TRUSWELL. Effect of citrus pectin on blood lipids and fecal steroid secretion in man. *Am. J. Clin. Nutr.* 30: 171, 1977.
8. JENKINS, D. J. A., A. R. LEEDS, M. A. GASSULL, H. HOUSTON, D. V. GOFF AND M. J. HILL. The cholesterol lowering properties of guar and pectin. *Clin. Sci. Mole. Med.* 51: 8, 1976.
9. GOERING, H. K., C. H. GORDON, R. W. HEMKEN, D. R. WALDO, P. J. VAN SOEST AND L. W. SMITH. Analytical estimates of nitrogen digestibility in heat damaged forages. *J. Dairy Sci.* 55: 1275, 1972.
10. VAN SOEST, P. J., AND R. W. MCQUEEN. The chemistry and estimation of fibre. *Proc. Nutr. Soc.* 32: 123, 1973.
11. VAN SOEST, P. J. Food and fibre. *Näringsforskning* 20: (Suppl. 14) 61, 1976.
12. SCHNEEMAN, B. O. The effect of plant fibre on trypsin and chymotrypsin activity in vitro. *Federation Proc.* 36: 1118, 1977.
13. DRASAR, B. S., D. J. A. JENKINS AND J. H. CUM-

- MINGS. The influence of a diet rich in wheat fibre on the human faecal flora. *J. Med. Microbiol.* 9: 423, 1976.
14. BAIRD, I. M., R. L. WALTERS, P. S. DAVIES, M. J. HILL, B. S. DRASAR AND D. A. T. SOUTHGATE. The effects of two dietary fibre supplements on gastrointestinal transit, stool weight and frequency, and bacterial flora, and faecal bile acids in normal subjects. *Metabolism* 26: 117, 1977.
 15. FUCHS, H.-M., S. DORFMAN AND M. H. FLOCH. The effect of dietary fiber supplementations in man. II. Alteration in fecal physiology and bacterial flora. *Am. J. Clin. Nutr.* 29: 1443, 1976.
 16. DRASAR, B. S., AND D. J. A. JENKINS. Bacteria, diet and large bowel cancer. *Am. J. Clin. Nutr.* 29: 1410, 1976.
 17. TROWELL, H. Dietary fibre, ischaemic heart disease and diabetes mellitus. *Proc. Nutr. Soc.* 32: 151, 1973.
 18. WALKER, A. R. P., B. F. WALKER AND B. D. RICHARDSON. Glucose and fat tolerances in Bantu children. *Lancet* 2: 51, 1970.
 19. JENKINS, D. J. A., A. R. L. LEEDS, M. A. GASSULL, B. COCHET AND K. G. M. M. ALBERTI. Decrease in postprandial insulin and glucose concentrations by guar and pectin. *Ann. Internal Med.* 86: 20, 1977.
 20. JEFFREYS, D. B. The effect of dietary fibre on the response to orally administered glucose. *Proc. Nutr. Soc.* 33: 11A, 1974.
 21. JENKINS, D. J. A., A. R. LEEDS, M. A. GASSULL, T. M. S. WOLEVER, D. V. GOFF, K. G. M. M. ALBERTI AND T. D. R. HOCKADAY. Unabsorbable carbohydrates and diabetes: decreased post-prandial hyperglycaemia. *Lancet* 2: 172, 1976.
 22. KIEHM, T. G., J. W. ANDERSON AND K. WARD. Beneficial effects of a high carbohydrate, high fiber diet on hyperglycemic diabetic men. *Am. J. Clin. Nutr.* 29: 895, 1976.
 23. DOUGLASS, J. M. Raw diet and insulin requirements. *Ann. Internal Med.* 82: 62, 1975.
 24. LEEDS, A. R., M. A. GASSULL, G. L. METZ AND D. J. A. JENKINS. Food: influence of form on absorption. *Lancet* 1: 1213, 1975.
 25. JENKINS, D. J. A., A. R. LEEDS, H. HOUSTON, L. HINKS, K. G. M. M. ALBERTI AND J. H. CUMMINGS. Carbohydrate tolerance on man after six weeks of pectin administration. *Proc. Nutr. Soc.* 36: 624, 1977.
 26. VAN DER WESTHUIZEN, J., M. MBIZRI AND J. J. JONES. Unrefined carbohydrate and glucose tolerance. *Lancet* 2: 719, 1972.
 27. DRODRIBB, A. J. M., AND D. M. HUMPHREYS. Diverticular disease: three studies. Part III. Metabolic effects of bran in patients with diverticular disease. *Brit. Med. J.* 1: 428, 1976.
 28. SOUTHGATE, D. A. T., W. J. BRANCH, M. J. HILL, B. S. DRASAR, R. L. WALTERS, P. S. DAVIES AND I. M. BAIRD. Metabolic responses to dietary supplements of bran. *Metabolism* 25: 1129, 1976.
 29. SOUTHGATE, D. A. T., AND I. DURNIN. Calorie conversion factors. An experimental reassessment of the factors used in the calculation of the energy value of human diets. *Brit. J. Nutr.* 24: 517, 1970.
 30. WILLIAMS, R. D., AND W. H. OLMSTED. The effect of cellulose, hemicellulose and lignin on the weight of stool. A contribution to the study of laxation in man. *J. Nutr.* 11: 433, 1936.
 31. MILTON-THOMPSON, G. J., AND B. LEWIS. The breakdown of dietary cellulose in man. *Gut* 12: 853, 1971.
 32. WERCH, S. C., R. W. JUNG, H. PLENK, A. A. DAY AND A. C. IVY. Pectin and galacturonic acid and the intestinal pathogens. *Am. J. Diseases Children* 63: 839, 1942.
 33. SOUTHGATE, D. A. T. Fibre and the other unavailable carbohydrates and their effects on the energy value of the diet. *Proc. Nutr. Soc.* 32: 131, 1973.
 34. DAWSON, A. M., C. D. HOLDSWORTH AND J. WEBB. Absorption of short chain fatty acids in man. *Proc. Soc. Exptl. Biol. Med.* 117: 97, 1964.
 35. MCNEILL, N. I., J. H. CUMMINGS AND W. P. T. JAMES. Short chain fatty acid absorption in the human large bowel. *Gut* 18: A425, 1977.
 36. VAN SOEST, P. J. The uniformity and nutritive availability of cellulose. *Federation Proc.* 32: 1804, 1973.
 37. KEYS, J. E., P. J. VAN SOEST AND E. P. YOUNG. Effect of increasing dietary cell wall content on the digestibility of hemicellulose, and cellulose in swine and rats. *J. Animal Sci.* 31: 1172, 1970.
 38. KEYS, J. E., P. J. VAN SOEST AND E. P. YOUNG. Comparative study of the digestibility of forage, cellulose and hemicellulose in ruminant and non ruminants. *J. Animal Sci.* 29: 11, 1969.
 39. LUTHER, L., R. SANTINI, C. BREWSTER, E. PEREZ-SANTIAGO AND C. E. BUTTERWORTH. Folate binding by insoluble components of American and Puerto Rican diets. *Ala. J. Med. Sci.* 3: 389, 1965.
 40. RUSSELL, R. M., F. ISMAIL-BEIGI AND J. G. RHEINHOLD. Folate contents of Iranian breads and the effect of their fiber content on the intestinal absorption of folic acid. *Am. J. Clin. Nutr.* 29: 799, 1976.
 41. NAGASE, H., AND A. FUGITA. The synthesis of vitamins by intestinal bacteria in man and the effect of cellulose. II. Synthesis of thiamine. *J. Vitaminol. Japan* 2: 107, 1956.
 42. NAGASE, H., AND A. FUGITA. The synthesis of vitamins by intestinal bacteria in man and the effect of cellulose. I. Synthesis of riboflavin. *J. Vitaminol. Japan* 2: 102, 1956.
 43. CULLEN, R. W., AND S. M. OACE. Cellulose and pectin enhance vitamin B₁₂ depletion in rats. *Federation Proc.* 36: 1118, 1971.
 44. MASON, J. B., N. GIBSON AND E. KODICEK. The chemical nature of the bound nicotinic acid of wheat bran: studies of nicotinic acid-containing macromolecules. *Brit. J. Nutr.* 30: 297, 1973.
 45. McCANCE, R. A., AND E. M. WIDDOWSON. Mineral metabolism of healthy adults on white and brown bread dietaries. *J. Physiol.* 101: 44, 1942.
 46. McCANCE, R. A., AND E. M. WIDDOWSON. Mineral metabolism on dephytinized bread. *J. Physiol.* 101: 304, 1942.
 47. RHEINHOLD, J. G., K. NASR, A. LAHIMGARZADEH AND H. HEDAYATI. Effects of purified phytate and phytate-rich bread upon metabolism of zinc, calcium, phosphorus and nitrogen in man. *Lancet* 1: 283, 1973.
 48. RHEINHOLD, J. G., B. FARADJI, P. ABADI AND F. ISMAIL-BEIGI. Decreased absorption of calcium, magnesium, zinc and phosphorus by humans due to increased fiber and phosphorus consumption of wheat bread. *J. Nutr.* 106: 493, 1976.

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- J. AND B. LEWIS. The effect of cellulose in man. *Gut*. 12: 117, 1971.
- G. H. PLENK, A. A. D. AND J. W. WIDDOWSON. The effect of galacturonic acid and other carbohydrates on the metabolism of zinc in man. *J. Diseases Children* 112: 100, 1969.
- AND THE OTHER UNAVAILABLE. The effects of cellulose on the energy metabolism. *Ann. N.Y. Acad. Sci.* 32: 131, 1973.
- OLDSWORTH AND J. W. WIDDOWSON. The effect of fatty acids in man. *Proc. Nutr. Soc.* 97: 1964.
- CUMMINGS AND W. P. T. JAMES. The effect of acid absorption in the human. *Nutr. Rep. Int.* 425, 1977.
- AND NUTRITIVE AVAILABILITY. The effect of cellulose on the absorption of calcium. *Nutr. Rep. Int.* 1804, 1977.
- ROEST AND E. P. YOUNG. The effect of cellulose on the cell wall content of the human. *Nutr. Rep. Int.* 1172, 1970.
- ROEST AND E. P. YOUNG. The effect of cellulose on the digestibility of forage in ruminant and non-ruminant. *Nutr. Rep. Int.* 29: 11, 1969.
- C. BREWSTER, E. PEREZ, J. W. WIDDOWSON AND P. A. M. OACE. Folate binding in man. *Nutr. Rep. Int.* 3: 389, 1965.
- ISMAIL-BEIGI AND J. G. RHEINHOLD. The effect of Iranian breads and their bran on the intestinal absorption of calcium. *Clin. Nutr.* 29: 799, 1976.
- ISMAIL-BEIGI AND J. G. RHEINHOLD. The synthesis of vitamin B12 in man and the effect of thiamine. *J. Vitaminol.* 1976.
- ISMAIL-BEIGI AND J. G. RHEINHOLD. The synthesis of vitamin B12 in man and the effect of riboflavin. *J. Vitaminol.* 1976.
- M. OACE. Cellulose and its effect on the depletion in rats. *Federation Proc.* 34: 120A, 1975.
- AND E. KODICEK. The effect of cellulose and nicotinic acid of wheat bran on the metabolism of calcium-containing macromolecules. *Nutr. Rep. Int.* 7: 1973.
- M. WIDDOWSON. Mineral absorption in white and brown bread. *J. Physiol.* 101: 44, 1942.
- M. WIDDOWSON. Mineral absorption in white and brown bread. *J. Physiol.* 101: 44, 1942.
- SR. A. LAHIMGARZADEH AND M. NADIMI. The effect of purified phytate and cellulose on the metabolism of zinc, calcium and iron in man. *Lancet* 1: 493, 1976.
- ABADI AND F. ISMAIL-BEIGI. The effect of cellulose on the absorption of calcium, magnesium and phosphorus by humans due to phytate consumption. *Nutr. Rep. Int.* 493, 1976.
- BIRKBYNE, G. M., J. B. ARI, E. NORD AND R. SHANKIN. Bedouin osteomalacia due to calcium deprivation caused by high phytic acid content of unleavened bread. *Am. J. Clin. Nutr.* 26: 910, 1973.
- RHEINHOLD, J. G. Rickets in Asian immigrants. *Lancet* 2: 1132, 1976.
- FORD, J. A., E. M. COLHOUN, W. B. MCINTOSH AND M. G. DUNNIGAN. Biochemical response of late rickets and osteomalacia to a chupatty-free diet. *Brit. Med. J.* 3: 446, 1972.
- WIDDOWSON, E. M. Interrelations of dietary calcium with phytates, phosphates and fats. *Nutr. Dieta* 15: 1970.
- RHEINHOLD, J. G., H. HEDAYATI, A. LAHIMGARZADEH AND K. NASR. Zinc, calcium phosphorus and nitrogen balances of Iranian villagers following change from phytate-rich to phytate-poor diets. *Ecol. Food Nutr.* 2: 157, 1973.
- DELUCA, L. AND C. E. BUGG. Calcium binding to D-glucuronate residues: crystal structure of a hydrated calcium bromide salt of D-glucuronic acid. *Carbohydrate Res.* 41: 19, 1975.
- RHEINHOLD, J. G., F. ISMAIL-BEIGI AND B. FARADJI. Fibre vs. phytate as determinant of the availability of calcium, zinc and iron of breadstuffs. *Nutr. Rept. Internat.* 12: 75, 1975.
- FRANCH, W. J., D. A. T. SOUTHGATE AND W. P. T. JAMES. Binding of calcium by dietary fibre: its relationship to unsubstituted uronic acids. *Proc. Nutr. Soc.* 34: 120A, 1975.
- WALKER, A. R. P., F. W. FOX AND J. T. IRVING. Studies in human mineral metabolism. 1. The effect of bread rich in phytate phosphorus on the metabolism of certain mineral salts with special reference to calcium. *Biochem. J.* 42: 452, 1948.
- CAMPBELL, G. J., J. G. RHEINHOLD, J. S. CANNELL AND I. NOURMAND. The effects of prolonged consumption of wholemeal bread upon metabolism of calcium, magnesium, zinc and phosphorus of two young American adults. *Pahlavi Med. J.* 7: 1, 1976.
- HEATON, K. W., A. P. MANNING AND M. HARTOG. Lack of effect on blood lipid and calcium concentrations of young men on changing from white to wholemeal bread. *Brit. J. Nutr.* 35: 55, 1976.
- CANNELL, A. M., C. L. SMITH AND M. SOMSEL. Absence of effect of bran on blood-lipids. *Lancet* 1: 493, 1975.
- KAY, R. M., AND A. S. TRUSWELL. The effect of wheat fibre on plasma lipids and faecal steroid excretion in man. *Brit. J. Nutr.* 37: 227, 1977.
- FRANSSON, L., K. RABY, P. FONT-BECH AND E. JENSEN. Effect of prolonged bean administration on serum levels of cholesterol, ionised calcium and iron in the elderly. *J. Am. Gerontol. Soc.* 24: 334, 1976.
- PRASAD, A. S., J. A. HALSTEAD AND M. NADIMI. Syndrome of iron deficiency anemia, hepatosplenomegaly, dwarfism, hypogonadism and geophagia. *Am. J. Med.* 31: 532, 1961.
- PRASAD, A. S., A. MIALE, Z. FARID, H. H. SANDSTEAD, A. R. SCHULERT AND W. J. DARBY. Biochemical studies on iron deficiency anemia, dwarfism and hypogonadism. *Arch. Internal Med.* 141: 407, 1963.
- PRASAD, A. S., A. MIALE, Z. FARID, A. SCHULERT AND H. H. SANDSTEAD. Zinc metabolism in normals and patients with the syndrome of iron deficiency anemia, hypogonadism and dwarfism. *J. Lab. Clin. Med.* 61: 537, 1963.
- PRASAD, A. S., A. R. SCHULERT, A. MIALE, Z. FARID AND H. H. SANDSTEAD. Zinc and iron deficiencies in male subjects with dwarfism but without acylstomatias schistosomiasis or severe anemia. *Am. J. Clin. Nutr.* 12: 437, 1963.
- RONAGHY, H. A., J. G. REINHOLD, M. MAHLOUJII, P. GHAVAMI, M. R. S. FOX AND J. A. HALSTEAD. Zinc supplementation of malnourished schoolboys in Iran: Increased growth and other effects. *Am. J. Clin. Nutr.* 27: 112, 1974.
- HAMBIDGE, K. M., AND P. A. WALRAVENS. Zinc deficiency in infants and pre-adolescent children. In: *Trace Elements in Human Health and Disease. Zinc and Copper*, edited by A. S. Prasad and D. Oberleas. New York: Academic Press, 1976, vol. 1, pp. 21-32.
- RHEINHOLD, J. G., B. FARADJI, P. ABADI AND F. ISMAIL-BEIGI. Binding of zinc to fiber and other solids of wholewheat bread. In: *Trace Elements in Human Health and Disease. Zinc and Copper*, edited by A. S. Prasad and D. Oberleas. New York: Academic Press, 1976, vol. 1, pp. 163-180.
- SANDSTEAD, H. H., L. KLEVAY, J. MUNOZ, R. JACOB, S. RECK, D. TUCKER, G. LOGAN, L. EELKEMA, G. INGLET, F. DINTZIS AND W. SHUEY. Zinc and copper balances in humans fed fiber. *Federation Proc.* 36: 1118, 1977.
- BESHGETOOR, D., C. KIES AND H. M. FOX. Zinc utilisation by human adults as affected by dietary pectin, cellulose and hemicellulose. *Federation Proc.* 36: 1118, 1977.
- KLEVAY, L. M. Coronary heart disease: the zinc/copper hypothesis. *Am. J. Clin. Nutr.* 28: 764, 1975.
- BJORN-RASMUSSEN, E. Iron absorption from wheat bread. Influence of various amounts of bran. *Nutr. Metab.* 16: 101, 1974.
- WIDDOWSON, E. M., AND R. A. MCCANCE. Iron exchanges of adults on white and brown bread diets. *Lancet* 1: 588, 1942.
- HALLBERG, L., L. GARBY, R. SUWANIK AND E. BJORN-RASMUSSEN. Iron absorption from South-East Asian diets. *Am. J. Clin. Nutr.* 27: 826, 1974.
- JENKINS, D. J. A., M. J. HILL AND J. H. CUMMINGS. Effect of wheat fibre on blood lipids, fecal steroid excretion and serum iron. *Am. J. Clin. Nutr.* 28: 1408, 1975.
- MORRIS, E. R., AND R. ELLIS. Isolation of monoferric phytate from wheat bran and its biological value as an iron source to the rat. *J. Nutr.* 106: 753, 1976.

A G E N D A

Clinical Applications and Prevention Advisory Committee
January 11, 1979
Federal Building, Room 6C01
Bethesda, Maryland

New Initiatives of Research on Sodium and Hypertension

January 11, 1979

Chairman, Dr. Robert Shank

1:15 p.m.	Call to order and opening remarks	Dr. Zukel
1:20 p.m.	Plan of Agenda	Dr. Shank
1:30 p.m.	Remarks on DiVD interests	Dr. Jesse
1:45 p.m.	Summary of deliberations of Salt and Water Subgroup of the Hypertension Task Force	Dr. Coleman
2:15 p.m.	Research on Sodium and Hypertension in the Intramural Research Program	Dr. Keiser
2:30 p.m.	Testing the research question of the role of Sodium as a contributing factor in hypertension	Dr. McMillan
2:45 p.m.	Discussion of methodology needs	
3:15 p.m.	Discussion of testing the efficacy of moderate sodium reduction in the therapy of mild/moderate hypertension.	
4:00 p.m.	Identification of other research areas needing investigation in relation to Sodium and hypertension.	
4:30 p.m.	Adjournment	

Additional Invited Participants

Thomas G. Coleman, Ph.D.
Professor, Dept. of Physiology
and Biophysics
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University of Mississippi Medical Center
Jackson, Mississippi

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Chairman, Dept. of Post-Graduate
Medicine and Health Profession
Education
Univ. of Michigan Medical Center
Ann Arbor, Michigan

Harry Keiser, M.D.
Deputy Chief
Hypertension-Endocrine Branch
Intramural Research Programs
National Heart, Lung, and Blood Institute

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Epidemiology Branch, DHVD

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Hypertension and Detection Follow-Up Program, DHVD
Preventive Cardiology Branch, DHVD

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Cynthia Ford
Nutritionist
Preventive Cardiology Branch, DHVD

Jeanne Tillotson
Nutritionist
Preventive Cardiology Branch, DHVD

William J. Zukel, M.D.
Associate Director for Clinical Applications
and Prevention, DHVD

COMMISSION
OF
NUTRITION COMMITTEE

- NUMBER OF MEMBERS: Eight
- METHOD OF APPOINTMENT: The Steering Committee Chairman in consultation with Nutrition Committee Chairman
- TERM OF OFFICE: Three Years
- USUAL FREQUENCY OF MEETINGS: Minimum of Two Per Year
- RESPONSIBILITIES:
1. To be fully informed concerning new scientific developments and the advances in knowledge concerning nutrition and/or diet as it relates to health and the occurrence or treatment of cardiovascular disease; synthesize the pertinent knowledge for the development of policy and position papers; provide advisory and resource information in these areas for AHA.
 2. Stimulate basic and applied research in nutrition, identifying areas of needed or expanded investigative effort and emphasis.
 3. Promote in cooperation with the Scientific Councils and committees of AHA appropriate and well designed nutrition programs in coronary artery disease and hypertensive disease; to promulgate in cooperation with AHA affiliates public education programs concerning nutrition in these areas.
 4. Advise the Working Group on Public Policy and Government Affairs in matters concerning legislation and government regulation related to food, nutrition, health and cardiovascular disease.
 5. Cooperate with and assist other scientific and health agencies in programs concerning nutrition and cardiovascular disease.
 6. Disseminate information pertinent to nutrition and cardiovascular disease to health professionals through news and medical media and with the cooperation and assistance of affiliates.

AMERICAN HEART ASSOCIATION

(Name of Committee)

(Date)

Record of Compliance with the Association's Conflict of Interest Policy

As a Board and/or Committee Member, the following is a record of my abstention from voting at this meeting with respect to any instance which may be deemed a potential conflict of interest:

Council Affairs Committee:

Fund Raising Committee:

Management and Finance Committee:

Sub-Committee on Investments and Business Operations:

Sub-Committee on Personnel and Training:

Minority Involvement Working Group:

Program Committee:

Publications Committee:

Public Relations/Public Information Working Group:

Research Committee:

Steering Committee on Medical and Community Program:

Other:

(Signature)

(Print Name)

AMERICAN HEART ASSOCIATION
NUTRITION COMMITTEE

AHA National Center
Dallas, Texas

Thursday, April 5, 1979
Friday, April 6, 1979

AGENDA

			<u>Exhibit</u>
I.	Review of Minutes and Action Items	E. Bierman	A ✓
II.	Report from Subcommittee	C. Ford	B ✓
III.	Reports from Liaison Members		
	A. Council on Arteriosclerosis	F. Mattson ✓	
	B. Council on CVDY	W. Weidman ✓	
	C. American Diabetes Association	B. El-Beheri ✓	
IV.	Report from Ad Hoc Committee to Design A Dietary Treatment for Hyperlipoproteinemia	S. Grundy and C. Ford	C ✓
V.	Nutrition Film	E. Bierman	D ✓
VI.	Nutrition/CVD Conference	E. Bierman	E-1 E-2
VII.	Review of Public Policy Paper/Nutrition	E. Bierman	F ✓
VIII.	Nutrition Consortium Support	E. Bierman	G
IX.	HDL Professional Educational Material	E. Bierman	H
X.	Fat Modification of Foods	E. Bierman	I
XI.	TV Nutrition Ads to Children	E. Bierman	J

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|--------|---|-------------|---|
| XII. | Public Education Piece on Triglyceride | J. Farquhar | K |
| XIII. | Heart Healthy Airline Meals --
Chicago Heart's Proposal | E. Bierman | L |
| XIV. | Co-sponsorship of Heart Association
Nutrition Symposiums | E. Bierman | M |
| XV. | Position Papers on Nutrition/Hypertension | E. Bierman | |
| ✓ XVI. | Suggestions for Cardiovascular Conferences
and How-To Sessions for Annual Meeting
November 1979 | | ✓ |
| XVII. | Guest Speakers | | |
| | A. Relationship of Salt to Hypertension | R. Prineas | ✓ |
| | B. Cholesterol in Childhood --
Toward a Public Policy | D. Berwick | |
| | C. Overcoming Obesity | G. Bray | ✓ |
| XVIII. | Future Meeting Dates | | |

AMERICAN HEART ASSOCIATION
NUTRITION COMMITTEE
MINUTESBahia Mar Hotel
Ft. Lauderdale, Florida

October 25-26, 1978

Members PresentEdwin Bierman, M.D., Chairman
John Brunzell, M.D.
Robert Corwin, M.D.
Cynthia Ford, R.D.
Jack C. Geer, M.D. (26th)
Scott Grundy, M.D., Ph.D.
William Insull, Jr., M.D.
Lewis Kuller, M.D. (26th)
Fred Mattson, Ph.D.
John Mueller, M.D. (26th)
Robert Shank, M.D.
William Weidman, M.D. (25th)GuestsBarbara El Beheri, R.D. (25th)
Donald Zilversmit, Ph.D. (26th)StaffMary Winston, Ed.D.
Isabel Johnson, Recorder

Minutes of the February 27-28, 1978 meeting were reviewed and approved with the following change on the first page of the Nutrition and Public Policy attachment: "Changes the recommendation for salt intake from 2-3 grams/day to 5 grams/day."

ACTION ITEMS

1. APPOINTED Drs. Brunzell and Corwin as liaison to planning group for a workshop on obesity sponsored jointly by AHA and CVDY Council.
2. AGREED Dr. Grundy will work with Mary Winston on changes in the Position Paper on Nutrition Education.
3. AGREED Mary Winston will draft a letter of response to United Airlines article and send to Dr. Bierman for review.
4. AGREED Dr. Glueck will be asked to revise his statement for Affiliates on high density lipoprotein cholesterol, incorporating suggestions from Committee's review. AHA science writer will review, Dr. Bierman will receive before distribution to Committee.
5. AGREED Dr. Mueller will revise his statement for Affiliates on alcohol, send to Mary Winston, AHA science writer will review, Dr. Bierman will receive before distribution to Committee.

6. RECOMMEND professional education material in the field of HDL be developed.
7. AGREED Mary Winston will send piece on triglycerides from Washington Affiliate to Committee for review.
8. AGREED Committee supports the "one diet" concept.
9. MSC that Committee requests final report from Committee to Design a Dietary Treatment for Hyperlipoproteinemia by March 1, 1979.
10. MSC that Cynthia Ford's recommended version of "Eat to Your Heart's Content" will tentatively be used as Phase I, and this will be submitted to MRFIT coding center for computer analysis.
11. MSC that Committee go on record as strongly encouraging support for up-dating of food composition tables to validate data from nutrition intervention studies, e.g. fat, cholesterol, sodium, and other nutrients of concern to CVD.
12. MSC that letters to this effect go to NHLBI, Dr. Robert Levy, with cc to Dr. Zukel; to Agriculture, Dr. Mark Hegstead, with cc to Dr. Jack Iacano; to Director, Hanes Study, with cc to Basil Rifkind.
13. MSC that AHA award funds to the Nutrition Committee for the determination of variations in the diet, "The Way to a Man's Heart", as determined by computer analysis of foods as purchased in the market place.
14. AGREED Committee commends Cynthia Ford and her committee for their work on calculations for the revision, "Eat to Your Heart's Content."
15. CONSENSUS of Committee that the Nutrition/CVD Conference in progress be repeated in two years at a location in the Western U.S.
16. MSC to send letter to Dr. Levy to urge that a workshop be funded to review Cholesterol Index tool and other educational tools. Dr. Kuller will draft letter with cc to Don Frederickson at NIH, and William Zukel. A similar letter will be sent to Senators Dole and McGovern.
17. MSC to invite an expert on salt and hypertension to the next meeting.
18. AGREED next meeting will be either March 1-2 or March 5-6, 1979 at National Center.

DISCUSSION

Report from Subcommittee of Nutritionists was received from Cynthia Ford: The Creative Cuisine program package has been distributed to all heart associations. The low sodium cookbook, Cooking Without Your Salt Shaker, will be available after the first of the year. Plans are being made to have the Food for Thought game available by April,

1979. The first regional Nutrition Counseling Workshop was held in Newark, NJ in October. It was well attended by approximately 200, a small percentage of whom were physicians. The Association is involved with NHLBI in a pilot nutrition education research program in the Giant Food Stores in the Washington, DC area. The nutrition aspect of the Heart Health Education in the Young program is being designed to mesh with the school lunch program. The material will be organized in a total program and will consist of information on how to implement a workshop. The general guidelines for children following a prudent diet will be adapted to the requirements of the school lunch. Phase I of the program will be designed for food service directors and workers and will consist of the following:

- 1) Introduction and rationale: risk factors, obesity, legislation, evaluation, bibliography.
- 2) Clear explanation of implications of risk factor education to school food service; how to do it (Food purchasing, specifications, recipe modification, eating patterns, breakfast, snacks, lunch, use of seasonings to reduce sodium content of diet).
- 3) Change from the food service workers point of view, food abuse, attitudes and behavior, resistance to change, understanding significance of risk factor education.
- 4) The program will be developed in modules (1 hour, 2-3-4 hours). Emphasis will be placed on the necessity of the sessions being participatory.

Guidelines will be developed for use of heart association program staff in approaching school personnel for purposes of encouraging them to participate in the program, suggestions will be given for publicity of program.

After review of the proposed Position Paper on Nutrition Education, Dr. Grundy was asked to assist Mary Winston with developing a paragraph dealing with familial hyperlipidemia.

Information Item

Letter to United Airlines - The article, "To Your Health" (see Attachment I) which appeared in United Airlines Friendly Times, was discussed. United Airlines offers passengers a choice of fat-modified meals, and a letter from American Heart was previously sent complimenting them on their efforts. In view of this, it was felt that a letter should be sent to the airline pointing out the misinformation in the article. Mary Winston will draft and send to Dr. Bierman for revision.

Liaison Reports

- A. Council on Cardiovascular Disease in the Young - Dr. William Weidman reported that the Council is planning a closed workshop on obesity in childhood, hopefully to be sponsored by NHLBI, AHA, and the American Academy of Pediatrics. He

requested liaison representation from the Nutrition Committee for the planning. Drs. Brunzell and Corwin were appointed to serve in this capacity. The representative from the American Diabetes Association, Barbara El Beheri, expressed interest in working with this planning group. Dr. Weidman requested assistance from the Nutrition Committee on the issue of salt in children's diets, e.g. what is the average salt intake of children, how can sodium input in children be measured, and the sodium content of processed foods.

- B. Council on Arteriosclerosis - Fred Mattson, Ph.D. had no report at this time as the executive committee meets in November.
- C. American Diabetes Association - John Brunzell, M.D. reported that ADA has employed a nutritionist, Barbara El Beheri, who attended the meeting. He suggested that if there is no objection from the Nutrition Committee, it would be mutually beneficial to have her attend our meetings. However, an official liaison from the ADA, a volunteer with policy making powers, should also be appointed to AHA's Nutrition Committee.

ADA is publishing a cookbook based on AHA dietary principles. Dr. Brunzell expressed the need for long-term studies in diabetics using nutritive sweeteners such as fructose and sucrose. He referred the Committee to an article in the September 1978 issue of Diabetic Care.

Information Statements for Affiliates

- A. HDL -Committee reviewed the draft prepared by Dr. Glueck. He will incorporate suggestions for revision, and send revised statement to Dr. Bierman for approval. Statement will be referred to a science writer for editing, and circulated to committee members for final approval.
- B. Alcohol - Comments were received on the draft statement on alcohol. Dr. Mueller will incorporate them in the revised statement and return to Mary Winston who will ask science writer to review. Statement will then be sent to Dr. Bierman for approval and circulated to Committee.
- C. Triglyceride - Committee reviewed statement prepared by Dr. Brunzell. Comments had been received previously from committee members. Additional suggestions were made at the meeting. These will be incorporated in the statement and it will be recirculated to Committee for final approval. A statement on triglycerides prepared for the general public by Washington State Heart Association's nutrition committee will be circulated to committee members for review and possible publication by AHA.
- D. Salt and Hypertension, Salt and Obesity - Papers were prepared on these topics by Drs. Mueller and Geer. Each presented a different point of view. The Committee expressed concern about the kind of recommendation that could be made. It was agreed that the first step is an in depth committee self-education on the subject. Dr. Bierman feels the Committee will benefit greatly by the appointment of Dr.

Harriet Dustan, an expert in hypertension. In addition, effort will be made to identify a scientist particularly knowledgeable in regard to salt and hypertension and invite him to the next meeting.

Report from Committee to Design a Dietary Treatment for Hyperlipoproteinemia

Dr. Grundy reported on the committee's July 14 meeting and requested guidance from Nutrition Committee. The Nutrition Committee supports the "one diet concept." Dr. Grundy will prepare a report to the Committee to Design a Dietary Treatment for Hyperlipoproteinemia based on Nutrition Committee's discussion. (See Attachment II) Committee members were requested to look over Dr. Grundy's memo. It was MSC that Nutrition Committee requests final report from this group by March 1, 1979.

Review of Material to be Prepared by Subcommittee of Nutritionists for Above Committee

Cynthia Ford presented revised calculations for food tables reflecting the teaching in "The Way to a Man's Heart" as well as calculations for the revision to be entitled "Eat To Your Heart's Content." Since AHA is planning to revise both this basic diet pamphlet and the cookbook, preparation of therapeutic diets for the Phase II modification has been deferred until decisions on the basic diet (Phase I) are made. It was MSC that the proposed "Eat To Your Heart's Content" which will be used as Phase I be submitted to the MRFIT coding center for computer analysis. MSC that the following motion prepared by Dr. Shank be sent to Dr. Levy at NHLBI, and to the Director of the Hanes Study. Dr. Mattson will develop a similar letter to be sent to Dr. Mark Hegstead at the U.S.D.A.

Motion -

The Nutrition Committee of the American Heart Association, increasingly aware of the large problems associated with completing and updating tables of food composition for purposes of evaluation of the dietary intake of Americans and seeking information concerning the health importance of intakes of other nutrients not usually included in these tables, proposes that appropriate government agencies be encouraged to develop support for programs which will provide for the collection and analysis of meal aliquots selected and consumed by an appropriate sample of the American population.

MSC that AHA award funds to the Nutrition Committee for the determination of variations in the diet, "The Way to a Man's Heart," as determined by computer analysis of foods as purchased in the market place. AGREED Committee commends Cynthia Ford and her committee for their work on calculations for the revision, "Eat to Your Heart's Content."

Guest Report

Cholesterol Index of Foods - Dr. Donald Zilversmit presented this to Committee. He has developed an index based on the cholesterol, polyunsaturated and saturated fat content of food, and previously developed equations, which allow one to predict the

effect of a particular food on serum cholesterol. If an individual follows AHA dietary recommendations, his total cholesterol index will add up to 100; an individual following a typical American diet will have a total index of 200-220. The Cholesterol Index is a form of food exchange list, each food being assigned a specified number of points, e.g. egg has a cholesterol index of 49. The index is additive. It has been field tested on forty patients. A 2400 calorie diet was used as the base. The formulas used to calculate the index are as follows:

$$CI = (1.0I (S-0.5P) + 0.05C) \times 3.51$$

$$CI_2 = (0.81S - 0.62P + 0.0677C) \times 3.94$$

In the discussion, the Committee members cautioned that it is necessary to heed the caloric value of food. The question arose as to whether or not this is the best way to weight these foods. Also, it appears that an individual could add more than 10% polyunsaturated fat to his diet. The suggestion was made that a fat-calorie index would be a more ideal tool.

Dr. Kuller recommended that a workshop be designed to look at all of the available educational tools related to the fat-modified diet. It was MSC that a letter be sent to Dr. Robert Levy at NHLBI encouraging him to designate funds for organizing a workshop for this purpose. A similar letter should go to Senators Dole and McGovern.

Nutrition Film - Committee feels they have specified objectives for the film and this should be sufficient. Official diet statements must be followed in regard to the message. Committee agreed they were particularly impressed with the film, "High Blood Pressure: If Only It Hurt A Little", and asked if a similar technique might be considered for the nutrition film.

Professional Education - Council on Epidemiology is sponsoring a Conference on Exercise in March 1979. Dr. Kuller suggested this Council would be enthusiastic about co-sponsoring a course with the Nutrition Committee in 1980. CONSENSUS of Committee that the Nutrition/CVD Conference be repeated in two years at a location in the Western U.S. Also, AGREED that faculty be asked to send bibliographies of their presentations given at the Florida conference which, when compiled, will be mailed to all participants.

Future Meeting Date - AGREED next meeting will be at National Center March 5-6, 1979 (Monday and one-half day Tuesday), March 15-16 (Thursday and one-half day Friday), or March 14-15 (Wednesday and one-half day Thursday). Committee will be polled to determine exact date.

Friendly Times

United Airlines
P.O. Box 66100, Chicago, Ill. 60666

If undeliverable, please destroy.

EMPLOYEE CHANGE OF ADDRESS: Whenever you move, report your new address immediately to your supervisor. A correcting UG 100 then will be sent to EXO Payroll, which maintains the master employee home address list used for all such company mailings. Be sure to include your ZIP CODE.

ATTACHMENT I

By
DR. RICHARD HARPER
Ex 0 MD.

To Your Health

The dangers of high fat diets have been known for years, but the American Heart Association has chosen not to emphasize significant reductions in total fat intake. (Our diets contain 40 to 50 percent of total calories in fat.) The AHA decided instead to push the substitution of unsaturated fat for saturated fat.

Unsaturated fats or oils are found most readily in vegetable products such as corn and safflower. They contain no cholesterol. Studies show that an increased intake of unsaturated fats causes a slight reduction in the cholesterol blood level of some people.

"If you eat less cholesterol-containing fat and more unsaturated fat, you are not accomplishing much except supporting the vegetable oil industry!"

The AHA approach held out the possibility to the public that it need not forego its eating habits or endure the low-fat, low-cholesterol diet to secure immunity from heart disease. The approach suggested one need merely substitute unsaturated for saturated fats.

The food industry, ever sensitive to profitable situations, quickly responded. Foods high in unsaturated fats, such as margarine and mayonnaise, were touted as relative remedies for coronary heart disease.

New products appeared which substituted saturated fats for unsaturated fats in ice cream, sauces and other favorite edibles, such as cholesterol-free egg substitutes.

The preoccupation with this substitution of unsaturated fats has dominated the nutritional world since the American Heart Association's sanction of this approach by adopting it as its primary preventive recommendation.

The available evidence is emphatic, however, about the utter futility of this approach.

Here is a capsule summary of two studies on unsaturated fat diets:

London researchers in 1968 compared, after six years, 200 people on a 46 percent fat diet with unsaturated fats with 200 people eating a 46 percent fat diet with "normal" ingredients. All 400 persons had recovered recently from heart attacks.

"It's time for the game of fats to stop. Fat is fat is oil is fat... whether it's from animals, coconuts, peanuts or cashews."

Results showed no difference in total deaths due to coronary heart disease between "dieters" and those on regular diets and no difference in major relapses.

In the United States, half of the 846 men living in an institution where their food was specially prepared were given a high unsaturated fat diet and the other half the "normal" diet. Both diets had 40 percent of the total daily calories in fat.

Eight years later the results showed no significant difference in death rate or heart

This eight-year study was the longest clinical trial in the U.S. of a diet high in unsaturated fat. I wonder whether the American Heart Association has reviewed the literature?

Many animal studies show that diets high in unsaturated fats may lower the cholesterol in the blood. That's great, but where does the cholesterol go?

It is forced into the tissues, and when cholesterol levels in the tissues become elevated, cholesterol crystals begin to form. In this crystal form, cholesterol can be a potent cancer-causing agent.

Studies showing the fallacy of substituting different fats instead of reducing total fats have been ignored by the National Dairy Council, the National Heart and Lung Institute, the American Heart Association and the general food industry.

When research showed unsaturated fat dangers, the dairy industry cheered from the saturated-fat bleachers. When research showed that total fat, regardless of its type, was related strongly to arterial disease, the dairy industry gave its annual award to an esteemed researcher who showed that unsaturated fats forced the cholesterol into the walls of arteries and tissues. This, of course, shifted the onus of guilt back to the unsaturated fat food industry.

I think it's time for the game of fats to stop. Fat is fat is oil is fat. Whether its from animals, coconuts, peanuts or cashew-nuts, by the way, are 70 percent fat—is not the issue. It is the total amount that counts.

"... vitamin B 12 is the only essential ingredient necessary for good health that is not found in the vegetable kingdom."

If you eat less cholesterol-containing fat and more unsaturated fat, you are not accomplishing much except supporting the vegetable oil industry. You are still developing the fatty plaques in your and your children's arteries.

What should we eat? Foods such as whole grains, pasta, beans, vegetables and potatoes that contain more complex carbohydrates.

For example, instead of spaghetti sauce with oil and one pound of hamburger (70 percent fat) use four ounces of lean ground stew meat and eliminate the oil. Learn to use meat more as a condiment or not at all.

Remember that vitamin B12 is the only essential ingredient necessary for good health that is not found in the vegetable kingdom. Simple rotation of low-fat dairy products such as skim milk and dry curd cottage cheese with fruit, and vegetables, beans, potatoes and whole grains will provide all the essential minerals, vitamins (except B12), amino acids (protein building block) and fatty acids.

In normal health, if you ate only a half a pound of meat per week you would have more than adequate intake of vitamin B12. In fact, vitamin B12 is stored in the body and would take several years to deplete if you ate no meat.

Employees interested in recipes for low-fat cookery should invest in a copy of "Live Longer Now," written by Jon Leonard and Elaine Taylor and published by Grosset & Dunlap, N.Y.

Published by United Airlines

July 12, 1978

Friendly Times

October 30, 1978

TO: Committee to Design a Dietary Treatment for Hyperlipoproteinemia

FROM: Scott M. Grundy
Representative of Committee to Nutrition Committee

SUBJECT: PROPOSAL GROWING OUT OF DISCUSSION AT NUTRITION
COMMITTEE MEETING OF OCTOBER 25 AND 26, 1978

After a thorough discussion of the one-diet concept for the treatment of hyperlipoproteinemia, the Nutrition Committee was in general agreement with the following principles:

1. The "one-diet" concept for treatment of hyperlipoproteinemia was accepted. By "one-diet" is meant a low-fat, low-cholesterol diet. This diet can be used for all forms of hyperlipoproteinemia ranging from familial hypercholesterolemia to hyperchylomicronemia in its various forms. This one-diet is intended to be used in the place of a) a high-polyunsaturated fat diet that has been recommended for treatment of hypercholesterolemia and b) a low-carbohydrate, high-fat diet that has been advocated for hypertriglyceridemia.
2. A step-wise approach to lowering of fat and cholesterol content of the diet would seem reasonable. Logically, Step I (or Phase I) should be identical to the AHA diet recommended for the general public. This diet would contain 30-35% of calories as fat and approximately 300 mg cholesterol per day. Approximately 10% of total calories would be as polyunsaturated fats.
3. Phase II would be lower in fat (25-30%) and cholesterol (approximately 200 mg per day). Again, 10% of total calories would be as polyunsaturated fats.
4. Phase III would be still lower in fat (20-25%) and would contain about 100 mg cholesterol per day. Polyunsaturated fats would again constitute 10% of total calories. It was recognized that this diet would be difficult to achieve by most patients. There was considerable question whether it is a realistic diet, and the possibility was raised that it might not be nutritionally adequate.
5. Weight reduction should be recommended in addition to institution of a low-fat, low-cholesterol diet. It was recognized that this kind of diet is ideal for caloric restriction. Weight reduction is an integral part of treatment of hyperlipoproteinemia.

6. In patients with severe hypertriglyceridemia and chylomicronemia, rapid reduction of fat content is imperative. The purpose of rapidly reducing fat is to prevent acute pancreatitis. This purpose should be distinguished from the development of a long-term diet for prevention of atherosclerosis.
7. It was recommended that polyunsaturated fats be kept at a maximum of 10% of calories throughout the progression to lower contents of fat. This would increase the P/S ratio which in itself should contribute to further lowering of plasma lipids. A higher level of polyunsaturated fats cannot be recommended at this time because of the unknown effects of long-term ingestion of large quantities of these fats.
8. The recommendation of the Committee for Dietary Treatment of Hyperlipoproteinemia should take note of the fact that drug therapy may be indicated for severe forms of hyperlipidemia in addition to dietary therapy. However, there is considerable evidence that maintenance of strict dietary control will accentuate the action of hypolipidemic drugs, and diet therapy should always be continued during drug treatment. It was recommended that should polyunsaturated be given above 10% of calories for hypercholesterolemic patients, this increment should be considered in the category of drug therapy. It is because the benefit-risk ratio of polysaturated fats at higher levels of intake has never been determined.

The Committee had mixed feelings about the advisability of recommending Phase III (20-25% fat; 100 mg cholesterol) at this time. This diet has not been given an extensive trial, and we do not know whether it is possible to prepare a nutritionally-adequate diet that can be well tolerated or accepted for prolonged periods of time. The suggestion was also made that the use of three phases also detracts from the one-diet concept because the three phases are really three different diets. One approach would be to recommend the Phase II diet as the "one-diet" for treatment of hyperlipidemia.

The Nutrition Committee urged the Committee for Dietary Therapy to present a final recommendation by the time of the next meeting of the Nutrition Committee meeting, that is, March 1, 1979.

/sea

AMERICAN HEART ASSOCIATION
AD HOC COMMITTEE ON SCHOOL NUTRITION

MINUTES

AHA National Center
Dallas, TexasFriday, January 19, 1979
8:30 a.m. - 2:30 p.m.

Members Present: Chairman, Cynthia Ford; Deborah Deatricks; Joan Luck; Annamarie Shaw; Kathleen Stitt, Ph.D.

Staff: Mary Winston, Ed.D.; Betty Tevis, Ph.D.; Isabel Johnson, Recorder

ACTION ITEMS

1. APPROVED the minutes of the September 8, 1978 meeting with the following deletions: Under Discussion Item III, (1) "obesity" and (3) "food abuse".
2. AGREED the Position Paper on Nutrition Education in the Young will stand as it is as support for the Committee's work with the addition of more emphasis on the food service worker's role, as well as the listing of the Cooperative Extension Service.
3. AGREED Deborah Deatricks's curriculum material entitled "Risk Factors of Cardiovascular Disease" will serve as a reference paper for the person presenting the workshop.
4. AGREED Annamarie Shaw will prepare a list of food and menu modifications incorporating ideas from the California nutrition workshop. These will be designed for answering: "What is fat-controlled cookery?" "What can you, the food service worker, change?" Suggested activities will be included.
5. AGREED Cynthia Ford will share the paper she presented at the Nutrition/CVD course with Annamarie Shaw.
6. AGREED Joan Luck will prepare a listing of "gatekeepers" from national level through school lunch worker.
7. AGREED Joan Luck will send Mary Winston an updated list of state nutrition education coordinators.
8. AGREED committee members will each be sent a copy of "Children's Help Your Heart Cookbook" as soon as they can be obtained.
9. AGREED Joan Luck will prepare section on legislation for the guide.
10. AGREED Joan Luck will discuss A General Guide to Food Choices in School Lunch Pattern Requirements with Dorothy Callahan and together they will modify these to be as practical as possible. Joanne Styer has some comments on this guide which she will share with the committee.

11. AGREED Joan Luck will send Mary Winston basic guidelines developed by USDA relative to salt, fat, sugar.
12. AGREED Annamarie Shaw will attempt to identify slides which would demonstrate marketing food and dealing with obstacles in school lunch.
13. AGREED Joan Luck will find out whether cartoon slides on the above subjects developed by USDA are still available.
14. AGREED Deborah Deatruck will work on an evaluation section for the guide.
15. AGREED Cynthia Ford will develop section on Nutritional Habits and Change.
16. AGREED Kathleen Stitt will develop the first section of the guide following the format of Guidelines for Nutrition Programming in the Community.
17. AGREED committee members will send completed assignment materials to Mary Winston by May 1, 1979.
18. AGREED next meeting will be May 18 at the National Center.

DISCUSSION

I. Position Paper on Nutrition Education in the Young

It was agreed the Position Paper on Nutrition Education in the Young will stand as support for the Committee's work. More emphasis on the food service worker's role will be added, as will the listing of the Cooperative Extension Service.

II. Concepts for Developing Risk Factor Education

Deborah Deatruck presented this curriculum material on risk factors of cardiovascular disease, a pre/post test, and an outline for a demonstration/activity on blood pressure and smoking. Results of the Baylor study will be added when they are available. Mary Winston suggested reference to heredity and to lowering weight should be included in the curriculum material. It was agreed this paper will serve as a reference for the person presenting the workshop. An accompanying set of slides would be helpful. It was suggested possibly the first two pages of this material could be a curriculum guide with options for the instructor. All components will be enclosed in a binder. Members were requested to look over this material and send comments or suggestions to Deborah Deatruck.

III. General Guidelines for Children Following a Prudent Diet

It was suggested these guidelines could be used as reference for workshop instructor. They would be difficult to implement as they are. It is expected that Joanne Styer's material will be a practical application of these guidelines.

A recipe presentation (before modification, after modification) was suggested. Also presentation of practical information on cholesterol, saturated fat; whole milk - 2% - skim (differences, cost); cooking beef patties; salt, sugar, fat; "what is fat-control cookery?" - education tool.

U.S.D.A. requirements for school meal patterns will be included in this section. Annamarie Shaw will make a list of ideas (modifications, changes) from the California nutrition workshop. Original lunch menus, same menus with changes. Practical suggestions and basic teachings on what we want them to know. Where does cholesterol come from, including an activity, slide suggestions. Cynthia Ford will share her Florida presentation with Annamarie, particularly examples presented of modifying recipes. Consideration will be given to preparing a card similar to Creative Cuisine waitress card for food service workers in schools.

IV. Food Selection, Buying Guide, Recipe Modifications

Since Joanne Styer could not be present, this material will be circulated to committee members for review.

V. Nutritional Habits and Change

This section will be personalized to get the interest of the food service workers and teachers. Teachers should be included because they work so much more with the child. Levels of "gatekeepers" from government down to child will be described. Joan Luck will list "gatekeepers" starting with chart of national level. This concept is a good one in regard to helping others form good food habits. Obstacles in workers own school should be identified and ways of overcoming these would be good topics for group discussions. "Neutral" and "non conflict" foods will be explained.

VI. Legislation Governing Child Nutrition Programs

This material is seen as resource (background) for workshop leader. It will also be appropriate for parents' section. After review of all the material, there is much which seems appropriate for the parent and teacher section. Dorothy Callahan's breakfast list could be used for a PTA presentation, e.g., "Does your school have a breakfast program?" This is also true of material on snacks prepared by Joan Luck.

VII. Heart Health Education In The Young

Betty Tevis, AHA Chief of Heart Health Education in the Young, gave the group a background and explanation of the guide, "Put Your Heart Into Curriculum". Each member was given a copy of this publication.

VIII. Proposed Organization of the Workshop Guide

Committee reviewed the material discussed and outlined the organization of the guide as follows:

A. Introduction

1. Statement of purpose: What is it? Who is the audience? How is guide to be used? How units in guide have been set up. How to use the subject matter modules.
2. How to set up a workshop. (Kathleen Stitt will prepare this portion following outline of Guidelines for Development of Nutrition Programs.)

3. Resources (individuals, organizations)
 4. Legislation related to School Lunch (could be personalized for each state). (J. Luck)
 5. Outline of Workshop Module
 6. Position Paper
- B. Risk Factors (Deborah Deatruck)
1. How to use this section
 2. Background paper on risk factors (Arrange in narrative form and underline topics). Include Baylor study, stress importance of relationship of obesity to high blood pressure; address issue of heredity/CVD. This paper can be the basis for the development of a talk suitable for various audiences.
 3. Objectives for section
 4. Options for workshop leaders in presenting material
 5. Suggested activities (include take-home pieces)
 6. Visuals (slides, overheads, etc.)
 7. Evaluation tool
- C. Nutritional Habits and Change (Cynthia Ford) (follow above outline and format)
- In this section role of gatekeeper in school lunch will be expanded - levels of gatekeepers will be described, leading to child himself. Joan Luck will develop list of gatekeepers. One suggested activity in this section is role playing relative to serving food.
- D. How To Section (Annamarie Shaw, Joan Luck, Dorothy Callahan, Joanne Styer)
1. Introduction (narrative as in other sections)
 2. Objectives for unit
 3. General guide to food choices (this will be liberalized in keeping with realities of school lunch)
 4. Principles of fat-controlled cookery (outline)
 5. Suggestions for seasonings to reduce salt and sugar (Joan Luck will send basic guidelines on salt - fat - sugar)
 6. Suggestions to food service worker for making changes

7. Visuals (e.g. slide on modifying a recipe), (difference in whole and skim milk and chocolate milk relative to cost and fat content - slide describing saturated fat, polyunsaturated fat), marketing food - Annamarie Shaw and Joan Luck will try to locate this information.
8. Activities, e.g. demonstration of fat content of various types of ground beef, cooking meat, draining, blotting - discussion of cost factor; group discussions on identification of obstacles in each school to implementing changes; marketing food.

E. Evaluation (Deborah Deatrick)

- IX. Copies of the "Children's Help Your Heart Cookbook" will be sent to each committee member.
- X. Mary Winston will send samples of risk factor educational materials to Deborah Deatrick.
- XI. Assignments were accepted as indicated above and members were requested to send completed assignment materials to National Center for duplication before mailing of the next meeting agendas on May 1, 1979.
- XII. Next meeting will be May 18 at the National Center.

EXHIBIT C

MEMORANDUM

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
NATIONAL INSTITUTES OF HEALTH
NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

TO : Antonio M. Gotto, M.D.
Chairman, Committee to Design a Dietary
Treatment for Hyperlipoproteinemia

DATE: March 19, 1979

FROM : Cynthia Ford

SUBJECT: Phase I and Phase II Diets

Enclosed are food tables, diet calculations, food patterns and sample menus designed to meet the Phase I and II specifications (listed below) as agreed upon at the November 12 meeting of the Committee to Design a Dietary Treatment for Hyperlipoproteinemia.

Phase I

30-35% calories from fat
300 mg cholesterol

Phase II

30% calories from fat
200-250 mg cholesterol

8-10% calories from polyunsaturated
fatty acids for all diets.

All diets have been calculated by hand using the food composites and weightings which were sent to the Nutrition Coding Center and calculated by computer using nutrient values that are currently in use for both the LRC and MRFIT programs.

The material is presented in 3 sections:

- Part I - Diet calculations, food patterns, and sample menu to illustrate the Phase I and II 2300 calorie diets and low calorie versions (1200 and 1800 calories) of Phase II.
- Part II - A week's menu planned using only general guidelines (not a specific food pattern) for the Phase I diet and calculated by computer at the Nutrition Coding Center. This procedure was requested by the Nutrition Committee to evaluate a diet that might be planned by a housewife using only general guidelines (similar to information in The Way to a Man's Heart) to see how closely it might follow the desired level of nutrient intake. Menus were prepared by Marilyn Farrand and submitted to the NCC for calculation. Menus are included along with a summary table showing the average daily intake of nutrients. In addition, the average daily food pattern as derived from the sample menus is compared to the Phase I diet plan which is under consideration.

Part III - Food tables for Phase I and II diets, weighting factor analyses for obtaining values for meat, fish, and poultry and composites for each food group are included. All calculations were done by computer at the NCC using the latest nutrient data.

Please disregard all food tables and diet calculations that have been circulated for previous meetings. They were based on food tables calculated by hand; also several of the foods included within a food group composite have been changed.

cc: Members, Committee to Design a Dietary Treatment for Hyperlipoproteinemia
Nutrition Committee Members
William J. Zukel, M.D.
Marilyn Farrand
Lynne Scott

PART I

Table 1 - 2300 calorie diet.

Values are close to task force guidelines. Low fat milk will be recommended -- no specific percentage of fat will be specified (for calculations, 2% used by NCC for unknown types). Eggs reduced to 2/wk from present 3/wk on AHA basic diet. Fats and oils will not be specified by type or P/S for Phase I. Calculations used by NCC were for margarine, unknown brands, home use, and unknown salad oil. Baked goods and desserts are calculated by NCC from recipes calling for low fat milk, egg substitute and unknown type salad oil and margarine. P/S slightly lower than desired but 4 TB. (12 tsp.) oil, margarine or dressings seems about the limit. To incorporate this amount, I found it necessary to plan a deep-fried item on the menu. The amount cannot be reduced or the total fat is lowered from the 30-35% goal. With 300 mg cholesterol, a sizeable amount of saturated fat is present so poly intake needs to be high.

The one way polys might be increased without altering total fat would be to specify high poly oils and margarine. I felt this step should be considered therapeutic and left for Step II where instructions on exact oils and label reading of margarines would be included.

If a lower fat content milk is used, total fat and cholesterol levels are below Phase I task force goals.

Table 2 - Phase II 2300 calorie diet.

Eggs deleted. Margarine and oil usage decreased from 12 to 7 tsp/day. One additional serving of CHO added to bring up calorie intake. Poly oils and margarine will be specified thus P/S ratio improves.

Table 3 - Phase II 1800 calorie diet.

When calories decreased, fat and cholesterol percentages drop. P/S is higher but the actual amount of poly oil and margarine recommended is decreased. Iron intake starts to fall -- caution might be advised for certain individuals.

Table 4 - Phase II 1200 calorie diet.

Percentage of calories from fat and cholesterol intake lower than goal due to low calorie level. If maintained at 30% fat, 200-250 mg cholesterol, unpalatable diet plan would result. Poly intake is reduced to 4 tsp. although P/S is high. Iron intake is low. Values for skim milk are shown on bottom line -- total fat then reduced to 26% of calories and cholesterol to 117 mg. Food choices are limited. For 1200 calorie diets, lower fat diets provide more variety in food choice and more total food. Usually they are only for a specified period of time and then are gradually increased.

Table 5 - Shows the progression of the food pattern from Phase I to Phase II and compares it to low calorie versions.

Table 6 - A menu designed to meet Phase I 2300 calories and then adjusted to fit Phase II diet plans. Numerical values shown were for tallying fat and oil intake.

Phase I Diet
2300 Calories

Food	Amt.	Calories	Protein	Fat	Cho.	Chol.	SF	MF	PF	Alc.	Fe
MFP	7 oz	399	56.0	16.45	0.7	184.8	5.60	6.16	1.96	0	6.3
Low Fat Milk (Unknown % fat)	2 cp	284	20.2	9.60	28.8	27.8	5.76	2.78	0.38	0	0.4
Eggs	2/7	24	1.8	1.64	0.2	72.0	0.48	0.64	0.20	0	0.4
Fats (Phase I)	4 TB	388	1.6	42.20	2.4	3.2	6.76	21.48	13.44	0	0
Breads, Cereals, St. Veggies, Soups	5 sv	580	20.0	9.50	104.5	4.5	2.65	2.75	3.35	0	5.5
Fruits	4 sv	312	3.6	1.08	79.2	0	0	0	0	0	3.2
Vegetables	3 sv	72	4.2	0.54	14.4	0	0	0	0	0	1.8
Baked Goods and Desserts	4/7	133	3.43	5.73	18.29	0.91	0.89	2.59	1.40	0	0
Alcoholic Beverages or Non-Fat Sweets	1 sv	117	0.4	0	6.0	0	0	0	0	13.2	0.1
		2309	111.23	86.74	254.49	293.21	22.14	36.40	20.73	13.2	17.7
			x4	x9	x4		x9	x9	x9	x7	
P/S = 0.94			444.92	780.66	1017.96		199.26	327.60	186.57	92.4	
% Cal			19.27	33.81	44.09		8.63	14.19	8.08	4.0	

Task Force Goal

30-35%

300

<10%

<10%

Table 2

Phase II Diet
2300 Calories

Food	Amt.	Calories	Protein	Fat	Cho.	Chol.	SF	MF	PF	Alc.	Fe
MFP	7 oz	399	56.0	16.45	0.7	184.8	5.60	6.16	1.96	0	6.3
Low Fat Milk (Exact % Fat Unkn)	2 cp	284	20.2	9.60	28.8	27.8	5.76	2.78	0.38	0	0.4
Eggs	0	0	0	0	0	0	0	0	0	0	0
(Phase II) Fats	1 Tsp 2 TB	37 224	0.1 0.4	4.17 25.04	0.1 0.4	0.1 0.6	0.56 3.34	1.14 6.86	2.33 14.00	0 0	0 0
Breads, Cereals, St. Veg., Soups	6 sv	696	24.0	11.4	125.4	5.4	3.18	3.30	4.02	--	6.6
Fruits	4 sv	312	3.6	1.08	79.2	0	0	0	0	0	3.2
Vegetables	3 sv	72	4.2	0.54	14.4	0	0	0	0	0	1.8
Baked Goods and Desserts	4/7 (4 sv/wk)	133	3.43	5.73	18.29	0.91	0.89	2.59	1.40	0	0
Alcoholic Beverages or Non-Fat Sweets	1 sv	117	0.4	0	6.0	0	0	0	0	13.2	0.1
		2274	112.33	74.01	273.29	219.61	19.33	22.83	24.09	13.2	18.4
			x4	x9	x4		x9	x9	x9	x7	
P/S = 1.25			449.32	666.09	1093.16		173.97	205.47	216.81	92.4	
% Cal			19.76	29.29	48.07		7.65	9.04	9.53	4.06	

Task Force Goal

30%

200-250

<10%

<10%

Table 3

Phase II Diet
1800 Calories

Food	Amt.	Calories	Protein	Fat	Cho.	Chol.	SF	MF	PF	Alc.	Fe
MFP	6 oz	342	48.0	14.10	0.6	158.4	4.80	5.28	1.68	0	5.4
Low Fat Milk (1% Fat)	2 cp	226	18.8	4.8	26.8	14.0	2.88	1.34	0.14	0	0
Eggs	0	0	0	0	0	0	0	0	0	0	0
Fats (Phase II)	2 TB	224	0.4	25.04	0.4	0.6	3.34	6.86	14.00	0	0
Breads, Cereals, St. Veggies, Soups	5 sv	580	20.0	9.5	104.5	4.5	2.65	2.75	3.35	0	5.5
Fruits	4 sv	312	3.6	1.08	79.2	0	0	0	0	0	3.2
Vegetables	3 sv	72	4.2	0.54	14.4	0	0	0	0	0	1.8
Baked Goods and Desserts	0	0	0	0	0	0	0	0	0	0	0
Alcoholic Beverages or Non-Fat Sweets	0	0	0	0	0	0	0	0	0	0	0
		1756	95.00	55.06	225.9	177.5	13.67	16.23	19.17	0	15.9
			x4	x9	x4		x9	x9	x9	x7	
			380.0	495.54	903.6		123.03	146.07	172.53	0	
% Cal.			21.64	28.22	51.46		7.01	8.32	9.83	0	

P/S = 1.40

Task Force Goal

30%

200-250

<10%

<10%

Table 4

Phase II Diet
1200 Calories

Food	Amt.	Calories	Protein	Fat	Cho.	Chol.	SF	MF	PF	Alc.	Fe
MFP	4 oz	228	32.0	9.40	0.9	105.6	3.20	3.52	1.12	0	3.6
Low Fat Milk (1% Fat)	2 cp	226	18.8	4.8	26.8	14.0	2.88	1.34	0.14	0	0
Eggs	0	0	0	0	0	0	0	0	0	0	0
Fats (Phase II)	1 tsp	37	0.1	4.17	0.1	0.1	0.56	1.14	2.33	0	0
	1TB	112	0.2	12.52	0.2	0.3	1.67	3.43	7.00	0	0
Breads, Cereals, St. Veggies., Soups	3 sv	348	12.0	5.70	62.7	2.7	1.59	1.65	2.01	0	3.3
Fruit	3 sv	234	2.7	0.81	59.4	0	0	0	0	0	2.4
Vegetables	2 sv	48	2.8	0.36	9.6	0	0	0	0	0	1.2
Baked Goods and Desserts	0	0	0	0	0	0	0	0	0	0	0
Alcoholic Beverages or Non-Fat Sweets	0	0	0	0	0	0	0	0	0	0	0
		1233	68.60	37.76	159.70	122.70	9.90	11.08	12.60	0	10.5
P/S = 1.27			x4	x9	x4		x9	x9	x9	x7	
			274.40	339.84	638.80		89.10	99.72	113.40		
% Cal.			22.25	27.56	51.81		7.23	8.09	9.20		
If Skim Milk Used		1179	22.73	25.53	53.37	117.3	5.57	7.54	9.51		

Task Force Goal

30%

200-250

<10%

<10%

Table 5

Foods	For General Public		Therapeutic	
	Phase I 2300 Calories	Phase II 2300 Calories	Phase II 1800 Calories	Phase II 1200 Calories
MFP	7 oz.	7 oz.	6 oz.	4 oz.
Milk	2 cp (low fat) ¹	2 cp (low fat) ¹	2 cp (1% fat milk or less)	2 cp (1% fat milk or less)
Eggs	2/wk	No egg yolk	No egg yolk	No egg yolk
Fats	4 TB (12 tsp) ²	2 TB + 1 tsp (7 tsp) ³	2 TB (6 tsp) ³	1 TB + 1 tsp (4 tsp) ³
Breads, Cereals, St. Veg., Soups	5 sv.	6 sv.	5 sv.	3 sv.
Fruits	4 sv.	4 sv.	4 sv.	3 sv.
Vegetables	3 sv.	3 sv.	3 sv.	2 sv.
Baked Goods and Desserts	4 serv/wk. (Homemade) ²	4 serv/wk. (Homemade) ³	None	None
Alcoholic Beverages or Non-Fat Sweets	1 sv.	1 sv.	None	None
	30-35% Fat <300 mg Chol. <10% SF <10% PF	30% Fat 200-250 mg Chol. <10% SF <10% PF	Phase II Specs. - Adjusted in line with calorie level	

¹Low Fat Milk - Pt. Instructions will not specify fat content. Calc. based on 2% fat.

²Pt Instr. will call for margarine and veg. oil - No specs as to brand, type or P/S.

³Pt Instr. will teach label reading of marg. - For P/S of 2:1 or more. Oil will be corn, safflower, pt. hydrog. soy, or cottonseed.

Sample Menu to Illustrate Food Patterns For Phases I and II

	2300 Calories - Phase I	2300 Calories - Phase II	1800 Calories - Phase II	1200 Calories - Phase II
Breakfast	1/2 grapefruit 1 poached egg (2/wk) 1 slice of toast 1 tsp. margarine ¹ 1 cup 2% fat milk coffee	1/2 grapefruit 1 cup cold cereal 1 slice of toast 1 tsp. poly margarine ¹ 1 cup 2% fat milk coffee	1/2 grapefruit 1 slice of toast 1 tsp. poly margarine ¹ 1 cup 1% fat milk coffee	1/2 grapefruit 1 slice of toast 1 tsp. poly margarine ¹ 1 cup skim (<1%) milk coffee
Lunch	vegetable soup roast beef sandwich (2 oz.) w/2 tsp. margarine ² 1 cup chef's salad w/1 TB. commercial french dressing ¹ 1/2 cup fresh fruit cup 1 cup 2% fat milk	vegetable soup roast beef sandwich (2 oz.) w/1 tsp. poly margarine ¹ 1 cup chef's salad w/1 TB. homemade french dressing (2 tsp. poly oil) 1/2 cup fresh fruit cup 1 cup 2% fat milk	vegetable soup roast beef sandwich (2 oz.) (no margarine) 1 cup chef's salad w/1 TB. homemade french dressing (2 tsp. poly oil) 1/2 cup fresh fruit cup 1 cup 1% fat milk	vegetable soup 1/3 cup low fat (1%) cottage cheese 1/2 cup fresh fruit 1 small pan roll 1 tsp. poly margarine ¹ tea or coffee
Dinner	cocktail 5 oz. breaded fried haddock (in salad oil) ⁵ 1 TB. tartar sauce ¹ 1/2 cup mashed potatoes w/ 1 tsp. margarine ¹ 1/2 cup green beans w/ 1 tsp. margarine ¹ assorted raw veg. relishes homemade orange cake (4 sv/wk) coffee	cocktail 5 oz. baked haddock w/ 1 tsp. poly margarine ¹ lemon wedges 1/2 cup mashed potatoes w/ 1 tsp. poly margarine ¹ 1/2 cup green beans w/ 1 tsp. poly margarine ¹ assorted raw veg. relishes homemade orange cake (4 sv/wk) coffee	4 oz. baked haddock w/1 tsp. poly margarine ¹ lemon wedges 1/2 cup mashed potatoes w/ 1 tsp. poly margarine ¹ 1/2 cup green beans w/ 1 tsp. poly margarine ¹ assorted raw veg. relishes 1/4 canteloupe coffee	4 oz. baked haddock w/1 tsp. poly margarine ¹ lemon wedges 1/2 cup green beans w/ 1 tsp. poly margarine ¹ 1 cup chef's salad w/ low calorie dressing 1/4 canteloupe coffee
Snack	1 large apple	1 large apple	1 small apple	-----

PART II

Table 7 - Summary table showing average daily nutrient intake of week's menu planned according to general guidelines for AHA basic Phase I diet.

Table 8 - Calculations show menus to average a higher than desired intake of total fat. When viewed in terms of a food pattern, fat and oil usage does not appear excessive. Obviously, there is some hidden fat in food preparation that is not accounted for in a food grouping system even though baked goods and desserts were calculated as a separate category.

Table 9 lists some of the obvious discrepancies that have been detected. The menus were not expected to conform exactly to a Phase I pattern as they were not planned with a specific number or size of portions or aimed at a set calorie intake but from general guidelines similar to the form used in The Way to a Man's Heart.

The areas of concern are the high total and saturated fat content as well as the surprisingly low level of iron. This reinforces the concern regarding iron content in diets that are low in calories or in animal protein foods as these diets averaged 7 oz. meat, fish and poultry daily.

Table 7

Phase I Diet - 2300 Calories
Summary Table for Menu Calculations

	Cal	Pro	Fat	Cho	Chol	SF	MF	PF	Alc	Fe
Sunday	2017	127.5	95.24	194.4	484.4	24.66	32.72	16.80	0	15.8
Monday	2327	103.8	91.51	284.6	208.5	26.37	38.20	20.80	0	13.3
Tuesday	2252	88.2	90.55	253.9	261.0	24.76	30.27	30.43	10.4	14.4
Wednesday	2672	116.0	118.59	291.9	346.1	30.23	62.27	18.23	0	17.5
Thursday	2021	88.5	74.09	261.4	198.0	27.64	25.72	13.75	0	13.8
Friday	2412	86.3	114.65	262.9	196.0	28.72	45.99	32.82	0	7.9
Saturday	3150	127.5	117.89	376.0	228.3	33.23	37.43	23.31	13.0	22.7
Total	16851	737.8	702.52	1925.10	1922.30	195.61	272.60	156.14	23.4	105.4
Av.	2407	105.4	100.36	275.01	274.61	27.94	38.94	22.31	3.34	15.06
		x4	x9	x4		x9	x9	x9	x7	
		421.6	903.24	1100.06		251.50	350.49	200.75	23.40	
% Cal.		17.52	37.53	45.70		10.45	14.56	8.34	0.01	

P/S = 0.80

Task Force Goal

30-35%

300 mg

<10%

<10%

Table 8

Sample Menus - Food Group Distribution

	<u>Sample Week's Menus</u>	<u>2300 Cal - Phase I Diet Plan</u>
	Average/Day	Average/Day
MFP	7 oz.	7 oz.
Low Fat Dairy Products	2 cp	2 cp
Eggs	1-1/2	2
Fats and Oils	10 Tsp.	12 Tsp.
Breads, Cereals, St. Veg, Soups	5 sv	5 sv
Fruits	3 sv	4 sv
Vegetables	2 sv	3 sv
Baked Goods and Desserts	5 sv/week	4 sv/week
Alcoholic Beverages and Non-Fat Sweets	{ 130 cal/day from sugars, sweets + 2 alc bev/week (235 cal) Total 164 cal/day }	1 sv { alc bev or equiv. cal in sugars, sweets 117 cal/day }

Obvious Discrepancies - Sample Menu and Food Pattern

- 1) Gravy 2 x in 1 wk Had not been added to food composites.
Contributed to SF & total fat calories.
- 2) Portion sizes. Food table - average serv. nuts - 1 TB.
Menu - 1 serv. = 2 oz.
Contributed to total & mono fat calories
- 3) Consumption of sugars, LF sweets and alcohol (mainly sugar syrup)
calc. from menu/day 164
according to food plan/day 117
42

SUNDAY

Breakfast

1 c orange juice
1 egg scrambled in
1 tsp. margarine
1 slice toast
1 tsp. margarine
coffee

Lunch

4 oz. roast chicken, no skin
 $\frac{1}{2}$ c mashed potatoes (Margarine and low fat milk)
 $\frac{1}{2}$ c chicken gravy
 $\frac{1}{2}$ c Brussels sprouts with margarine
2 T. cranberry sauce
 $\frac{1}{2}$ c fresh fruit
coffee

Dinner

1 c vegetarian baked beans, canned
3 oz. mock sausage patties (AHA)
1 c spinach salad
2 T. vinegar and oil dressing
 $\frac{1}{2}$ c canned pears
1 c low fat milk
coffee

Snack

1 c low fat milk
-3 sugar cookies (AHA)

M.F.
12/27/78

MONDAY

Breakfast

1 medium banana
1 c cornflakes
1 tsp. sugar
1 c low fat milk
coffee

Lunch

tomato soup, canned
4 saltines
tuna fish salad (2 oz.) sandwich
 $\frac{1}{2}$ c celery and carrot sticks
1 medium apple
1 c low fat milk
coffee

Dinner

5 oz. baked ham
 $\frac{1}{2}$ c candied sweet potatoes with margarine
 $\frac{1}{2}$ c green beans with margarine
 $\frac{1}{2}$ c coleslaw with pineapple cubes
1 slice bread
1 tsp. margarine
 $\frac{1}{2}$ c lemon pudding
coffee

Snack

2 oz. peanuts

M.F.
12/27/78

TUESDAY

Breakfast

½ grapefruit
1 c oatmeal
2 tsp. sugar
1 c low fat milk
coffee

Lunch

chicken sandwich (2 oz.)
2 T. mayonnaise
1 2x2 square jellied fruit salad
½ c canned apricots
1 c low fat milk
coffee

Dinner

4 oz. meat balls
½ c tomato sauce
1 c spaghetti
2 c tossed salad
2 T. vinegar and oil dressing
1 bread stick
1 tsp. margarine
1 cantaloupe wedge (¼ of 6" melon)
3½ oz. red wine
coffee

Snack

1 small apple

M.F.
12/27/78

WEDNESDAY

Breakfast

- 1 c orange juice
- 3 slices French toast I (AHA)
($\frac{1}{2}$ egg yolk)
- 2 T. pancake syrup
- 2 T. margarine
- 1 c low fat milk

Lunch

- 4 oz. pot roast of beef
- 1 medium baked potato
- 1 T. margarine
- $\frac{1}{2}$ c sliced carrots with margarine
- 1 3"x2"x2" hard roll
- 1 T. margarine
- $\frac{1}{2}$ c fruit cocktail
- coffee

Dinner

- 3 oz. pork chop
- $\frac{1}{2}$ c broccoli with margarine
- $\frac{1}{2}$ c corn with margarine
- $\frac{1}{2}$ c apple crisp
- 1 c low fat milk
- coffee

M.F.
12/27/78

THURSDAY

Breakfast

½ c stewed prunes
1 c rice krispies
1 tsp. sugar
1 c low fat milk
coffee

Lunch

2 oz. Swiss cheese sandwich with lettuce
2 tsp. margarine
2 c tossed salad
2 T. French dressing
1/3 c sherbet
coffee

Dinner

3 oz. Good and Easy Sauerbraten (AHA)
½ c noodles with margarine
½ c beets
½ c gravy
1 slice bread
1 tsp. margarine
1 c sliced oranges and bananas
1 c low fat milk
coffee

Snack

1 small bunch grapes

M.F.
12/27/78

FRIDAY

Breakfast

½ c apple juice
2 slices toast
2 tsp. margarine
1 c low fat milk
coffee

Lunch

3 oz. ham salad
½ c potato salad
1 small roll
1 tsp. margarine
1 small tomato, sliced
1 T. salad dressing
½ c gelatin dessert
1 c low fat milk

Dinner

4 oz. baked fish with 1 T. margarine
½ c parsleyed rice with margarine
½ c peas with margarine
½ c Waldorf salad
1 slice bread
1 tsp. margarine
1/6 of 9" blueberry pie
coffee

M.F.
12/27/78

SATURDAY

Breakfast

½ c grapefruit juice
2 slices applesauce toast (AHA)
1 c low fat milk
coffee

Lunch

1 c vegetable soup
4 saltines
3 oz. roast beef on
1 bun
2 tsp. margarine
1 medium pear
1 c low fat milk
coffee

Dinner

Chili con carne (1/3 AHA recipe - 4 oz. meat)
1 piece French bread, 3x3x1½
1 T. margarine
2 c tossed salad
2 T Italian dressing
½ c canned peaches

Snack

12 oz. beer
6 pretzels, 3" diameter

M.F.
12/27/78

	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	NF	PF	ALC.	FE
III. Baked Goods & Desserts (Con't)										
c. apple pie, 1 X model, Crust w/ margarine; filling w/ margarine.	409	3.9	18.68	58.4	0.0	3.60	11.91	2.48	0.0	--
d. 3 ea. pancakes from incomplete mix, w/ salad oil (unkn) egg subst, & low fat milk, 4 " diam.	341	14.4	12.88	56.0	4.0	0.65	1.67	1.62	0.0	--
e. 1 ea. muffin, bran, w/ egg subst, low fat milk & marg. (unkn brand, home)	61	1.6	2.11	8.7	0.6	0.45	1.11	0.50	0.0	--
f. 1 SV cornbread, incomplete mix w/ salad oil, egg subst. low fat milk, 3" square	184	4.2	6.91	26.1	0.7	0.91	3.02	2.89	0.0	--
g. 2 sl French toast, egg subst ckd in marg. (unkn, home)	349	14.4	16.96	34.4	3.8	2.97	7.40	6.05	0.0	--
Average	233	6.0	10.03	32.0	1.6	1.55	4.54	2.45	0.0	--
7. Mar. oils & other fats (Phase II) Average of										
a. oil										
1. Safflower, 1 TB	124	0.0	14.0	0.0	0.0	1.32	1.75	10.33	0.0	0.0
2. Sunflower	124	0.0	14.0	0.0	0.0	1.44	2.93	8.93	0.0	0.0
3. Corn	124	0.0	14.0	0.0	0.0	1.78	3.46	8.15	0.0	0.0
4. Soybean, p. Hyd.	124	0.0	14.0	0.0	0.0	1.82	6.58	5.60	0.0	0.0
5. Soy/Corn 9% blend	124	0.0	14.0	0.0	0.0	2.26	3.17	7.96	0.0	0.0
b. Marg. P/S 2-2.5, 1 TB	100	0.1	11.34	0.0	0.0	2.08	4.10	5.02	0.0	0.0
c. . avg of other fats from 3-C	65	1.0	6.30	1.6	2.4	1.01	2.03	3.01	0.0	0.1
Average	112	0.2	12.52	0.2	0.3	1.67	3.43	7.00	0.0	0.0

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
XI. Vegetables (Con't)										
e. ½ cp green beans, (62.5 gm)	16	1.0	0.06	3.6	0.0	0.00	0.00	0.00	0.0	0.4
f. ½ cp winter squash (100.5 gm)	39	1.1	0.31	9.4	0.0	0.00	0.00	0.00	0.0	0.5
g. ½ cp onions (105 gm)	330	1.3	0.11	6.8	0.0	0.00	0.00	0.00	0.0	0.4
h. ½ cp tomato sauce w/o fat (136 gm)	49	1.8	0.18	9.9	0.0	0.00	0.00	0.00	0.0	0.7
i. ½ cp cabbage, raw (27.5 gm)	7	0.4	0.06	1.5	0.0	0.00	0.00	0.00	0.0	0.1
j. ½ cp turnip, ckd (77.5)gm)	18	0.6	0.16	3.8	0.0	0.00	0.00	0.00	0.0	0.3
k. 1 cp T-salad	13	0.9	0.10	2.9	0.0	0.00	0.00	0.00	0.0	0.5
l. 1 tomato, raw, 3" diam. (200 gm)	44	2.2	0.40	5.4	0.0	0.00	0.00	0.00	0.0	1.0
Average	24	1.4	0.18	4.8	0.0	0.00	0.00	0.00	0.0	0.6
XII. Alcoholic Beverages										
a. Beer, 12 oz, reg. (360 gm)	151	1.1	0.00	13.7	0.0	0.00	0.00	0.00	13.0	0.0
b. Wine, 3½ oz, dry, table (105 gm)	89	0.1	0.00	4.4	0.0	0.00	0.00	0.00	10.4	0.4
c. Whiskey, 1½ oz, (45 gm)	112	0.0	0.00	0.0	0.0	0.00	0.00	0.00	16.2	0.0
Average	117	0.4	0.00	6.0	0.0	0.00	0.00	0.00	13.2	0.1
XIII. Baked Goods & Desserts (Fat Modified)										
a. 1 SV layer cake, 1/16 of 8" white cake from scratch, salad oil, low fat milk, egg whites (56036)	196	2.4	7.38	30.2	2.0	1.23	3.36	2.76	0.0	--
b. 2 oatmeal cookies, homemade w/ marg. (unkn home) & egg	91	1.2	5.26	10.0	0.2	1.02	3.30	0.84	0.0	--

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
X Con't										
i. ½ cp apple juice unswt,	56	0.1	0.00	14.3	0.0	0.00	0.00	0.00	0.0	0.7
j. 10 grapes (53 gm)	36	0.3	0.16	9.2	0.0	0.00	0.00	0.00	0.0	0.2
k. ½ grapefruit, med (100 gm)	41	0.5	0.10	10.6	0.0	0.00	0.00	0.00	0.0	0.4
l. 1 nectarine med, 2½" diam. (150 gm)	96	0.9	0.00	25.7	0.0	0.00	0.00	0.00	0.0	0.8
m. 1 orange, med 2-¾" diam. (116 gm)	57	1.2	0.23	14.2	0.0	0.00	0.00	0.00	0.0	0.5
n. 1 sl watermelon, 4" arc, 8" radius (426 gm)	111	2.1	0.85	27.3	0.0	0.00	0.00	0.00	0.0	2.1
o. 1 plum, 2-1/8" diam, (60 gm)	45	0.5	0.12	11.8	0.0	0.00	0.00	0.00	0.0	0.3
p. 1 cp strawberries unswt (149 gm)	55	1.0	0.75	12.5	0.0	0.00	0.00	0.00	0.0	1.5
q. 1 pear, 2½" diam, (182 gm)	111	1.3	0.73	27.8	0.0	0.00	0.00	0.00	0.0	0.5
r. 1 peach, 2-¾" diam (296 gm)	112	1.8	0.30	28.7	0.0	0.00	0.00	0.00	0.0	1.5
s. 2 sl P/A, cnd in own juice (200 gm)	104	0.8	0.40	27.4	0.0	0.00	0.00	0.00	0.0	0.8
t. ½ cp prunes ckd, unswt (135 gm)	161	1.4	0.41	42.4	0.0	0.00	0.00	0.00	0.0	2.4
u. 1 TB raisins (9.45 gm)	27	0.2	0.01	7.3	0.0	0.00	0.00	0.00	0.0	0.3
Average	78	0.9	0.27	19.8	0.0	0.00	0.00	0.00	0.0	0.8
Vegetables										
a. ½ cp broccoli, ckd (77.5 gm)	20	2.2	0.23	3.6	0.0	0.00	0.00	0.00	0.0	0.5
b. ½ cp carrots (72.5 gm)	22	0.7	0.15	5.1	0.0	0.00	0.00	0.00	0.0	0.4
c. ½ cp greens (72.5 gm)	14	1.6	0.15	2.4	0.0	0.00	0.00	0.00	0.0	0.7
d. ½ cp spinach (90 gm)	21	2.7	0.27	3.3	0.0	0.00	0.00	0.00	0.0	1.9

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
IX. Con't										
j. ½ cp oatmeal (120 gm)	66	2.4	0.12	11.6	0.0	0.20	0.42	0.49	0.0	0.7
k. 1 cp spoon size shredded wheat (50 gm)	177	5.0	1.00	40.0	0.0	0.17	0.16	0.53	0.0	1.8
l. 2 saltines (4 crax), (10.5 gm)	45	0.9	1.26	7.5	0.5	0.50	0.56	0.15	0.0	0.1
m. 1 cp soup, cream of mush., with water (120 gm)	133	2.3	9.6	10.1	1.2	2.5	1.73	4.32	0.0	0.4
n. 1 cp split pea w/H ₂ O, (240 gm)	161	7.7	5.52	20.9	5.3	1.46	2.06	1.73	0.0	2.2
o. 1 cp tomato w/ H ₂ O, (180 gm)	86	1.9	2.52	15.2	0.0	1.51	0.90	0.11	0.0	0.7
p. 1 cp vegetarian with water, (240 gm)	67	3.8	2.64	7.0	2.4	0.60	0.67	1.2	0.0	1.0
q. 1 cp bean with water	161	7.7	5.52	20.9	5.3	1.46	2.06	1.73	0.0	2.2
Average	116	4.0	1.90	20.9,	0.9	0.53	0.55	0.67	0.0	1.1
X. Fruits & Juices, cnd & fresh, dried, average of										
a. 1 apple, raw, med. (150 gm)	81	0.3	0.45	21.2	0.0	0.00	0.00	0.00	0.0	0.5
b. ½ cp apricots cnd, light syrup (123 gm)	106	0.7	0.12	27.1	0.0	0.00	0.00	0.00	0.0	0.4
c. 1 banana, 7" peeled (136 gm)	116	1.5	0.27	30.2	0.0	0.00	0.00	0.00	0.0	1.0
d. 1 cp cantaloupe (162 gm)	49	1.1	0.16	12.2	0.0	0.00	0.00	0.00	0.0	0.6
e. 10 cherries, fresh (67 gm)	47	0.9	0.20	11.7	0.0	0.00	0.00	0.00	0.0	0.3
f. ½ cp fruit salad, cnd, swt. (128 gm)	97	0.5	0.13	25.2	0.0	0.00	0.00	0.00	0.00	0.5
g. ½ cp orange juice	54	0.8	0.12	12.8	0.0	0.00	0.00	0.00	0.0	0.1
h. ½ cp P/A juice, cnd, unswt	66	0.5	0.12	16.2	0.0	0.00	0.00	0.00	0.0	0.4

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
VIII. Con't										
6. French dressing, comm., (avg. clear & creamy) (15.5 gm)	75	0.1	7.66	1.9	0.0	1.16	1.77	4.42	0.0	0.0
7. Italian dressing, comm. (15 gm)	83	0.0	9.0	1.0	0.0	1.36	2.08	5.18	0.0	0.0
8. 1000 Island (16 gm)	80	0.1	8.03	2.5	8.6	1.30	1.78	4.24	0.0	0.1
9. Mayo-type (15 gm)	65	0.2	6.35	2.2	7.5	1.22	1.67	3.98	0.00	0.0
10. English walnuts (8 gm)	52	1.2	5.12	1.3	0.0	0.56	0.79	3.34	0.0	0.2
11. Mixed nuts (12.8 gm)	76	2.8	6.80	2.6	0.0	1.00	4.31	1.47	0.0	0.5
c. Average of 1-11	65	1.0	6.30	1.6	2.4	1.01	2.03	3.01	0.0	0.1
Overall average (A-C)	97	0.4	10.55	0.6	0.8	1.69	5.37	3.36	0.0	0.0
X. Plain breads, cereals, starchy veg, soups - average of										
a. ½ cp lentils, ckd (125 gm)	144	10.0	0.38	25.0	0.0	0.00	0.00	0.00	0.0	2.1
b. ½ cp lima beans (85 gm)	84	5.1	0.09	16.2	0.0	0.00	0.00	0.00	0.0	1.4
c. ½ cp corn w. k. (83 gm)	66	2.5	0.42	15.6	0.0	0.05	0.09	0.22	0.0	0.7
d. ½ cp potato (79.5 gm)	52	1.5	0.08	11.5	0.0	0.00	0.00	0.00	0.0	0.4
e. 1 swt potato, baked 5" x 2" (146 gm)	166	2.5	0.58	38.4	0.0	0.00	0.00	0.00	0.0	1.0
f. 1 cp macaroni, spaghetti (130 gm)	192	5.3	0.65	39.1	0.0	0.10	0.08	0.29	0.0	1.4
g. 1 cp rice, white (2.05 gm)	223	4.1	0.21	49.6	0.0	0.06	0.06	0.08	0.0	1.8
h. 1 sl. bread, white (30 gm)	81	2.6	0.96	15.2	0.5	0.27	0.33	0.30	0.0	0.8
i. 1 sl bread, whole grain (25 gm)	61	2.6	0.75	11.9	0.4	0.16	0.18	0.28	0.0	0.6

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	
IV. Con't 30 gm,										
d. scallops	34	7.0	0.42	0.0	15.9	0.07	0.04	0.16	0.0	0.9
e. crabmeat	30	5.2	0.75	0.3	30.3	0.11	0.16	0.31	0.0	0.2
f. lobster	29	5.6	0.45	0.1	25.5	0.04	0.05	0.14	0.0	0.2
g. clams	29	4.7	0.75	0.6	18.9	0.14	0.14	0.16	0.0	0.0
Average 30 gm	34	5.4	1.05	0.4	21.3	0.19	0.22	0.36	0.0	0.5
V. Egg, whole, 1 med (50 gm)	82	6.5	5.75	0.5	252.0	1.70	2.27	0.69	0.0	1.2
VI. Low Fat milk, exact % fat unkn, 1 CP	142	10.1	4.80	14.4	13.9	2.88	1.39	0.19	0.0	0.2
VII. Low fat milk, 1%, 1 CP.	113	9.4	2.4	13.4	7.0	1.44	0.67	0.07	0.0	0.0
VIII. Oil, marg. & other fats composite										
a. oil (unkn salad oil) - 1 TB	124	0.0	14.00	0.0	0.0	1.82	6.58	5.60	0.0	0.0
b. margarine (unkn brand, home use) - 1 TB	101	0.1	11.34	0.1	0.0	2.24	7.50	1.46	0.00	0.0
c. other fats - 1 TB										
1. peanuts (9 gm)	62	2.4	4.38	1.9	0.0	0.84	2.02	1.31	0.0	0.2
2. peanut butter (16 gm)	94	4.0	8.10	3.0	0.0	1.55	3.72	2.43	0.0	0.3
3. avocado (9.38)	16	0.2	1.54	0.6	0.0	0.23	0.89	0.18	0.0	0.1
4. ripe olives, sliced (8.44 gm)	11	0.1	1.16	0.2	0.0	0.17	0.84	0.10	0.0	0.1
5. salad dressing, comm., mayo, (14gm)	101	0.2	11.19	0.3	9.8	1.68	2.51	6.45	0.0	0.1

FOOD	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	FF	ALCO.	
Beef, lamb, Pork composite (trimmed of sep. fat), average of 30 gm										
a. beef rump, choice	62	8.7	2.79	0.0	27.3	0.98	0.95	0.16	0.0	1.1
b. beef round, choice	57	9.4	1.83	0.0	27.3	0.81	0.82	0.14	0.0	1.1
c. beef sirloin - 1 lb	65	9.2	2.85	0.0	27.3	0.99	0.98	0.16	0.0	1.1
d. beef, ground, lean (unkn fat content other than lean)	98	7.8	7.17	0.0	28.2	2.85	3.25	0.32	0.0	1.0
e. pork loin, fresh ham, whole	76	8.8	4.26	0.0	26.4	1.41	1.93	0.45	0.0	1.1
f. pork leg, smoked ham	56	7.6	2.64	0.0	26.4	0.89	1.22	0.29	0.0	1.0
g. pork leg, smoked ham, canned	56	7.6	2.64	0.0	26.4	0.89	1.22	0.29	0.0	1.0
h. lamb leg	62	8.6	2.10	0.0	29.7	0.89	0.81	0.13	0.0	0.7
i. lamb loin	62	8.6	2.10	0.0	29.7	0.89	0.81	0.13	0.0	0.7
Average	66	8.5	3.15	0.0	27.6	1.18	1.33	0.23	0.0	1.0
I. a. Chix - w/o skin	49	8.5	1.38	0.0	24.7	0.36	0.36	0.33	0.0	0.5
b. Turkey - w/o skin	57	9.5	1.83	0.0	26.7	0.52	0.46	0.49	0.0	0.5
c. Veal, 6% fat	62	10.3	2.01	0.0	29.7	0.61	0.57	0.20	0.0	1.3
Average	56	9.4	1.74	0.0	27.0	0.50	0.46	0.34	0.0	0.8
I. Shrimp, ckd, 30 gm.	35	7.3	0.33	0.2	45.0	0.04	0.04	0.13	0.0	0.9
V. a. low fat fish (2%) 30 gm	27	5.9	0.27	0.0	19.8	0.06	0.05	0.11	0.0	0.2
b. med fat fish (12%)	65	6.8	4.02	0.0	25.2	0.68	0.95	1.37	0.0	0.3
c. oysters	23	2.6	0.66	1.5	13.5	0.23	0.13	0.25	0.0	1.7

Table III Summary Sheet - Food Composites for Phase II Diet

FOOD	DATA SOURCE	AMT	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
Meat, Fish Poultry	Table I	30 g	57	8.0	2.35	0.1	26.4	0.80	0.88	0.28	0	0.9
(Z Fat Unkn.) Low Fat Milk	Composite VI	1 CP	142	10.1	4.80	14.4	13.9	2.88	1.39	0.19	0	0.2
(1% Fat) Low Fat Milk ✓	Composite VII	1 CP	113	9.4	2.4	13.4	7.0	1.44	0.67	0.07	0	0.00
Eggs	Composite V	1/7	12	0.9	0.82	0.1	36.0	0.24	0.32	0.10	0	0.2
Fats, Oils	Composite XIV	1 TB	112	0.2	12.52	0.2	0.3	1.67	3.43	7.00	0	0.00
Breads, Cereals, St. Veg, Soups	Composite IX	1 SV	116	4.0	1.90	20.9	0.9	0.53	0.55	0.67	0	1.1
Fruits	Composite X	1 SV	78	0.9	0.27	19.8	0.0	0.00	0.00	0.00	0	0.8
Vegetables	Composite XI	1 SV	24	1.4	0.18	4.8	0.0	0.00	0.00	0.00	0	0.6
Alcoholic Beverages	Composite XII	1 SV	117	0.4	0.00	6.0	0.0	0.00	0.00	0.00	13.2	0.1
Baked Goods & Desserts (fat modified)	Composite XIII	1 SV	233	6.0	10.03	32.0	1.6	1.55	4.54	2.45	0	--
<1% fat milk	MRFIT Food Table	1 cp	86	8.6	0.24	12.2	4.3	0.14	0.07	---	---	--

Table II Summary Sheet - Food Composites for Phase I Diet

FOOD	DATA SOURCE	AMT	CALORIES	PROTEIN	FAT	CHO.	CHOL	SF	MF	PF	ALC.	FE
Meat, Fish Poultry	Table I	30 gram	57	8.0	2.35	0.1	26.4	0.80	0.88	0.28	0	0.9
(Exact % Unkn.)												
Low Fat Milk	Composite VI	1 CP	142	10.1	4.80	14.4	13.9	2.88	1.39	0.19	0	0.2
Eggs	Composite V	1/7	12	0.9	0.82	0.1	36.0	0.24	0.32	0.10	0	0.2
Fats, Oils	Composite VIII	1 TB	97	0.4	10.55	00.6	0.8	1.69	5.37	3.36	0	0.0
Breads, Cereals St. Vegetables, Soups	Composite IX	1 SV	116	4.0	1.90	20.9	0.9	0.53	0.55	0.67	0	1.1
Fruits	Composite X	1 SV	78	0.9	0.27	19.8	0.0	0.00	0.00	0.00	0	0.8
Vegetables	Composite XI	1 SV	24	1.4	0.18	4.8	0.0	0.00	0.00	0.00	0	0.6
Alcoholic Beverages	Composite XII	1 SV	117	0.4	0.00	6.0	0.0	0.00	0.00	0.00	13.2	0.1
Baked Goods & Desserts (fat modified)	Composite XIII	1 SV	233	6.0	10.03	32.0	1.6	1.55	4.54	2.45	0	—

Table I Weighting Factor Analysis

FOOD	FOOD COMPOSITE	AMT G	WEIGHTING FACTOR	CALORIES	PROTEIN	FAT	CHO.	CHOL.	SF	MF	PF	FE
Beef, Lamb, Pork	p. 1-2 I	30 X	7.75	512	65.9	24.41	0.0	213.9	9.15	10.31	1.78	7.8
Poultry, Veal	II	30 X	3.0	168	28.2	5.22	0.0	81	1.50	1.38	1.02	2.4
Fish, Shellfish	IV	30 X	3.0	102	16.2	3.15	1.2	63.9	0.57	0.66	1.08	1.5
Shrimp	III	30 X	0.25	9	1.8	0.08	0.1	11.3	0.01	0.01	0.03	0.2
			14	791	112.1	32.86	1.3	370.1	11.23	12.36	3.91	11.9
				57	8.0	2.35	0.1	26.4	0.80	0.88	0.28	0.9

Weighted \bar{X} 30 g

PART III

PART III

"HOW TO" SESSIONS

Purpose and Format

- PURPOSE: These Sessions are designed to provide an opportunity for physicians attending the Annual Scientific Sessions of the American Heart Association to participate in discussions of widely used techniques in Cardiology and new therapeutic interventions with noted authorities in an informal setting. These Sessions were initiated at the Annual Meeting of the American Heart Association in 1970 and were enthusiastically received by the participants.
- FORMAT: Every attempt should be made for maximal audience participation. The audience should be encouraged to raise questions and participate actively following the discussion.
- ATTENDANCE: As attendance at the Sessions is limited to 25, admission is by ticket only and panel members are requested to respect this limitation. The small audience insures an active inter-change among all attending. This is the main purpose of the Session.
- PROJECTION EQUIPMENT: These Sessions differ from the Cardiovascular Conferences and equipment for projecting slides and illustrated material will be provided when arrangements are made in advance. However, a didactic presentation is not the purpose and material projected should be used for discussion purposes and for audience participation. In addition to projection equipment, a blackboard will be made available in each room.
- PRESS COVERAGE: Because of the informal nature of these Sessions, they are closed to the press. The committee asks the panel participants not to have press representatives attend.

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CARDIOVASCULAR CONFERENCES

Purposes and Format

- PURPOSE: These conferences provide an opportunity for physicians and scientists attending the annual Scientific Sessions to discuss daily problems with noted authorities in an informal setting and have been enthusiastically received by the audience.
- FORMAT: Every attempt should be made for maximum audience participation. It is suggested that the moderator outline the material to be covered in short opening remarks, no longer than five minutes by each panelist. The audience should then be encouraged to raise questions and actively participate in the ensuing discussion.
- ATTENDANCE: Based on past experience, attendance is limited. Admission is by ticket only and panel members are requested to respect this limitation. A small audience insures free discussion from the floor, which is the main purpose of these conferences.
- PROJECTION EQUIPMENT: The purpose of the conference is to provide group discussion. Based on past experience, the Committee planning these conferences believes that the projection of slides stifles the informal aspect of the conferences and does not significantly contribute to group discussion. Because of these reasons, no projection equipment will be provided. A blackboard will be available in each room.
- PRESS COVERAGE: Because of the informal nature of these conferences, they are closed to the press. The Committee asks that panel participants do not invite press representatives to attend their conferences.

CARDIOVASCULAR CONFERENCES

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Address _____

Alternate Name _____

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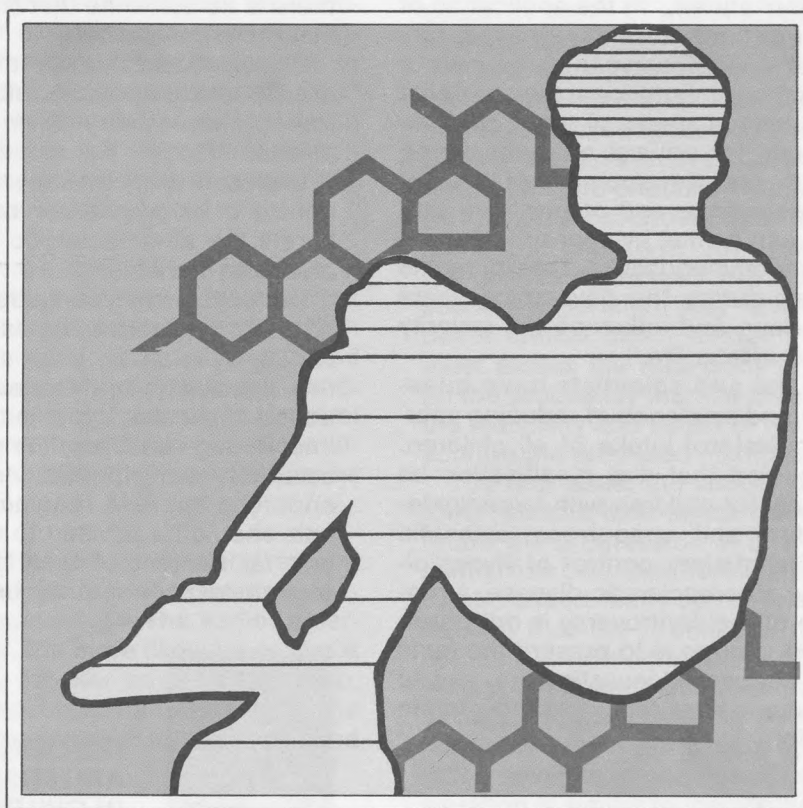
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The Value and Safety of Diet Modification To Control Hyperlipidemia*

In Childhood and Adolescence
A Statement for Physicians

A statement for physicians and
other health professionals
prepared by an ad hoc
committee of the Steering
Committee for Medical and
Community Program of the
American Heart Association.



Hyperlipidemia clearly is a risk factor for arteriosclerotic disease in adults and very likely contributes to atherogenesis in children. The American Heart Association recommends that children with elevated plasma cholesterol or triglyceride be placed on an appropriate diet in order to reduce their risk of hyperlipidemia and possibly to reduce their risk of arteriosclerotic disease when they become adults. Although the evidence does not yet support the recommendation that cholesterol and saturated fat should be reduced in the diet of all children, the public should be advised that such modification appears safe and very likely to be beneficial.

Measuring plasma lipids of all children in order to prevent arteriosclerotic disease probably is not cost effective. However, children of high risk families should be examined for hyperlipidemia.

*Reprinted from *Circulation*, (58:381A, 1978)

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INTRODUCTION

The American Heart Association (AHA), after careful evaluation of the scientific evidence, took the position in 1965¹ that the general public, including children, should be advised to modify the fat and cholesterol content of its diet. It was hoped that such dietary modification would lead to reduced levels of plasma cholesterol, retard the development of atherosclerosis, and reduce the incidence of all forms of atherosclerotic disease.

This position has been reviewed periodically, and has generated considerable controversy primarily because unequivocal evidence that morbidity and mortality are reduced by diet modification is not yet available. The latest (1973) version of the AHA position² stated, "In the application of these recommendations to family groups, any change in the diet must preserve the principles of good nutrition. Although nutritional requirements differ during the various stages of the normal life cycle, the demands for optimal nutrition during periods of growth and development of infants, children and adolescents, and of pregnant and lactating women can be met by appropriate modifications of the recommendations. Dietary habits which are formed during the developing years may continue lifelong and influence the severity of atherosclerosis in later life."

Some physicians and scientists have questioned the safety and prudence of reducing saturated fat and cholesterol intake of all children. They have suggested that diet modification be recommended only for children with hyperlipidemia.³⁻⁷ Without firm and unequivocal scientific demonstration that dietary control of hyperlipidemia will reduce atherosclerotic disease, a reasoned resolution of the controversy is not possible. The best AHA can do is to present the facts agreed upon by concerned investigators, and to describe the issues where facts are incomplete and opinions differ.



THE RISK FACTORS FOR CORONARY HEART DISEASE IN ADULTS

Thirty years of intensive epidemiological research have identified six major risk factors for the atherosclerotic diseases and have implicated several others.⁸⁻¹¹ The established risk factors are age, sex, hypercholesterolemia, hypertension, cigarette smoking, and diabetes. Less certain risk factors are obesity, hypertriglyceridemia, personality type, and lack of physical activity. Recently, the concentrations of cholesterol carried in low density lipoprotein (LDL) and in high density lipoprotein (HDL) have been recognized as more specific risk factors (positive for LDL, negative for

HDL) than total plasma cholesterol concentration.¹²⁻¹⁵

The established risk factors may be causal agents, intervening variables, or secondary indicators of a more fundamental disturbance. In the context of prevention, the AHA is concerned primarily with those factors which are susceptible to manipulation, a consideration that excludes age and sex. Numerous studies have examined the effects of risk factor control on incidence of atherosclerotic disease.

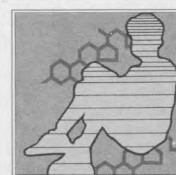
There is convincing evidence that elimination of cigarette smoking leads to reduced risk for coronary heart disease (CHD).¹⁶

Drug treatment of hypertension reduces risk of stroke and congestive heart failure, but as yet there is no evidence that it reduces the impact of hypertension on CHD.¹⁷⁻¹⁸

Unequivocal evidence is lacking that reduction of LDL cholesterol in adults by diet or drugs will lower risk of any form of atherosclerotic disease.¹⁹⁻²⁰

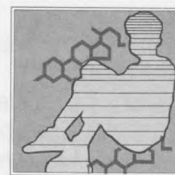
Diet and drug treatment of diabetics allows control of blood glucose, but appears not to ameliorate the atherosclerotic complications associated with diabetes.²¹

Reduction of hyperlipidemia may reduce risk, but not to a degree that can be detected unequivocally by relatively small clinical trials. In view of this possibility, and because most current evidence implicates hyperlipidemia as an important intervening variable in the progression of atherosclerosis, many physicians and scientists have endorsed the AHA recommendation that individuals should be advised to modify the cholesterol and fat content of their diets so as to reduce plasma lipid concentrations.²²



THE ORIGIN OF ATHEROSCLEROSIS IN CHILDHOOD

It is generally accepted that atherosclerosis begins in childhood and undergoes rapid progression in adolescence and young adulthood, even though the serious clinical manifestations do not appear until middle age or later.²³ Many questions remain about the causes of fat deposition in children's arteries and whether these fat deposits lead directly to advanced arterial lesions. However, studies of U.S. men who died from accidental causes have shown that clinically significant lesions begin to appear in the third decade of life, shortly after the childhood years.²⁴⁻²⁷ This observation is one reason why many physicians and scientists believe that prevention, to be effective, must begin in the teenage years and possibly earlier.



RELATIONSHIP OF RISK FACTORS AMONG CHILDREN TO THOSE IN ADULTS AND ADULT DISEASE

Plasma Lipids

Surveys in the past 5 years have established unequivocally that U.S. children have higher plasma lipid concentrations than do children of other populations in which adult atherosclerotic disease is less frequent.²⁸⁻³⁹ About 5% of 5-18 year old U.S. children have plasma cholesterol levels greater than 200-220 mg/dl.^{31,35-37} Furthermore, children maintain a similar rank order through several years of childhood. Most individuals who have one of the genetic hyperlipidemias, particularly familial hypercholesterolemia, can be identified in childhood.⁴⁰⁻⁴³ Investigations now in progress will determine more precisely the degree to which plasma lipid levels in childhood predict those in adulthood. Meanwhile, many hyperlipidemic children probably will become hyperlipidemic adults, and therefore will be at greater risk of adult atherosclerotic disease.

Blood Pressure

The evidence pertaining to blood pressure is less complete than that for hyperlipidemia, but many children have blood pressures that are high even by adult standards.^{31,44} Rank order of blood pressure is maintained less well through childhood than is rank order of plasma cholesterol concentration. As with hyperlipidemia, these individuals are likely to become hypertensive as adults.

Smoking

The prevalence of cigarette smoking among teenagers is high and the rate of cigarette smoking among girls is increasing.⁴⁵ The earlier a person begins to smoke, the more likely he or she is to become a heavy smoker as an adult. Also, smoking may be especially harmful during the early stages of atherogenesis in adolescence and young adulthood.

Diabetes and Obesity

Juvenile diabetics are prone to precocious atherosclerotic diseases as are adult onset diabetics. Obesity predisposes to atherosclerosis principally through its contribution to hypertension and diabetes. Obesity in childhood is associated with adult obesity and thereby to adult atherosclerotic disease.⁴⁶ The long-term effects of infant obesity remain unclear.

Other Risk Factors

Much less is known about the relationship of the other suspected risk factors (lack of physical activity, personality type) in children to similar conditions in adults. Both patterns of physical activity and of personal and social behavior are probably established in childhood, but little more can be said until their relationship to adult disease is clarified.



HYPERLIPIDEMIA IN CHILDREN AS A CONTRIBUTOR TO ATHEROSCLEROSIS

Because hyperlipidemia is the most consistent and most frequent risk factor for atherosclerotic disease (other than age and sex), it has received the most attention as a point of attack for the prevention of atherosclerosis. This emphasis remains despite the reservation, described previously, that reduction in morbidity and mortality by lowering plasma lipid concentrations has not been proven conclusively. The relatively small clinical trials that have been completed may have failed to detect a substantial effect because they began in adults in whom atherosclerosis already was well advanced.

Much epidemiological and pathological evidence in humans and much evidence from experimental animals suggests that maintenance of low plasma cholesterol levels beginning in childhood would be more effective in retarding the progression of atherosclerosis than reducing levels beginning in the 4th or 5th decades. A controlled test of this hypothesis has not been done, and such an experiment may never be feasible. Without a critical direct test of the hypothesis, we must assess the total body of evidence bearing on the probability that the premise is true; and on the feasibility and safety of measures required to achieve the proposed manipulation.

The first issue, the validity of the premise that lower plasma lipid levels from childhood would retard the progression of atherosclerosis significantly, has been discussed already and little more can be added. Many currently active investigators believe that the premise is probably true, but opinions on the strength of the probability vary widely. The American public must share with physicians and scientists the burden of this uncertainty, and must participate in making decisions in the absence of conclusive scientific proof. The situation is similar to many others in everyday life in which total knowledge and absolute certainty are not attainable. The two issues that remain are (1) the feasibility and (2) the safety of controlling plasma lipids by diet modification.

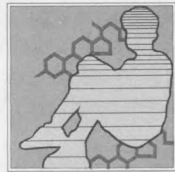


FEASIBILITY OF CONTROLLING HYPERLIPIDEMIA IN CHILDREN BY DIET MODIFICATION

The feasibility of maintaining low plasma lipid levels in children by diet modification has received much less attention than the related problem in adults, but there is sufficient evidence to provide a reasonably certain answer. There is a

strong genetic component in control of plasma cholesterol concentration which is expressed early in life.^{40,41,47,49} However, reduced cholesterol intake combined with decreased total fat and saturated fat lower plasma cholesterol concentrations in many children, even in individuals with genetic hyperlipidemia.^{50-55,43}

Based on the epidemiological risk factor data, a lifelong serum cholesterol differential of only 10 mg/dl could be expected to have a substantial effect on risk of coronary heart disease.⁵⁶ The 1973 AHA statement² on diet and coronary heart disease recommended that the adult diet should contain less than 300 mgs. of cholesterol per day, that not more than 35% of the calories should be derived from fat, and that saturated fat should provide no more than 10% of total calories. Such a diet requires careful food shopping, careful preparation, and some reorientation of food preferences for most U.S. adults and children, but it is feasible and does not require highly specialized food preparations which are expensive and not widely available. The National Diet Heart Study⁵⁷ showed that such a diet would reduce plasma cholesterol levels in a large group of free living U.S. adults. A similar diet consumed by children should produce comparable effects.



SAFETY OF MODIFIED DIETS IN CHILDHOOD

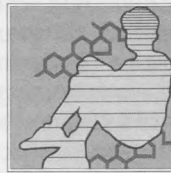
The third issue is the safety of the dietary regimens proposed to achieve a reduction in plasma cholesterol. Currently, about 80% of U.S. infants are fed commercially prepared formulas containing very little cholesterol and a high proportion of polyunsaturated fatty acids.⁴ No immediate or long-term deleterious effects of these formulas due to differences in fat or cholesterol content of infant formula have been conclusively demonstrated, but there is conflicting evidence and the issues remain under study.^{54,58-65}

Gallstones have been reported to occur more frequently in adults who consume large amounts of polyunsaturated fats and plant sterols than in those who consume predominantly saturated fats and cholesterol.⁶⁶ We do not yet know whether the diet recommended for adults, if introduced in childhood, would increase the incidence of gallstones in later life. Further study of this potential hazard is needed.

Low cholesterol intake in infancy has been suggested as possibly retarding myelination of the central nervous system. Children from areas with generations of low cholesterol diets have no distinctive neurologic disorders, but whether more subtle neurologic differences exist has never been assessed. Children with the very low plasma

cholesterol values of familial hypobetalipoproteinemia show normal neurologic development.⁴³

Despite the possibility of unknown potential hazards, there seem to be no demonstrated major hazards involved in consumption of the AHA diet by children. However, as these diet recommendations are adopted, new food products whose safety has yet to be determined will be developed. For example, soybean meat substitutes and egg substitutes currently available contain relatively large amounts of sodium which may raise blood pressure in some subjects. Investigation into the potential benefits and risks of modified diets in childhood is encouraged.



SCREENING FOR AND DIAGNOSIS OF ACQUIRED AND GENETIC HYPERLIPIDEMIAS

Although opinion is widely divided about the desirability of recommending the AHA fat-modified diet for all U.S. children, opinion is nearly unanimous that children falling in the top decile of their age and sex distribution of plasma cholesterol and triglyceride levels should be treated.^{43,67} The recommendation to treat such individuals is based on the association between hyperlipidemia in adults and risk of disease, and the presumption that such levels in childhood also predict a high degree of risk. The problem then becomes how to detect hyperlipidemia in children and how to establish a specific diagnosis when detected.

Screening entire populations for hyperlipidemia probably is not feasible with present methods. However, children from families with hyperlipidemia or hypertension in one or both parents, or from families with myocardial infarction, stroke, or peripheral vascular disease in parents or grandparents before age 50, have a much higher prevalence of risk factors and should be examined carefully. Analysis of plasma lipids of such children should include at least 2, preferably 3, repetitive samplings. Once the hyperlipidemic child is identified, the physician should realize that the hyperlipidemia may be due to diet excess, to other diseases such as hypothyroidism or the nephrotic syndrome, or to an interaction between diet and genetic factors. The physician should differentiate between acquired hyperlipidemia and one of the familial (and presumably genetic) hyperlipidemias.

Acquired hypertriglyceridemia may be related to estrogen containing oral contraceptives, alcohol, or excessive calories and carbohydrates, and often will respond to modification of these factors. For the child with hypercholesterolemia not secondary to some disease state, and possibly related to excess cholesterol and saturated fat intake, cholesterol should be reduced to less than

300 mgs/day and saturated fat intake should be reduced to achieve a P/S ratio of 1:1.

Diet induced hypercholesterolemia usually will respond to reduction in excess cholesterol and saturated fat intake, but long-term nutrition modification in children is not easily accomplished, and some children will not respond. Recommendations for management of the genetic hyperlipidemias, and of hyperlipidemias not easily categorized but not responsive to the above diet, are outlined below.

1. Familial hypercholesterolemia

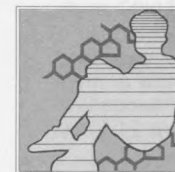
From age 1 to 10, approximately one-third of these children will attain normal levels of total and LDL cholesterol with dietary modification which includes the following: reduce fat to 25-30% of total calories, substitute polyunsaturates for saturates to reach a P/S ratio of 1:1, and limit dietary cholesterol to less than 200 mgs/day adjusted downward for younger children. Above age 10, this diet alone may reduce total or LDL cholesterol by 10 to 20%, but it will not cause great reduction in plasma cholesterol levels.⁴³ Therapy with bile acid binding resins may be necessary in such children to attain adequate control.

2. Familial hypertriglyceridemia

Most children with familial hypertriglyceridemia will attain normal triglyceride levels with moderate reduction in body weight in relation to the growth curve.⁴³ Further reductions in plasma triglyceride in the lean child may be achieved by reducing carbohydrates and saturated fat intake. Drug therapy is not needed.

3. Hypercholesterolemia and hypertriglyceridemia

In familial combined hyperlipidemia, either cholesterol or triglyceride or both may be elevated in affected children. The dietary and other intervention measures described above are indicated. In the presence of obesity, calories also must be restricted.



SUMMARY

Hyperlipidemia clearly is a risk factor for atherosclerotic disease in adults and very likely contributes to atherogenesis in children. The American Heart Association recommends that children with elevated plasma cholesterol or triglyceride be placed on an appropriate diet in order to reduce their risk of hyperlipidemia and possibly to reduce their risk of atherosclerotic disease when they become adults. Although the evidence does not yet support the recommendation that cholesterol and saturated fat should be reduced in the diet of all children, the public should be advised

that such modification appears safe and very likely to be beneficial.

Measuring plasma lipids of all children in order to prevent atherosclerotic disease probably is not cost effective. However, children of high risk families should be examined for hyperlipidemia.

REFERENCES

1. American Heart Association. Committee on Nutrition *Diet and Coronary Heart Disease*. New York, American Heart Association, 1965.
2. American Heart Association. Committee on Nutrition *Diet and Coronary Heart Disease*. New York, American Heart Association, 1973.
3. Davidson M: Obesity and the question of cholesterol reduction in infancy and childhood. *Pediatr Ann* 4:101-111, 1975.
4. Fomon SJ: *Infant Nutrition*. 2nd ed. Philadelphia, WB Saunders Co, 1974, 575 pp.
5. Mitchell S, Blount SG Jr, Blumenthal S, Jesse MJ, Weidman, WH: The pediatrician and atherosclerosis. *Pediatrics* 49:165-168, 1972.
6. Mitchell SC, Jesse MJ: Risk factors of coronary heart disease — Their genesis and pediatric implications. *Am J Cardiol* 31:588-590, 1973.
7. Schubert WK: Fat nutrition and diet in childhood. *Am J Cardiol* 31:581-587, 1973.
8. Keys A (ed): *Coronary Heart Disease in Seven Countries*. (AHA Monograph 29). *Circulation* 41 (suppl 1):1-1 — 1-211, 1970.
9. Stamler J, Berkson DM, Lindberg HA: Risk factors: Their role in the etiology and pathogenesis of the atherosclerotic diseases, in Wissler RW, Geer JC (eds): *The Pathogenesis of Atherosclerosis*. Baltimore, Williams and Wilkins, 1972, pp 41-119.
10. Primary prevention of the atherosclerotic diseases, Atherosclerosis and Epidemiology Study Groups of the Inter-society Commission for Heart Disease Resources. *Circulation* 42:A55-A95, 1970.
11. Kannel WB, McGee D, Gordon T: A general cardiovascular risk profile: The Framingham study. *Am J Cardiol* 38:46-51, 1976.
12. Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, Kagan A, Zukel WJ: HDL cholesterol and other lipids in coronary heart disease. The Cooperative Lipoprotein Phenotyping Study. *Circulation* 55:767-772, 1977.
13. Rhoads GG, Gulbrandsen CL, Kagan A: Serum lipoproteins and coronary heart disease in a population study of Hawaii Japanese men. *N Engl J Med* 294:293-298, 1976.
14. Medalie JH, Kahn HA, Neufeld HN, Riss E, Goldbourt V: Five-year myocardial infarction incidence - II. Association of single variables to age and birthplace. *J Chronic Dis* 26:329-349, 1973.
15. Glueck CJ, Gartside P, Fallat RW, Sielski J, Steiner PM: Longevity syndromes: Familial hypobeta and familial hyperalpha lipoproteinemia. *J Lab Clin Med* 88:941-957, 1976.
16. Gordon T, Kannel WB, McGee D, Dawber, TR: Death and coronary attacks in men after giving up cigarette smoking. A report from the Framingham study. *Lancet* 2:1345-1348, 1974.
17. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension. Results in patients with diastolic blood pressures averaging 115 through 129 mm Hg. *J Am Med Assoc* 202:1028-1034, 1967.
18. Veterans Administration Cooperative Study Group on Antihypertensive Agents. Effects of treatment on morbidity in hypertension. II. Results in patients with diastolic blood

- pressure averaging 90 through 114 mm Hg. *J Am Med Assoc* 213:1143-1152, 1970.
19. Dayton S, Pearce ML: Prevention of coronary heart disease and other complications of atherosclerosis by modified diet. *Am J Med* 46:751-762, 1969.
 20. Coronary Drug Project Research Group. Clofibrate and niacin in coronary heart disease. *J Am Med Assoc* 231:360-381, 1975.
 21. Klimt CR, Knatterud GL, Meinert CL, Prout TE: A study of the effects of hypoglycemic agents on vascular complications in patients with adult-onset diabetes. I. Design, methods and baseline results; Meinert CL, Knatterud GL, Prout TE, Klimt CR: II. Mortality results. *Diabetes* 19 (suppl 2):747-783; 789-830, 1970.
 22. AMA Council on Foods and Nutrition and the Food and Nutrition Board of the National Academy of Sciences — National Research Council. Diet and coronary heart disease. A Council statement. *J Am Med Assoc* 222:1647, 1972.
 23. McGill HC Jr: Atherosclerosis: Problems in pathogenesis, in Paoletti R, Gotto AM Jr (eds): *Atherosclerosis Reviews*, vol 2, pp 27-65. New York, Raven Press, 1977.
 24. Strong JP, McGill HC Jr: The natural history of coronary atherosclerosis. *Am J Pathol* 40:37-49, 1962.
 25. Tejada C, Strong JP, Montenegro MR, Restrepo C, Solberg LA: Distribution of coronary and aortic atherosclerosis by geographic location, race, and sex. *Lab Invest* 18:509-526, 1968.
 26. Eggen DA, Solberg LA: Variation of atherosclerosis with age. *Lab Invest* 18:571-579, 1968.
 27. Strong JP, McGill HC Jr: The pediatric aspects of atherosclerosis. *J Atheroscler Res* 9:251-265, 1969.
 28. Whyte HM, Yee IL: Serum cholesterol levels of Australians and natives of New Guinea from birth to adulthood. *Australas Ann Med* 7:336-339, 1958.
 29. Hodges RE, Krehl WA: Nutritional status of teenagers in Iowa. *Am J Clin Nutr* 17:200-210, 1965.
 30. Lee VA: Individual trends in the total serum cholesterol of children and adolescents over a ten-year period. *Am J Clin Nutr* 20:5-12, 1967.
 31. Lauer RM, Connor WE, Leaverton PE, Reiter MA, Clarke WR: Coronary heart disease risk factors in school children: The Muscatine study. *J Pediatr* 86:697-706, 1975.
 32. Court MJ, Dunlop M: Plasma lipid values and lipoprotein patterns during adolescence in boys. *J Pediatr* 86:453-458, 1975.
 33. Scrimshaw NS, Balsam A, Arroyave G: Serum cholesterol levels in school children from three socio-economic groups. *Am J Clin Nutr* 5:629-633, 1957.
 34. Golubjatnikov R, Paskey T, Inhorn SL: Serum cholesterol levels of Mexican and Wisconsin school children. *Am J Epidemiol* 96:36-39, 1972.
 35. Frerichs RR, Srinivasan SR, Webber LS, Berenson GS: Serum cholesterol and triglyceride levels in 3,446 children from a biracial community: The Bogalusa Heart Study. *Circulation* 54:302-309, 1976.
 36. deGrott I, Morrison JA, Kelly KA, Rauh JL, Mellies JH, Edwards BK, Glueck CJ: Lipids in schoolchildren 6 to 17 years of age: Upper normal limits. *Pediatrics* 60:437-443, 1977.
 37. Srinivasan SR, Frerichs RR, Webber LS, Berenson GS: Serum lipoprotein profile in children from a biracial community: The Bogalusa Heart Study. *Circulation* 54:309-318, 1976.
 38. Hickie JB, Ruys J: Serum cholesterol and serum triglyceride levels in free-living and vegetarian Australian adolescent children, abstracted. *Circulation* 52 (suppl II): II-4(7), 1975.
 39. Wilmore JH, McNamara JJ: Prevalence of coronary heart disease risk factors in boys, 8 to 12 years of age. *J Pediatr* 84:527-533, 1974.
 40. Glueck CJ, Fallat R, Buncher CR, Tsang R, Steiner P: Familial combined hyperlipoproteinemia: Studies in 91 adults and 95 children from 33 kindreds. *Metab Clin Exp* 22:1403-1428, 1973.
 41. Kwiterovich PO Jr, Levy RI, Fredrickson DS: Neonatal diagnosis of familial type-II hyperlipoproteinemia. *Lancet* 1:118-122, 1973.
 42. Kwiterovich PO Jr, Farah JR, Brown WV, Bachorik PS, Baylin SB, Neill CA: The clinical, biochemical, and familial presentation of type V hyperlipoproteinemia in childhood. *Pediatrics* 59:513-525, 1977.
 43. Glueck CJ, Stein EA: Pediatric considerations in the treatment and management of hyperlipoproteinemia, in Levy RI, Rifkind BM, Dennis BH, Ernst N, (eds): *Nutrition and Coronary Heart Disease*. New York, Raven Press, (to be published), 1978.
 44. Voors AW, Foster TA, Frerichs RR, Webber LS, Berenson GS: Studies of blood pressures in children, ages 5-14 years, in a total biracial community: The Bogalusa Heart Study. *Circulation* 54:319-327, 1976.
 45. *Teenage Smoking: National Patterns of Cigarette Smoking, Ages 12 Through 18, in 1972 and 1974*. (DHEW Pub. No. (NIH) 76-931.) Bethesda, US Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, 1976.
 46. Coates TJ, Thoresen CE: Treating obesity in children and adolescents: A review. *Am J Public Health* 68:143-151, 1978.
 47. Fredrickson DS, Breslow JL: Primary hyperlipoproteinemia in infants. *Annu Rev Med* 24:315-324, 1973.
 48. Hennekens CH, Jesse MJ, Klein BE, Gourley JE, Blumenthal S: Cholesterol among children of men with myocardial infarction. *Pediatrics* 58:211-217, 1976.
 49. Tsang RC, Fallat RW, Glueck CJ: Cholesterol at birth and age 1: Comparison of normal and hypercholesterolemic neonates. *Pediatrics* 53:458-470, 1974.
 50. McGandy RB, Hall B, Ford C, Stare FJ: Dietary regulation of blood cholesterol in adolescent males: a pilot study. *Am J Clin Nutr* 25:61-66, 1972.
 51. McGandy RB: Adolescence and the onset of atherosclerosis. *Bull NY Acad Med* 47:590-600, 1971.
 52. Stein EA, Mendelsohn D, Fleming M, Barnard GD, Carter KJ, duToit PS, Hansen JDL, Bersohn J: Lowering of plasma cholesterol levels in free-living adolescent males; use of natural and synthetic polyunsaturated foods to provide balanced fat diets. *Am J Clin Nutr* 28:1204-1216, 1975.
 53. Stare FJ, McWilliams M: *Living Nutrition*. New York, John Wiley & Sons, Inc, 1973, 467 pp.
 54. Friedman G, Goldberg SJ: An evaluation of the safety of a low-saturated-fat, low-cholesterol diet beginning in infancy. *Pediatrics* 58:655-657, 1976.
 55. Glueck CJ, Tsang R, Balistreri W, Fallat R: Plasma and dietary cholesterol in infancy: Effects of early low or moderate dietary cholesterol intake on subsequent response to increased dietary cholesterol. *Metab Clin Exp* 21:1181-1192, 1972.
 56. *Coronary Risk Handbook*. New York, American Heart Association, 1973.
 57. National Diet-Heart Study Research Group, *The National Diet-Heart Study: Final Report*. (AHA Monograph 18.) *Circulation* 37 (suppl 1):I-1 - I-428, 1968.
 58. Fomon SJ: A pediatrician looks at early nutrition. *Bull NY Acad Med* 47:569-578, 1971.
 59. Hahn P, Kirby L: Immediate and late effects of premature weaning and of feeding a high fat or high carbohydrate diet to weaning rats. *J Nutr* 103:690-696, 1973.
 60. Hahn P, Koldovsky O: Late effect of premature weaning on blood cholesterol levels in adult rats. *Nutr Rep Int* 13:87-91, 1976.
 61. McBean LD, Speckmann EW: An interpretive review: Diet in early life and the prevention of atherosclerosis. *Pediatr Res* 8:837-842, 1974.
 62. Reiser R, Sidelman Z: Control of serum cholesterol homeostasis by cholesterol in the milk of the suckling rat. *J Nutr* 102:1009-1016, 1972.
 63. Glueck CJ, Tsang RC: Pediatric familial type II hyperlipoproteinemia: Effects of diet on plasma cholesterol in the first year of life. *Am J Clin Nutr* 25:224-230, 1972.
 64. Friedman G, Goldberg SJ: Concurrent and subsequent serum cholesterols of breast- and formula-fed infants. *Am J Clin Nutr* 28:42-45, 1975.
 65. Hodgson PA, Ellefson RD, Elveback LR, Harris LE, Nelson RA, Weidman WH: Comparison of serum cholesterol in children fed high, moderate, or low cholesterol milk diets during neonatal period. *Metab Clin Exp* 25:739-746, 1976.
 66. Sturdevant RAL, Pearce ML, Dayton S: Increased prevalence of cholelithiasis in men ingesting a serum-cholesterol-lowering diet. *N Engl J Med* 288:24-27, 1973.
 67. American Academy of Pediatrics. Committee on Nutrition. Childhood diet and coronary heart disease. *Pediatrics* 49:305-307, 1972.

This statement was prepared by an ad hoc committee of the Steering Committee for Medical and Community Program of the American Heart Association. Membership of the Committee at the time this statement was prepared:

Charles J. Glueck, M.D., Henry C. McGill, Jr., M.D.
 Chairman
 Ronald M. Lauer, M.D. Robert E. Shank, M.D.

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American Heart Association

National Center 7320 Greenville Avenue Dallas 75231

EXHIBIT D

February 15, 1979

TO; Edwin Bierman, M.D.
Robert Corwin, M.D.
Charles Glueck, M.D.

FROM: Mary Winston, Ed.D.
Chief, Nutrition Programs

SUBJECT: SCRIPT -- NUTRITION FILM -- TEENAGERS

We have a fourth script to review for our nutrition film. The script writer has spelled out her philosophy, objectives, and strategy which I think will help when you look at the script. Most of what will be done in teaching has to come from this supplemental material which accompanies this film. Because we have been procrastinating so long with this film, may I ask you to review and send your comments on the enclosed back to me by March 1.

I hope that we can have a revised script to present to the entire Committee in April.

Thank you very much.

/jf

Enclosure

HERE'S LOOKING AT YOU KID

A 10-12 minute 16mm film on nutrition for 7th graders

PHILOSOPHY:

7th grade students (13-14 yrs.) are in the stage of development when an individual seeks to establish his identity and independence by reacting against parental constraints or voices of authority, and when doing his own thing is most important. We feel that if we don't motivate the students first, good information stands a good chance of falling on deaf ears.

OBJECTIVE:

To motivate 7th grade students to want to eat foods that are good for them. We want the decision to eat right to be their own decision based on their own reasons.

STRATEGY:

To motivate, we must offer a motivating factor, a compelling reason why. Our motivating factor in HERE'S LOOKING AT YOU KID is that good nutrition goes hand in hand with feeling great, looking good (hair, skin, figure), maintaining a high energy level, and in general, a look of health, vitality and fitness from head to toe.

Our film will be an exciting visual presentation using voice over narration and on camera conversation by two young, healthy, appealing and very good looking kids.

We will feature a young, black prize fighter who is a middle-weight contender named Sugar Ray Phillips. He is very personable and exciting. We will also feature a young model, just starting out in her career. She is down to earth, excited about her work and anxious to share her secrets of staying in top shape. Both will emphasize good nutrition as a major part of their ability to stay fit and healthy.

Through them, we will develop the concept of energy balance (calories taken in versus those burned during exercise) and good foods (foods low in fat, high in nutrients). They will also illustrate the fact that foods that are good for you can be eaten any time of the day (i.e., chicken for breakfast, cereal for lunch if so desired, etc.).

They will not spout information like textbooks. Instead, they will talk about their lives and what they do to stay fit and healthy. Good nutrition points will be worked in naturally as they discuss how they take care of themselves.

Our film will make eating right, looking good and feeling good, look like and sound like the greatest way to be in the world.

MAIN NUTRITION POINTS:

- 1) Foods that are good for you are low-fat, low-calorie and carry nutrients.
- 2) Good foods can be eaten at any time of the day. (Chicken for breakfast, ect...).
- 3) Energy balance concept. The calories from food you eat should be in balance with the calories you burn up during exercise. Fat is food we ate and didn't need.

Adolescents will perk up and listen to ways of looking and feeling better. Our film will suggest that one way to look and feel better is through good nutrition. The results? Better health in the eyes of the American Heart Association. A way to improve skin, hair, physique/figure and maintain high energy levels in the eyes of the kids.

An example of the excitement and feeling we want to create in the film can be seen in McDonald's commercials geared toward adolescent audiences currently running on television. These establish an image for McDonald's in the eyes of the kids. The spots don't mention hamburgers. Instead, they show hamburgers and they show kids having a great time, outdoors, playing, running, laughing, living life the way an active kid loves to live it. The footage is intercut with them taking bites of McDonald's hamburgers. The McDonald's jingle is playing in the background and in closing, we see the logo. We can borrow from this approach and create the same highly charged atmosphere of life, health, happiness and fun putting special visual emphasis on good skin, good physical condition, good hair and good nutrition. We will visually suggest the link between eating well and feeling and looking good. Good nutrition will be introduced by Sugar Ray and our model and reinforced by intercutting them eating well during the film.

We will not flood this film with textbook nutrition information. We will use the film medium to its advantage creating excitement through the visuals and adding some nutrition information in the dialogue and voice over. We are suggesting to the audience that some of the results of good nutrition are right before our eyes. We are making no guarantees....rather, we are saying, "Okay, if you want to be the best you can be, here's a way to start."

Information in the film is pared down to important points. If the audience comes away with just a few good ideas about nutrition and anxious to find out more, we've succeeded.

The film should serve as a trigger, a springboard for the teacher to teach the specifics of nutrition. Because the information is limited and because this is a motivational, not an informational nutrition film, it must be used in conjunction with special materials developed for the teachers and with special student activities to teach specifics.

HERE'S LOOKING AT YOU KID

Nutrition Film for 7th Grade Students

CHARACTERS

Sugar Ray: A black prizefighter, somewhat Rocky-ish, hard-working, appealing. Expresses himself simply, but intelligently.

Model: Very chic, fashionable, even in a warmup suit. Articulate, even chatty. Poised and self-assured, but down to earth, believable.

HERE'S LOOKING AT YOU KID

OPEN WITH BLACK.

SUDDEN PARTIAL ILLUMINATION FROM REFRIGERATOR LIGHT.

MS OF SUGAR RAY IN SILHOUETTE, REACHING INTO FRIDGE.

CU OF S.R. IN WARMUP SUIT PULLING OUT CHICKEN LEG, SKIM MILK, ETC.

MS S.R., YAWNS, TURNS ON WEAK KITCHEN LIGHT, TAKES BREAKFAST TO TABLE.

CU OF BLENDER. ZOOM OUT TO MS OF YOUNG GIRL IN WARMUPS PUTTING FRESH FRUITS, MILK, ETC., INTO BLENDER.

MS S.R. FINISHES DRUM STICK, DISPOSES.

PAN SHOT FOLLOWS S.R. OUT THE DOOR INTO DUSKY MORNING LIGHT.

LS OF S.R. STRETCHING EXERCISES IN THE YARD.

Live SFX

Footsteps coming downstairs, across floor. Click of refrigerator door.

SFX: Blender

V.O. (Sugar Ray)

I get up 5:30, 6:00 every morning. I like a good breakfast, usually some chicken and skim milk, some apples, sometimes a bowl of cereal and toast with orange juice. Food is like fuel to me. I gotta get plenty of vitamins and minerals and lay off the sweets 'cause I'm runnin' six miles every morning, in combat boots with ten pound weights in my hands. I'm gonna be Middle-weight

CU'S OF S.R. PUTTING ON RUNNING SHOES.

champion of the world. Name's Sugar Ray Phillips. Don't forget it.

LS OF MODEL DOING STRETCHING EXERCISES IN LIVING ROOM.

V.O. (Model)

I've had to do a major overhaul on my eating habits, or else . . . I never used to care what I ate as long as it filled me up fast. I've got this appetite . . . you just wouldn't believe. But now, eating right is like a part of my job. I'm a model, well, I'm just beginning . . . I've got to keep my weight down and I've got to look healthy. I've really worked hard at it, but now it's becoming second nature to reach for something low in fat and sugar when I'm hungry. I go for foods that carry something besides just calories. . . I really need the energy and nutrients they give me . . . like I'll eat yogurt, or an apple instead of a candy bar when I'm starving.

LS'S OF S.R. RUNNING BY THE LAKE, SHADOW BOXING.

V.O. (S.R.)

Somebody told me that Muhammed Ali's training table is loaded with a dozen eggs, five glasses of juice, a twelve ounce steak, a bowl of oatmeal and three glasses of skim milk every morning when he's in training. That's about twice what I take in, but most people don't even need half of what I eat.

LS'S OF MODEL RUNNING.

V.O. (Model)

Eating right used to be just so I could try to lose weight or keep it down. Especially when I first started working. But now it's something more. I just feel great! And I know eating right has had a lot to do with it. It shows! My skin has a glow I never ever thought it would have . . . and my hair shines more, it acts healthy too.

MS MODEL RUNS UP TO HER HOUSE.

Other girls I know -- a lot of them are models -- depend on how they look and feel for a living . . . and I would say one of the most important secrets they have is good nutrition. They also exercise a lot and try to balance the two out so they burn up a lot of what they eat. That's what I'm trying to do every morning.

ON CAMERA: "Whew, good Workout."

MS S.R. WALKS AROUND CAMERA, TRUCK SHOT FOLLOWS HIM UP STEPS INTO GYM.

OnCAM (S.R.): I love food . . . like to give it a good home . . . but I burn it all up. You gotta burn up what you take in. If you don't, you get flabby. That doesn't make it. Good food helps my body perform better.

MS'S & CU'S OF S.R. WORKING WITH LIGHT BAG.

SEQUENCE -- MODEL ARRIVES ON SET ...
ESTABLISH STUDIO WITH LIGHTS,
PHOTOGRAPHER, LIMBO BACKDROP, ETC.

...DOING HER MAKEUP... (CU'S LIPSTICK, EYES,
BLUSH, ETC.)

...DOING HER HAIR...DRESSING.

...BEING PHOTOGRAPHED, INTERCUT WITH
STILL PHOTOS OF HER IN VARIOUS POSES.

...EATING LUNCH (TORTILLAS WITH LOW FAT
CHEESE SPREAD).

LS'S S.R. JUMPING ROPE, INTERCUT WITH SLO-
MO.

...STOPPING TO EAT LUNCH.

V.O.: (S.R.): Eatin' more than you need to slows anybody up, and it can really mess up your blood vessels. Take a look at this pipe. See that crud in there. Well, fat latches on to the insides of your blood vessels just like the rust in this pipe, and there's not enough room for the blood to flow through to your heart. Might cause a heart attack some day when you're older. Feelin' good, that's what it is.

V.O.: (Model)

I'm in a very competitive business. When there's twenty other girls applying for the same job, you really feel the pressure, too. Knowing I'm super-healthy and looking it gives me the kind of edge, or self-confidence that a photographer looks for. I also know that by eating right I'm giving myself a better chance of living longer ... making things easier on my heart, my digestion ... and that's real important to me.

V.O. (S.R.)

I used to feel like I was the best. These days, I know I'm the best. I'm gonna win that title to prove it, and I'm gonna stay champ for a long time. A great fighter needs strong legs, fast hands, and a strong heart. Eatin' right and

exercising plenty keeps your heart healthy for a long time. That makes anybody a winner.

AFTERNOON SEQUENCE

RECAP SEQUENCE (2-3 MINUTES).

SLO-MO'S - RUNNING
MODEL
S.R.
MODEL DOING HAIR.
S.R. JUMPING ROPE.
MODEL POSING.
S.R. SHADOW BOXING.

V.O. (Model)

People really notice you when you're at your best . . . it's that glow. You radiate health & vitality. They know you think a lot of yourself. My diet thing has become some kind of philosophy . . . it's fun, and it pays off, too.

V.O. (S.R.)

Eatin' right . . . feelin' good. That's it.

FREEZE FRAME - S.R. PUNCHING INTO CAMERA -- DISSOLVES TO --TITLES.

FREEZE FRAME - MODEL IN POSE, SMILING -- DISSOLVES TO -- TITLES.

PUBLIC POLICY SUBCOMMITTEE
OF THE
AMERICAN HEART ASSOCIATION
COUNCIL ON EPIDEMIOLOGY

Excerpts From Summary Notes

November 11, 1978 Meeting

- (1) Nutrition Labeling - The subcommittee members expressed concern that, in spite of policies supporting fat and sodium content labeling, AHA had chosen not to endorse related petitions prepared by the Center for Science in the Public Interest because of presumed potential political problems involved in an association with that organization. Note was taken of the absence of AHA support despite the endorsement by a broad list of knowledgeable scientists and professional organizations, including the American College of Cardiology. The process by which endorsement was denied also came into question. It was recommended that, to the extent possible (given the frequent urgency of public policy decision-making), relevant Council expertise be sought before final action is taken on such matters.

Dr. Stamler suggested that in order to be most productive and to avoid what has been de facto a primarily reactive public policy program, public policy objectives, priorities and initiatives should be determined for the coming periods, with a focus on the next U. S. Congress.

- (3) Change in U. S. D. A. Atmosphere. It was noted that the Department of Agriculture may no longer be a unified front for agribusiness, at least in the nutrition arena. The appointments of Carol Tucker Foreman and Mark Hegsted to key spots in U. S. D. A. are an encouraging sign of change. Staff was asked to prepare a profile of the various forces concerned with the nutrition discussion in Washington. A personal meeting with Hegsted or Foreman was suggested as a method to augment AHA understanding of the prognosis for Washington action on nutrition and health. Ways should be sought in which AHA can work cooperatively with U. S. D. A. and with the nutrition subcommittees in Congress -- as well as with NHLBI - NIH - DHEW, consumer groups, etc. -- to achieve dietary objectives.

- (5) Exporting the American Diet. It was noted that Title XII of the foreign aid authorization statute authorizes the Agency for International Development to identify the nature of energy intake in developing countries, and to recommend changes. There is the possibility that these recommendations will not be optimal, e.g., in regard to emphasis on foods that are atherogenic. Staff was asked to prepare a further briefing on this for the subcommittee.

HOTEL DEL CORONADO

600 Rooms

1500 Orange Avenue, Coronado 92118. PHONE: (717) 435-6611. KEY PERSONNEL: Carleton Lichty, Pres. & Gen. Mgr.; Hal J. Carlsen, Exec. V.P. & Sales & Mktg. Dir.; Claude Webb, V.P. of Operations; Tom O'Hara, V.P. & Mgr.; Nino Hirsig, V.P. & Food & Bev. Dir.; Duane Spandl, V.P. Finance; Fred Patino, Asst. V.P. & Conv. Coord.; Heather Dobbins, Conv. Coord.

LOCATION/TRANSPORTATION FACTS—Year-round beach resort directly on the Pacific Ocean on Coronado Peninsula, 7 miles 10 minutes from downtown San Diego. 9 miles, 10-15 minutes from Lindbergh Field; taxi \$7.60. 12 miles from noncommercial Montgomery Field; 7 miles, 10 minutes from Amtrak railroad station. Five 6-14 passenger charter hotel limousines available for airport and railroad transfer at \$3.50 per person. Access via N/S Highway 5 to Coronado Bridge to southwest portion of Peninsula.

ACCOMMODATIONS—Traditional grand hotel built around a garden courtyard, a modern Hawaiian-style addition houses 600 rooms including 29 suites and 60 lanais. All have direct-dial phone. Many have individual-control air conditioning, color TV, bathroom phone, balcony, and full glass walls. Some have dressing room and balcony.

DINING/ENTERTAINMENT—Restaurants include the Crown/Coronet Room, the majestic Main Dining Room with a paneled domed ceiling dating from 1888, specializing in American and Continental cuisine, open 7 AM-9 PM with dinner entrees from \$7.50; and the Prince of Wales Grille. Dancing and entertainment in the Ocean View Room nightly from 9 PM. Cocktails in the Casino Lounge and the Lobby Lounge from 10 AM-1:45 AM.

SERVICES/FACILITIES/SHOPS—Terrace level shopping arcade. Car rental counter. Staff Language Capability: French, Spanish, German, Italian, Japanese, Greek, Danish, & Flemish. Outdoor self-service and valet parking, \$1.50 for each service; overnight valet \$2.

RECREATIONAL/AMUSEMENT FACILITIES—Heated outdoor swimming pool. Private white sand beach. Hotel boathouse on Glorietta Bay with sailboats, power boats, water skiing & boat slips; fishing charters arranged. Health club with sauna and steam baths. 7 illuminated outdoor tennis courts with pro; \$6 per hour for courts.

RATES—Year-round, Convention plan, Single \$35-45, Double/Twin \$45-55, Lanai \$65, Parlor Suite \$95, Kitchenette Apt.-1BR \$115, 2BR \$155. American plan \$19 additional per person. Additional guest in double room \$5 per person. Rates differ in July & August. Room tax: 6%. Check out: noon. **Credit Cards:** American Express, BankAmericard/Visa, Hotel del Coronado, Master Charge. **For Direct Billing:** arrange with Credit Mgr.

—Principal Meeting Facilities, Equipment and Services—

GENERAL COMMENTS—18 meeting rooms, several of which divide to provide an additional 22, are concentrated in the main building on three floors. All have Altec sound systems, multiple electrical/microphone outlets and individual controls for sound. Levels linked by stairways and elevators. Separate banquet kitchen.

The Grande Hall accommodates 71 8'x10' or 65 10'x10' booths. Electricity: 220V. Truck dock: 30' wide. Truck access to freight lift: 10' deep, 20' wide.

Name of Room	Ballroom	Grande Hall	International	Garden
Dimensions (LxWxH in feet)	dia. 160 ft. H-26'	88x134x26	H-10'	33x47x9
Square Feet	11,800	11,300	8500	1551
Floor Number	1	2	Arcade	1
Floor Cover	Carpet/Wood	Carpet	Carpet/Wood	Carpet
Portable Walls	No	Yes	No	No
Capacity				
Auditorium	1200	1200	600	200
Classroom	800	900	400	100
U-Shape	NA	NA	NA	NA
Reception	1100	1200	600	100
Banquet	1000	1100	500	90

MEETING EQUIPMENT:

Supplied by the hotel—at no charge: Permanent & Portable Stages; Installed PA System; Lectern, Portable and Floor Microphones; Lecterns; Podiums; Padded Metal Stack Chairs; Blackboards; Easels/Tablets; Cork Boards; Lobby Bulletin Board.—**at a charge:** Portable PA Systems; Reel & Cassette Tape Recorders/Players; Phonographs; A/V Replacement Parts; Overhead, 16mm Sound, Opaque & 35mm Slide Projectors; Remote Control Cords; Writing Pads, Pens, Pencils; Duplicating Machines; Individualized Direction Signs.

Supplied by local vendors—Rear Screen, 8mm Sound and Film Strip Projectors; Typewriters; Name Cards/holders; Truck/Van.

MEETING SUPPORT SERVICES:

Provided by the hotel—at no charge: Plumber; Locksmith; Laborers, Attendee Registration.—**at a charge:** Messenger; Notary Public; Print Shop; Photographer/Photo Shop; Carpentry & Paint Shop; Electrician; Musicians; Security Guards; A/V Operators & Repairmen.

Provided by local vendors—Stenographer; Display Builder; Sign Painter, Decorating Service; Tours & Entertainment.

COMMONWEALTH OF PENNSYLVANIA

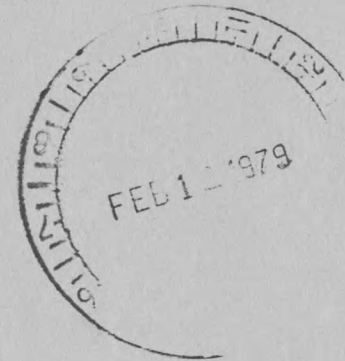


DEPARTMENT OF HEALTH

P. O. BOX 90

HARRISBURG 17120

February 5, 1979



American Heart Association
7320 Greenville Avenue
Dallas, Texas 75231

Dear Sir:

On October 26 through 28, 1978, the American Heart Association presented in Fort Lauderdale, Florida, a scientific session on "Coronary Atherosclerosis - A Review of Nutritional Factors in Prevention and Management".

I would like the American Heart Association to consider exploring the possibility of repeating this program in Pennsylvania in conjunction with the Division of Nutrition Services as a co-sponsor. The American Heart Association, Pennsylvania Affiliate, with whom I work very closely, would also be a potential sponsor.

If you would be willing to discuss this proposal at length, please contact me at (717) 787-5375 or 5376 between the hours of 7:30 a.m. and 4:00 p.m.

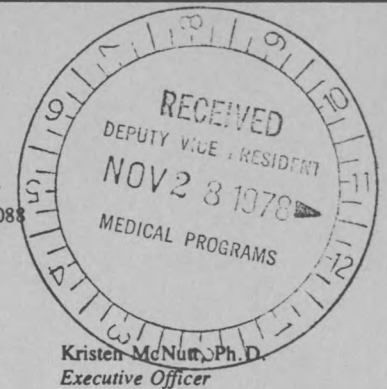
I will look forward to hearing from you.

Sincerely,

Janice M. Whitfield
(Mrs.) Janice M. Whitfield, R.D.
Nutrition Consultant for Adult Health
and Chronic Diseases
Division of Nutrition Services

EXHIBIT G

National Nutrition Consortium, Inc.
Suite 216 • 2121 P Street, N.W. Washington, D.C. 20037 • Telephone: (202) 659-0088



Member Societies

AMERICAN DIETETIC ASSOCIATION
AMERICAN INSTITUTE OF NUTRITION
AMERICAN SOCIETY FOR CLINICAL NUTRITION
INSTITUTE OF FOOD TECHNOLOGISTS
SOCIETY FOR NUTRITION EDUCATION
AMERICAN HOME ECONOMICS ASSOCIATION
AMERICAN ACADEMY OF PEDIATRICS
COMMITTEE ON NUTRITION
FOOD AND NUTRITION BOARD, NATIONAL ACADEMY
OF SCIENCES/NATIONAL RESEARCH COUNCIL

November 22, 1978

Dr. Richard E. Hurley, M.D.
Executive Deputy Vice President
in Medical Affairs
American Heart Association
7320 Greenville Avenue
Dallas, Texas 75231

1978 - 1979 Board of Directors

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University of Delaware
Miss Joan L. Sharp, R.D., (ADA)
Ohio State University Hospitals
Dr. Myron Winick (ASCN)
Columbia University
Dr. Esther Winterfeldt, R.D. (ADA)
U.S. Dept. of Agriculture

Dear Dr. Hurley,

The National Nutrition Consortium is a non-profit organization comprised of the major professional societies in food, nutrition and a dietetics. The Consortium is in the process of some major changes in staff and expanded programs. Some of these are described in the enclosed press release.

I am enclosing some materials that describe the activities of the Consortium prior to August 1978 and possible new Consortium projects in our four program areas. Some of these projects have now been started. Also, I am attaching the draft of copy for our new brochure. I apologize that we do not have the completed brochure but many of the programs described herein have only been initiated within the last few weeks. Because of our pressing needs for financial support in the very near future, I am sending this draft. I will of course send you a final copy as soon as they become available.

With our full time staff, downtown offices and expanded programs we will be operating in 1979 at an annual budget level of approximately \$100,000. We are guaranteed an income of \$16,000 from member organization dues. I hope that this will be considerably supplemented by individual voluntary contributions from the 80,000

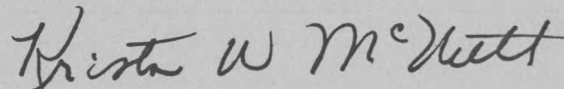
Page Two
Dr. Richard Hurley
November 22, 1978

persons in our member organizations. We also anticipate some income from sale of two Consortium publications. As stated in our brochure, the Consortium accepts contributions to defray general operating expenses from foundations, companies, and the consuming public.

I would greatly appreciate any consideration the American Heart Association might give to our request for a contribution. We are hoping to receive donations in the range of \$5,000. The Consortium is classified by the Internal Revenue Service as a 501(c)3 organization.

Thank you very much.

Sincerely,



Kristen W. McNutt
Executive Officer

KWM:dn

Enclosures: Brochure draft copy
Possible National Nutrition Consortium Projects
Overview of the Consortium
October 18 Press Release

COVER COPY

(Draft 11/17/78)

National Nutrition
Consortium, Inc.

Member Societies

American Dietetic Association
American Institute of Nutrition
American Society for Clinical NUTRITION
Institute of Food Technologists
Society for Nutrition Education

Liaison Societies

American Home Economics Association
American Academy of Pediatrics,
Committee on Nutrition
National Academy of Sciences,
Food and Nutrition Board

DRAFT

To Board for
comment and
approval 11/17/78

2121 P Street, N.W., Suite 216
Washington, D.C. 20037
(202) 659-0088

DRAFT

The National Nutrition Consortium, is a non-profit organization comprising the major professional societies in food, nutrition and dietetics. The cumulative membership of these societies totals approximately 80,000 scientists, physicians, educators and dietitians who have training and experience in nutrition.

Goals and Objectives

The primary goal of the National Nutrition Consortium is to provide accurate information about nutrition. In areas where there exists lack of scientific concensus among professional nutritionists, the Consortium offers the public and policy makers access to a spectrum of opinions rather than leaving them dependent upon polarized points of view.

The Consortium also provides coordination and communication among its member organizations. This is accomplished by board member interaction, monthly communication of activities of the Consortium and its member organizations, and joint participation of individuals from each society in Consortium committees.

A "Report From the Consortium" is printed every two months in the professional journals and newsletters of member organizations. Since many of these publications circulate in libraries, the readership audience greatly exceeds the total membership represented.

Services

The Consortium serves as a clearinghouse for information on nutrition. It provides access to the various services and committees of its member organizations located across the United States. The Consortium does not duplicate the functions of its member organizations but complements and strengthens them. The Consortium refers inquirers to nutrition experts and other sources of

information when requests fall beyond the scope and function of its own Washington staff.

The Consortium welcomes inquiries from the news media, consumer groups, congressional staff and other health and education organizations.

Staff and Committees

The Consortium maintains permanent offices with full time staff in Washington, D.C. These resources facilitate communications with government agencies, Congress, consumer groups, other health and education organizations, and news media.

Many of the programs of the Consortium are implemented by its committees which focus primarily on consumer information, public affairs and nutrition education. Ad hoc committees are appointed as required to carry ^{out} new or short term functions, programs and projects of the Consortium.

Fellowship Program

The Consortium sponsors a fellowship for graduate students in fields related to nutrition, health and food science. Candidates for Ph.D. or M.D. degrees, who are in nutrition and whose universities accredit a one-semester experience, are selected to work with the Consortium staff on specific projects. The students gain experience in nutrition policy development, government organization and the operations of professional nutrition societies. They prepare a report on their experiences for their academic committee and participate in graduate seminars of universities in the Washington area.

Governance

Policy and programs of the Consortium are determined by a board comprised of three delegates from each member society and one delegate from each

liaison society. The board meets regularly to review staff and committee activities, to initiate new programs, and to determine Consortium policy. The board provides leadership in the development and coordination of food and nutrition policies at the national, state and local levels.

Funding

The Consortium is supported primarily by dues from its member and liaison societies, contributions from individuals in these societies and sale of publications. It accepts contributions from foundations, companies and the consuming public to defray general operating expenses.

(Biosketch)

Dr. Kristen W. McNutt became executive officer of the National Nutrition Consortium in September 1978. Dr. McNutt earned her B.A. in chemistry at Duke University, M.A. in nutrition at Columbia University and Ph.D. in biochemistry at Vanderbilt University.

Prior to joining the Consortium, she served as FASEB Congressional Science Fellow with the Senate Agriculture, Nutrition and Forestry Committee for one year and as research associate with the Nutrition Foundation for four years. She also served as volunteer public health nutritionist with Project HOPE in Brazil.)

She and her husband, Dr. David McNutt, coauthored the college textbook Nutrition and Food Choices.

Member Organizations

Each member organization of the Consortium maintains permanent executive offices with professional staff. Each has a variety of committees and divisions responsible for implementing specific programs, developing position papers and statements, and responding to issues of public and professional interest. These societies also offer publications on food and nutrition.

Each society conducts an annual meeting designed to present to its membership the most recent information in its discipline. This is accomplished by research reports, panel discussions, workshops, and exhibits. Several organizations also administer professional recognition awards.

The professional journals of these societies include Journal of the American Dietetic Association, Food Technology, Journal of Nutrition Education, Nutrition, American Journal of Clinical Nutrition, Pediatrics, and the Journal of Home Economics. These have a cumulative subscription of approximately 150,000. In addition these societies publish newsletters informing ^{their members} of organization activities, current events and public affairs.

The AMERICAN DIETETIC ASSOCIATION (ADA) is the professional organization of 37,000 dietitians who work in hospitals, universities, health delivery facilities, food service institutions and a variety of other arenas. Their training includes food preparation ^{and} ~~an~~ delivery, the role of diet in health maintenance and dietary management of diseases. They counsel patients, educate the public, aid in nutrition training of other health professionals and provide numerous other professional services.

The ADA establishes educational standards for the profession of dietetics and maintains continuing education programs for practitioners. Each March the ADA sponsors National Nutrition Week, an event supported by the Consortium and its other member organizations.

DRAFT

The AMERICAN INSTITUTE of NUTRITION (AIN) has 1,700 members, most of whom conduct nutrition research and teach in departments of biochemistry or other related sciences such as physiology, molecular biology and toxicology. They work primarily in universities, colleges and medical schools. Membership in AIN is limited to persons whom have, in the opinion of their scientific peers, demonstrated expertise in well designed and executed research and who have made contributions to the growth and application of the science of nutrition. Most members have maintained an academic position for several years and have published their work in peer-refereed journals.

AIN participates in intra-hemisphere and international conferences on nutrition. It maintains information on the professional expertise and specific areas of interest of its members and provides information about graduate programs in nutrition. AIN is a member of the Federation of American Societies of Experimental Biology (FASEB).

The AMERICAN SOCIETY OF CLINICAL NUTRITION (ASCN) represents 400 clinical nutritionists who are also members of the American Institute of Nutrition. Many are physicians. Clinical nutritionists are active in graduate and undergraduate education in universities, schools of public health, and medical schools, and conduct research primarily related to the practice of medicine.

ASCN provides services similar to those of AIN. It holds an annual meeting in conjunction with several other professional societies which focus on various aspects of clinical research.

The INSTITUTE OF FOOD TECHNOLOGISTS (IFT) represents 17,000 professionals with training in food science and related disciplines such as nutrition, engineering, chemistry, biology, genetics, biochemistry, microbiology and toxicology. Most IFT members work in the food industry or universities, conducting research designed to maintain food quality and safety. Results

of this research are applied in the food processing and delivery systems.

IFT has an active public information program that includes a network of persons around the country who answer questions from the news media regarding food technology. IFT Scientific Status Reports are developed in response to consumer interest in various food-related topics.

The SOCIETY FOR NUTRITION EDUCATION (SNE) represents a cross-section of 5,000 professionals including teachers, nutritionists, health professionals and communicators. Members work in schools at all levels, state and community health facilities, agriculture extension programs, the news media, and many other areas. Nutrition educators are involved in improving educational methodologies and quality of nutrition information reaching students and the public.

SNE publishes resource materials for educators and the public and has produced several films on nutrition. It maintains the National Nutrition Education Clearing House, a comprehensive collection of print materials related to nutrition and education. SNE also reviews new print and audio materials for nutritional accuracy and educational appropriateness.

The AMERICAN ACADEMY OF PEDIATRICS (AAP) is comprised of 17,000 board certified pediatricians whose goal is the attainment by all American children of their full potential for physical, emotional and social health. These pediatricians work in private and group practices, in various health facilities including hospitals, pediatric practice and pediatric research.

The Committee on Nutrition of AAP is comprised of pediatricians and other scientists with expertise in nutrition. It compiles and publishes the essential facts which are the scientific basis for practical nutrition of infants, children and adolescents. The committee also comments on legislative issues which affect the nutritional well-being of mothers and children.

DRAFT

The FOOD and NUTRITION BOARD (FNB) , as an entity of the National Academy of Sciences-National Research Council, serves under the terms of the charter of the National Academy of Sciences as an advisory body to federal agencies and, on its own initiative, to the general public on science as related to food and nutrition. It participates in guiding the implementation of effective policies to assure an adequate, wholesome, and safe food supply. Support of research and educational processes necessary to further that effort is part of its mission.

The FNB is comprised of 16 members with rotating appointments. The liaison panels are a Consumer Panel, an Industry Panel, and Panels of representatives of federal agencies, scientific societies and trade associations.

Many functions of the FNB are performed by broader committees of persons with expertise in specific areas of food and nutrition. These committees include Clinical Nutrition, Dietary Allowances, Food Protection, GRAS List Survey, International Programs, Food Science and Technology, Nutrition of the Mother and Preschool Child, Dental Health, Sodium-Restricted Diets, and Nutrition, Brain Development and Behavior.

The AMERICAN HOME ECONOMICS ASSOCIATION (AHEA) represents 50,000 home economists who work in colleges and universities, extension, human services, business, homemaking and elementary, secondary, and adult education. Home economists teach, conduct research, and administer programs related to services for the young, elderly, handicapped, and the entire family. There are over 10,000 members of the Food and Nutrition Section of AHEA.

AHEA promotes professional standards and conduct, improves the quality of individual and family life, and encourages individual professional development. The Association's Center for Family directs attention and action to trends, related to the family, through advocacy, research, leadership development, resource referral and program generation.

National Nutrition Consortium, Inc.

Suite 216 • 2121 P Street, N.W., Washington, D.C. 20037 • (202) 659-0088

Member Societies

AMERICAN DIETETIC ASSOCIATION
AMERICAN INSTITUTE OF NUTRITION
AMERICAN SOCIETY FOR CLINICAL NUTRITION
INSTITUTE OF FOOD TECHNOLOGISTS
SOCIETY FOR NUTRITION EDUCATION
AMERICAN HOME ECONOMICS ASSOCIATION
AMERICAN ACADEMY OF PEDIATRICS
COMMITTEE ON NUTRITION
FOOD AND NUTRITION BOARD, NATIONAL ACADEMY
OF SCIENCES-NATIONAL RESEARCH COUNCIL

Kristen McNutt, Ph.D.
Executive Officer

THE NATIONAL NUTRITION CONSORTIUM *

The National Nutrition Consortium was organized early in 1973, with sponsor members American Institute of Nutrition, American Dietetics Association, Institute of Food Technologists, and the American Society for Clinical Nutrition. During the following five years, the Society for Nutrition Education, the American Academy of Pediatrics, the Food and Nutrition Board of the National Academy of Sciences, and in 1977 the American Home Economics Association joined as affiliate members.

It is recognized that in view of the greatly increased interest of the American public in nutrition, it is time for greater effort on the part of nutrition scientists and informed nutrition educators to bring to the public sound facts of nutrition, the requisites for a healthful, satisfactory diet, and the assurance that a well-balanced diet can provide all nutritional needs.

The National Nutrition Consortium, representing these professional nutrition societies, is in a particularly advantageous position to pull together and present the balanced judgements of the scientific nutrition community when addressing nutrition problems of special concern to the public and the representatives of the public.

One of the advantages of the Consortium is its vested interest in scientific facts of nutrition and the application of those facts for the public good. The Consortium is not selling a product, and any position taken is bound to be scientifically sound, cautious, and conservative.

The Consortium has been addressing problems of nutrition education, food quality and nutritional safety of food, National Nutrition Policy and its implementation, and nutrition research priorities and support.

Increasingly, the Consortium has become involved in providing resources of nutrition expertise to the executive and legislative arms of the Federal Government as consideration is given to the role of diet in health care and health maintenance, to food production policy, and to monitoring of food use, as key factors in the well-being of our people.

* Summary of activities prior to August 1978

In November 1973, the National Committee on Labeling and Education (a food industry committee) offered a grant to the Consortium to develop guidelines for the food industry that would be helpful in their meeting requirements of the Food and Drug Administration nutrient labeling regulations and provide an informative statement to the Consumer. Panels of nutritionists were appointed to prepare position papers on the subject of nutrition and suitable information on proposed labeling, which were reviewed by a committee which then led to the production of the booklet, "Nutrition Labeling - How It Can Work For You". Some 30,000 copies were distributed gratis to communicators with the public. Some 30,000 additional copies have been sold to the public.

In 1974 a Statement of a National Nutrition Policy was developed by the Consortium. Dr. Grace Goldsmith, Dr. William J. Darby, and Dr. Jean Mayer constituted a Committee to prepare the Statement, which was approved by the Board and offered to Senator McGovern's Senate Select Committee on Nutrition and Human Needs. The Statement was published by that Committee.

In 1974 the Consortium provided assistance to Senator Schweiker and Senator Kennedy in supporting the Nutrition Education Medical Act to provide funding of nutrition teaching in medical schools.

The Consortium was instrumental in supporting the requirement for paid services in nutrition, which appears in the regulations governing the Health Maintenance Organizations supported by HEW.

The so-called Proxmire Bill, S.548, offered in the Senate in 1974, prohibited the FDA from setting upper limits of amounts of vitamins and minerals to be contained in vitamin-mineral supplement preparations. The Consortium provided both testimony and a number of written statements which explained the hazards of such legislation. In this case, the National Health Federation was sufficiently strong to overcome objections of responsible scientists.

In September 1975, under contract with the Federal Trade Commission, the Consortium appointed a panel of nutritionists to study and evaluate proposed regulations on nutrition advertising and to provide revisions of the proposal. An excellent statement was the result. The problems with the proposed regulations have not yet been resolved.

The Consortium has offered comments to the Federal Trade Commission on the problems of protein supplements and proposed rule-making with respect to advertising these products.

A statement on the effective use of fluoride in the prevention of dental caries has been widely distributed, and a number of comments have been made in opposition to the claim of the National Health Federation* that fluoride is a cause of cancer. We have worked with the Dental Institute of the NIH and the American Cancer Society on this problem.

*An organization supported by the health food industries.

In view of the reduced authority of the FDA to control amounts of vitamins and minerals in supplements, and in view of the growing megavitamin craze, it was important to prepare a position paper on the Safety, Toxicity and Misuse of Vitamins and Minerals. This paper deals thoroughly with the vitamins and more particularly with the trace minerals, about which there is less general knowledge and with which there is a greater likelihood of toxicity in view of the small limits of safety. This paper is now in press.

For several years the Consortium has been active in regard to the rapidly growing use of the fake cancer cure Laetrile. This in part has been stimulated by the success of the National Health Federation in accumulating support for the Proxmire Bill which was passed by both Senate and House with a surprising majority. This prohibits the Food and Drug Administration setting maximum limits for vitamins and minerals in supplements and has led to a megavitamin craze that is almost uncontrollable. The Consortium's opposition to the megavitamin craze has taken several forms, the most recent being a brief statement prepared by a member of the Consortium Board, which in simple lay language states the case remarkably well. This is being picked up by the national press, and we are seeing wide use of the information, which should have an important effect upon the consumer who has been misled.

Dr. Victor Herbert of the Bronx VA Hospital has been particularly effective in testifying against the use of Laetrile and has prepared a strong statement that was distributed by the Consortium to the fifty Governors of the States. We have a number of appreciative responses, and the recent Consortium actions against the use of Laetrile are an encouraging indication that the tide may be turning.

The Consortium is now involved in an active effort to develop a program of nutrition education of the public that will meet the public's needs. The first step is to encourage the development of an adequate supply of well-informed nutrition educators who will increase the number of well-informed teachers, who will communicate with the public. This is applicable not only to the program of education in the schools, but also of consumers and of the older population. Mechanisms for this accomplishment are being constructed, and the Consortium will play an important role in this broad program of nutrition education.

It is clear, we believe, that the role of the Consortium in relation to the nutritional status in this country has expanded, with new opportunities and new responsibilities becoming apparent. We believe we have an important role to play in the improvement of the nutritional status of the people of our country.

POSSIBLE NATIONAL NUTRITION CONSORTIUM PROJECTS
(priorities depend on budget)
For discussion September 1978

Program Area #1
Interorganization Coordination

Brief monthly report from NNC Executive Officer to each Board member and to the Executive Officer/Secretary of all member organizations.

Submit bimonthly column about NNC activities to journal or newsletter of all member organizations; submit also to other food and nutrition periodicals.

Coordinate existing programs for answering public inquiries.

Circulate to the NNC Board new position papers and publications of each organization. Assure that each organization knows what publications and papers are being developed by other organizations.

Develop an exhibit booth for annual meetings of member organizations to increase awareness of each organization of the NNC and of the activities and objectives of other member organizations.

Prepare combined listings of publications from member organizations on certain topics of public interest.

Sponsor joint project for National Nutrition Week.

Sponsor special project for the International Year of the Child.

Program Area #2
Nutrition Education

Develop directory of available scholarships and student aid. A lot of this information probably exists in the files of member organizations; NNC already has some of it on file. It needs to be made more accessible to students.

Develop source book regarding public and private funding for nutrition education programs.

Develop and publish brochure for students on Careers in Nutrition and related disciplines (Compilation or packet of existing brochures from NNC member organizations).

Maintain and publicize roster of persons who would be willing to participate in career days or career fairs.

Provide assistance at the state level for the implementation of the USDA Nutrition Education Program

Create a well funded committee to develop and implement a strategy to improve nutrition education of health professionals and paraprofessionals (non-physician education).

Compile information about course requirements in existing graduate programs around the country for degrees related to the disciplines represented in the NNC.

Survey current projected needs for nutrition manpower under various possible changes in funding for nutrition education and nutrition research.

Project Area #3
Public Information Activities

Coordinate existing programs such as the Institute of Food Technologists (IFT) Public Information Program, American Institute of Nutrition (AIN) Public Affairs Committee, Society of Nutrition Education (SNE) regional communicators, and similar groups of other member organizations.

Publicize availability of improved second printing of the nutrition primer, "Nutrition Labeling--How It Can Work For You".

Review plans for proposed position papers of member organization to avoid duplication of efforts.

Develop lay language press releases/statements in response to issues that need a rapid response. We would need a mechanism for quickly researching, drafting, and reviewing such statements with knowledgeable persons in NNC organizations.

Develop media-nutritionist mailing list (both a media person and a nutrition person who know each other) for receipt of press releases and publicity of position papers and publications.

Maintain a roster of speakers with various interests and expertise in nutrition/foods who would be willing to speak to consumer/civic groups; publicize availability of this roster.

Hold a small meeting of six or eight food-nutrition editors for popular magazines and six or eight people trained in foods and nutrition to explore how the NNC can work with them to meet their needs for sound nutrition information. (Might also discuss such a project with a School of Journalism). Additional programs would depend on the results of this pilot project.

Publish (probably from existing camera ready copy) a compilation of book reviews of non-technical publications that have been published in NNC member organization journals. The compilation would be distributed to public and university libraries IN PERSON by someone in nutrition who lives in that community.

Public conferences/workshops on nutrition. Explore sponsoring with groups such as AAUW, LWV, National Foundation March of Dimes, or American Heart Association.

Develop and publish consumer booklets on topics such as "Misuse of Food" or "Diet and Cancer".

Provide technical advice for existing consumer-public newsletters on advances in nutrition research and other topics.

Program Area #4
Public Affairs

Coordinate activities and plans of public affairs persons of NNC member organizations. Be sure each is aware of the other's entire program both in DC and at staff headquarters.

Be sure that NNC Board and appropriate people are on the mailing lists for selected committees and members of Congress.

Maintain up-to-date files of reports and other publications related to nutrition policy issues. Files will be "open" for use in the office but NNC cannot distribute or place orders for others.

Coordinate and stimulate invitations to key Congressional staff and researchers as well as members of Congress to be on programs at NNC member organization meetings and legislative workshops.

Maintain a "Nutrition Witness Briefing Service":

1. Review testimony for style and level.
2. Provide information on special interests of various Committee members.

Develop and publish with the help of Congressional staff a brochure for nutritionists who are invited to testify before a committee (what it's all about, do's and don'ts, written vs. oral testimony).

Develop a newsletter for staffers regarding relevant activities of the NNC member organizations or the broader nutrition community.

Distribute member organization position papers to Congressional staff in cases where interest exists.

Write comment (for Board and Ad Hoc Committee approval) on selected proposed regulations.

Develop a Guide to various nutrition organizations. In non-technical language describe the expertise of each, how they differ, membership requirements, number of members, headquarters, objectives, and other relevant information.

Develop a Legislative Handbook (coordinate with existing handbooks of member organizations) for nutrition people.

Develop a Regulatory Handbook that explains programs of various Executive Agencies and catch phrases such as "Title I" or Section 19.

Washington Seminar in Foods and Nutrition (2-3 days) for graduate students and persons in NNC organizations. Program will describe activities of the legislative and executive branches of the government and explain how persons trained in foods and nutrition might contribute to nutrition policy development. Seminars will be partially funded by registrant but supplemented by special project funds.

Prepare a legislative newsletter to be distributed through or in cooperation with existing communications networks of NNC member organizations.

UNIVERSITY OF WASHINGTON
SEATTLE, WASHINGTON 98195

November 1, 1978

School of Medicine
Department of Medicine

HEAD, DIVISION OF METABOLISM, ENDOCRINOLOGY, AND GERONTOLOGY

Mary Winston, Ed.D.
Nutritionist
American Heart Association
7320 Greenville Avenue
Dallas, TX 75231

Dear Mary:

I have recently had the opportunity to discuss with industry scientists fat modification of a major food component of the diet, i.e., chocolate. As you well know, its fat content is high in saturated fat, mainly in the form of cocoa butter. The chocolate manufacturers have indicated to me that they can modify the milk fat using skim milk solids rather than whole milk, and would be able to label the product "skim milk chocolate". However, the FDA would prevent the modification of the major fat component, cocoa butter, if the manufacturers still want to refer to the product as "chocolate".

As it stands, milk chocolate has approximately (by weight) 10% chocolate liquor, 20% cocoa butter added, 25-27% milk solids including fat, 40-45% sugar, and less than 1% lecithin, vanillin, etc. The chocolate manufacturers would be willing to reduce the amount of added cocoa butter, using either substitution with vegetable fat derivatives or perhaps no substitution at all to reduce total calories if the flavor holds. In view of the position taken by the FDA, there is no incentive for the chocolate manufacturers to want to modify their product. Since in our Diet-Heart Statement deliberations, we would like to urge manufacturers of food products to modify the fat content of their products, I believe this is an example of where we run into a bureaucratic road block. I would be interested in your views as to whether or not the Heart Association should get involved with the FDA regarding the fat modification of foods, as illustrated by this example.

With best wishes,

Sincerely,

Edwin L. Bierman, M.D.
Professor of Medicine

ELB:jld

cc: Dawn Bryan, AHA Public Policy
Dr. Barry Zoumas, Hershey Foods Corp.
Richard T. O'Connell, Chocolate Manu. Assn.

11-6-78

<input type="checkbox"/> Sign & return	<input type="checkbox"/> For appropriate action
<input type="checkbox"/> Comment & return	<input type="checkbox"/> For your information
<input type="checkbox"/> See me about this	<input type="checkbox"/> DO NOT return
<input type="checkbox"/> File	<input type="checkbox"/> Return a copy to me
<input type="checkbox"/> Xerox <input type="checkbox"/> copies	<input type="checkbox"/> Please draft a reply

Memorandum

EXHIBIT H

February 28, 1979



TO: Len Cook
FROM: Mary Winston *Mary*
SUBJECT: HDL HANDBOOK

Dr. Hurley asked me to have the Nutrition Committee review the proposed HDL Handbook developed by Dr. Kannel.

The following is a summary of comments received from members of the Nutrition Committee:

Bierman, E. -- feels that the HDL information for CHD risk assessment is really an epidemiological concern and not a nutrition concern.

Mattson, F. -- proposed handbook is premature. Further evidence in support of the whole concept of HDL-CHD relationship is needed. The extent of knowledge does not support the indicated precision of three significant figures, as in Table I. There is no known method of altering the level of HDL and hence there should be no rush in getting such information into the hands of physicians or laboratories.

Mueller, J. -- fears that its use could be psychologically harmful to an individual patient and could lead to great misunderstandings and frustration on the part of individual practitioners attempting to use it.

The attempt to use finite numbers and mathematical formulae based on observations which may be random and are certainly variable in routine settings may be misleading.

Farquhar, J. -- concerned with the suitability of this piece as an AHA guideline because of the restrictive nature of the subject matter. It is concerned only with the statistics relating HDL cholesterol to the earlier Framingham risk tables. Feels that any AHA guideline on the subject should be more comprehensive, reviewing the evidence for or against the HDL-CHD association being casual (and including non-epidemiological evidence to this point), commenting on factors which may influence HDL cholesterol, mentioning the kinds of chemical analysis which are available and recommended, and establishing some provisional policy guidelines which will aid physicians and other interested professionals in their efforts to deal with the situation.

Len Cook
February 28, 1979
Page 2

Insull, W. -- recommends that this be published by the AHA to supplement the original "Coronary Risk Handbook" (EM 620-PE).

The HDL handbook is compatible with the original risk handbook. The HDL material is very timely as the HDL measurement is widely available and practicing physicians frequently request aid in its interpretation. Although there is no data on how HDL can be favorably altered by treatment, the capability of interpreting HDL effects on an individual's risk is a useful function for the physician and can offer substantial reassurance to patients.

The exact format of the HDL material needs to be carefully evaluated. Preferably, it should be integrated into the original handbook on future printings in order that the final document cover the entire adult age range and both sexes. This integration may require some revision of both documents.

/ij

ADDENDUM - March 6, 1979

Weidman, W. -- when you look at the relative risk tables they provide no further help than the usual statement that HDL cholesterol levels of 35 mg. or less are associated with an increased frequency of coronary heart disease. It seems logical that if hypertension is present, it increases the risk. What concerns us is that the tables, are purportedly based on scientific data; however, the data are certainly tenuous now in regard to HDL cholesterol, particularly when it is above 45 mg. per cent.

Furthermore, according to the handbook, the LDL cholesterol is more predictive; however, he provides a "fudge" factor for its calculation on the basis of total cholesterol and HDL cholesterol; I am concerned that perhaps the data are too soft to come up with such hard numbers.

ADDENDUM - March 8, 1979

Dustan, H. -- thinks it might be a very good idea to have a booklet on HDL-cholesterol that could be mailed out but finds this one to be almost unintelligible. If there were some ways to simplify the information that Dr. Kannel has presented, it would be a good idea to prepare the information for as wide a distribution as is requested.

MEMO FROM

Terry Mitchell

1/28/79

Many - we may have a
good opportunity
to run nutrition psas
soon. Is any thought
being given to developing
some or a new spot for
TV?

Top of the Week

ABC pares more off Saturday morning ad time

By 1981, it will be down to 6-½ minutes; other networks; stay mum, but advertisers call move inflationary

ABC-TV announced last week it would again reduce commercial time in children's programming, cutting it by 20% in two phases beginning next year.

ABC officials called on other networks to follow suit. Neither, however, had any immediate reaction.

The plan did bring quick reaction from Peter W. Allport, president of the Association of National Advertisers, and Peggy Charren, president of Action for Children's Television. They didn't like it.

The National Association of Broadcasters' commercial code now allows 9-1/2 minutes of nonprogram time per hour in Saturday and Sunday morning children's programs.

ABC said it uses 8-1/2 minutes of this allowance now and will reduce it to 7-1/2 minutes beginning next Jan. 1 and to 6-1/2 minutes beginning Jan. 1, 1981. The one-minute hourly allotment to ABC-TV affiliates will remain unchanged.

ABC said the freed network minutes—which are worth about \$20,000 each as commercial carriers—would be used for special messages on nutrition and health, public service announcements or general program information. Queried about the last category, an ABC spokesman said it could include program promotional announcements but that no decision had been made on this point.

With its reduction to 6-1/2 minutes, ABC said, the combination of network and local commercial time in and around

ABC children's shows will be "equal to or at times, less than" total commercial time in prime evening hours. The code now allows 9-1/2 minutes per prime time hour for all nonprogram materials, with an outside limit of 10 minutes per hour.

Announcing the reduction plan last Thursday, ABC officials recalled that they had advocated tighter and tighter commercial limits in children's programming since before the code maximum was first cut from 16 minutes an hour to 12 minutes in 1973, and that as the code limits have gone down, ABC has sold fewer minutes than the code allowed.

ANA's President Allport said he commended ABC's efforts to get more information to children but that this is "a bad approach" that is "without compensating social benefits."

"Fundamentally," Mr. Allport said, "we feel—and feel we can document—that advertising to children is a service to children. Hence any cutback in time devoted to advertising to children is in no way beneficial to children." Other advertising sources estimated ABC would have to raise commercial time costs at least 25% to make up for reduced inventory.

ABC's move came at a time when children's television advertising is under pressure at the Federal Trade Commission and the FCC—a point that did not escape Peggy Charren and ACT, which wants to delete all advertising from children's programs.

Mrs. Charren said she thought it too bad that ABC didn't change its "rules for children" until the FTC and FCC were considering changing the rules for the industry.

She also found other things wrong with the plan: It doesn't go "far enough" or "fast enough," she said, and it overlooks five other weekdays that carry children's programming. She also didn't like the idea that the freed minutes might be used to carry program promos.

More satisfied with the announcement, however, were Senator Ernest Hollings (D-S.C.), chairman of the Communications Subcommittee, and FCC Commis-

sioner Abbot Washburn.

Senator Hollings, who has on his subcommittee's agenda for this Congress a wide-ranging inquiry into children's TV, said he was "pleased to see this sort of constructive action being taken." He added: "It's my hope we will begin to see more initiatives by broadcasters and advertisers that demonstrate a willingness to address the issues."

Mr. Washburn, who noted his call at last year's NAB convention for cutbacks in children's advertising, said he was "delighted" with ABC's move, and he too, said he hoped "the other networks will follow suit."

Stuck between two extremes, FTC may try to compromise on children's TV

San Francisco hearing features hard-line positions of opponents, but there are indications that the commission may back down from trying for an outright ad ban

There weren't exactly fireworks, but the opening round of the Federal Trade Commission's children's advertising hearings got under way last week with more than just snap, crackle and pop.

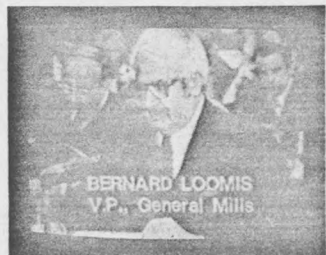
With emotions running high on both sides, the FTC opened proceedings in San Francisco concerning its controversial proposals to limit, and in some cases ban, advertising aimed at children.

By day two, however, reports had surfaced that FTC staff attorneys had given up the idea of an outright ban. Instead, the reports said, the FTC was hoping for some sort of compromise, which might include required public service announcements aimed at the younger audiences.

In Washington, an FTC spokesman sug-



FRED FURTH
Attorney Kellogg Co.



BERNARD LOOMIS
V.P., General Mills



PEGGY CHARREN
Action for Children's TV



NICHOLAS JOHNSON
Citizens Communications Lobby

Golden Gaters. Witnesses for the defense (Kellogg's Fred Furth, General Mills' Bernard Loomis) and prosecution (ACT's Peggy Charren,

Citizens Communications' Nicholas Johnson) before the FTC. Photos by KOED(TV) San Francisco, which carried prime time special on hearings.

gested that a close reading of commissioners' statements in early phases of the case indicate they never really favored an outright ban.

Only three FTC commissioners are participating in the case. Absent from the San Francisco hearings—which conclude this week—is FTC Chairman Michael Pertschuk. Mr. Pertschuk was disqualified from the proceedings by U.S. District Court Judge Gerhard Gesell. Also absent is Commissioner Robert Pitofsky, who disqualified himself because of his work in this area prior to joining the FTC.

Among those testifying against the proposals were toy and cereal manufacturers. Kellogg Co. attorney Frederick Furth denied allegations that that company's advertisements were misleading, and argued that the FTC had no jurisdiction over regulation of TV commercials.

Mattel Inc. attorney Michael Weinstock was even more emphatic. "Our position, simply stated, is that the proposed ban is unconstitutional, economically injurious and unnecessary," Mr. Weinstock said. He added that a ban on toy advertising would increase the cost of toys and decrease the quality and quantity of children's television programming. Mr. Weinstock predicted any such ban could cost Americans an estimated \$840 million a year in increased toy costs.

The issue of constitutionality, along with charges of the FTC's overstepping its regulatory bounds, have been major themes of opponents.

But advocates of the ban believe the commission's actions are justified. They claim industry self-regulation has not worked, and a ban is therefore necessary.

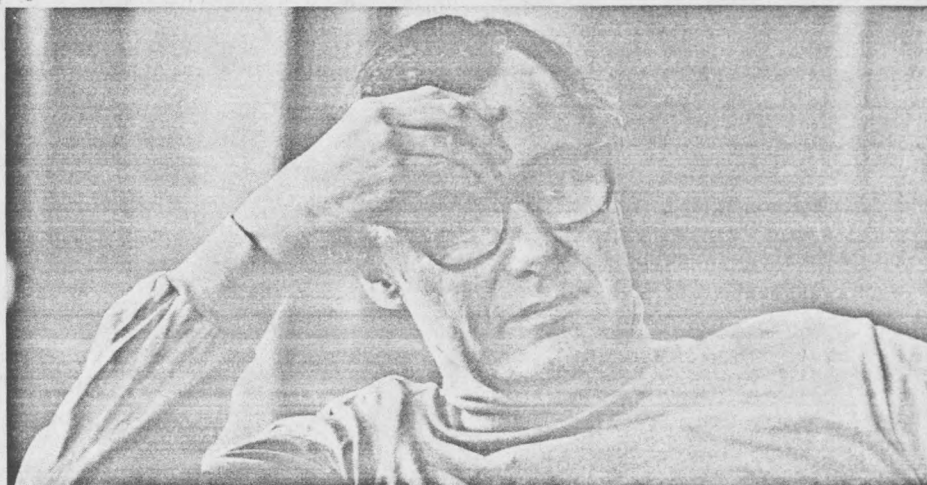
Peggy Charren, president of Action for Children's Television, described industry attempts at self-regulation as a "a disaster." At a news conference last week, Mrs. Charren called for a moratorium on advertising "harmful foods and toys."

"A child's desire for most unsafe toys and harmful foods is created by television advertisements," she said. "If the industry were forced to sell to adults, the products would very likely improve."

Another argument she offered is that children are unable to understand the real intent of an advertisement, that they are unable to distinguish between advertisements and programming, and that they thus become "targets" of the advertising industry.

One psychologist went a step further last week. Professor Robert Liebert of the State University of New York at Stony Brook claimed that a child might begin to mistrust a parent who contradicts the claims of a TV advertisement. An opposing view, however, was offered by one advertising agency executive who was quoted in the *Washington Post* as saying that advertisements were actually beneficial, since they helped a child develop a skeptical eye.

The second round of hearings will begin in Washington March 5 and is scheduled to go five weeks.



Pertschuk: bloodied but unbowed

The FTC chairman, tossed out of the children's TV proceeding, is undeterred from keeping his agency on consumerist course

Michael Pertschuk has a pretty good line on which way his agency is heading: "We're not retrenching."

The Federal Trade Commission chairman, who has been forced to wait in the wings while the hearings on children's TV advertising proceed (facing page) said last week that he felt "comfortable" with the agency's present direction.

"We've whittled away the petty stuff... and strengthened those programs that made sense," he said.

Mr. Pertschuk said in an interview he believes that, under his leadership, the commission is living up to its mandate. "By and large, I think it's important for those of us in this agency to stimulate public debate." He added that to let ideas "germinate secretly" would not be in the best interest of the American consumer.

The commission came under fire last week from groups opposed to the nature of the children's TV inquiry, charging that the FTC was overstepping its boundaries. And while Mr. Pertschuk declined to comment on the pending hearings (a federal judge disqualified Mr. Pertschuk from the case because the chairman had been found to have prejudged the issues [BROADCASTING, Nov 6, 1978]), he did say that actions the FTC have initiated were those he felt to be in the best interest of consumers.

Asked whether it was possible for the commission to remain objective, given its pro-consumer interests, Mr. Pertschuk said it was indeed. "To be a public interest law firm," he said, "means the agency's interest isn't shaped by an economic interest." With that attitude, he added, it is possible to look at the public interest as broadly as possible and still remain objective.

Mr. Pertschuk maintains that even though some view his agency as a collection of liberal, consumer-oriented lawyers, its primary concerns have actually been

quite traditional. Such practices as using information to redress balances and freeing up the marketplace have been the major thrust of the FTC's actions, he said.

"I think we're essentially a conservative agency," he added. "I think those who have observed the agency... have come away with the sense that we don't intervene in the marketplace unless that intervention will help consumers."

He believes that last year's symposium on media conglomerates (BROADCASTING, Dec. 18, 1978) was the type of information-gathering activity that can ultimately prove useful. That meeting, he said, was important because it gave both his staff and the public a chance to better understand a complex and important issue.

He said, however, that the meeting has thus far not resulted in any actions; no cases are pending before the commission, and all media mergers will be examined on a case-by-case basis.

As for the new Congress, the FTC chairman thinks his agency may be in for hard times. "This won't be a Congress friendly to regulation," he said.

But Mr. Pertschuk noted that he plans to make certain there are at least no misunderstandings with the Hill. He said he plans to establish good working relationships with key committee members and keep in close contact.

He has also tried to keep in close contact with his own staff. He said a week spent answering phones in the Chicago office was an interesting, although "frustrating," experience.

These kinds of actions, though, as well as meetings with consumer groups, have allowed him to get a good idea of what's on people's minds.

But he realizes that the marketplace changes, forcing the commission into "a continuous process of re-evaluating priorities."

And his own actions thus far? "I would say that if I started over I'd make all the speeches over again." To which he added, perhaps reflecting on the hearings he had been disqualified from, "Prudence in public debate is a good thing."



EXHIBIT K

STANFORD UNIVERSITY MEDICAL CENTER

STANFORD, CALIFORNIA 94305 • (415) 321-1200 Ext. 6051

STANFORD HEART DISEASE PREVENTION PROGRAM
Department of Medicine, Room S-005
Stanford University School of Medicine

January 3, 1979

Mary Winston, Ed.D., Chief
Nutrition Programs
American Heart Association
7320 Greenville Avenue
Dallas, Texas 75231

Dear Mary,

Jack Farquhar has asked me to join him in responding to your memo of November 28, regarding the Public Education Pieces on Triglycerides, because of my interest in the field and my role on the Executive Committee of the Epidemiology Council. In our opinion, the statement entitled "Elevated Triglyceride Levels" is entirely satisfactory as it presently stands. On the other hand, we would not recommend that the second document, entitled "High Triglycerides" and prepared by the Washington AHA, be distributed by the Heart Association. In our opinion, the latter document goes beyond what is at present prudent for the Heart Association to say about triglyceride in relation to CHD.

With best regards for the new year.

Sincerely,

Steve Hulley

Stephen B. Hulley, M.D., M.P.H.
Associate Professor

cc: Dr. Farquhar

1-8-79

COOKBOOKS:

Jones, J.: Diet for a Happy Heart.
San Francisco, CA: 101 Productions
1975

Jones, J.: The Calculating Cook.
San Francisco, CA: 101 Productions

MacRae, N.M.: How to have your cake
and eat it too! Anchorage, AK:
Northwest Publ. Co., 1975

Better Homes and Gardens: Eat and
Stay Slim. Des Moines, IA:
Better Homes and Gardens Books,
Meredith Corp., 1968

INFORMATION ABOUT ATHEROSCLEROSIS AND
HEART DISEASE, call:

Tel-Med (206) 285-4000 and ask for
the information on file.

American Heart Association of
Washington (206) 285-2415
333 First Avenue West
Seattle, WA 98119

Northeastern Chapter, AHA-W
(509) 838-4163
11 S. Washington Street
Spokane, WA 99204

For more detail about proper diets,
see your doctor or dietitian.

Developed by
Maryann W. Breskin
Undergraduate Program in Clinical Dietetics
University of Washington
for
American Heart Association of Washington
Nutrition Committee

DESIRABLE WEIGHTS

Weight in Pounds According to Frame (In Indoor Clothing)

	HEIGHT (with shoes on) 1-inch heels		SMALL FRAME	MEDIUM FRAME	LARGE FRAME
<i>Men of Ages 25 and Over</i>	5	2	112-120	118-129	126-141
	5	3	115-123	121-133	129-144
	5	4	118-126	124-136	132-148
	5	5	121-129	127-139	135-152
	5	6	124-133	130-143	138-156
	5	7	128-137	134-147	142-161
	5	8	132-141	138-152	147-166
	5	9	136-145	142-156	151-170
	5	10	140-150	146-160	155-174
	5	11	144-154	150-165	159-179
	6	0	148-158	154-170	164-184
	6	1	152-162	158-175	168-189
6	2	156-167	162-180	173-194	
6	3	160-171	167-185	178-199	
6	4	164-175	172-190	182-204	

	HEIGHT (with shoes on) 2-inch heels		SMALL FRAME	MEDIUM FRAME	LARGE FRAME
<i>Women of Ages 25 and Over</i>	4	10	92-98	96-107	104-119
	4	11	94-101	98-110	106-122
	5	0	96-104	101-113	109-125
	5	1	99-107	104-116	112-128
	5	2	102-110	107-119	115-131
	5	3	105-113	110-122	118-134
	5	4	108-116	113-126	121-138
	5	5	111-119	116-130	125-142
	5	6	114-123	120-135	129-146
	5	7	118-127	124-139	133-150
	5	8	122-131	128-143	137-154
	5	9	126-135	132-147	141-158
5	10	130-140	136-151	145-163	
5	11	134-144	140-155	149-168	
6	0	138-148	144-159	153-173	

For girls between 18 and 25, subtract 1 pound for each year under 25.

Metropolitan Life Insurance Company
One Madison Avenue, NY, NY 10010

HIGH
TRIGLYCERIDES?

WHAT DOES
THAT
MEAN?

American Heart Association
of
Washington
333 First Avenue West
Seattle, WA 98119
Phone: (206) 285-2415

1978

WHAT ARE TRIGLYCERIDES?

TRI GLY CER IDES are complex fats. This kind of fat is measured in your blood. It has been made by your body out of foods you have eaten.

CHOLESTEROL is a different fat often measured at the same time. Blood tests for these fats must be ordered by your doctor, who can tell you what they mean.

You must not eat or drink any food for at least 12 hours before such a test.

The TRIGLYCERIDE levels given below are the highest "normal" values accepted. 95% of the population has lower values.

Age	Men (mg%)	Women (mg%)
20-29	155	145
30-39	160	155
40-49	165	165
50-59	175	180
60-	180	190

Data from Bierman and Porte Ann. Intern Medicine, 1968

Excellent

WORRY? NO! DO SOMETHING!

because

WE KNOW THAT

Too many TRIGLYCERIDES increase your risks of atherosclerosis. In this disease, the walls of blood vessels thicken, making the inside smaller. Less blood goes through the smaller vessel, so less oxygen and fewer nutrients are carried to all parts of the body.

WE KNOW THAT

High TRIGLYCERIDE levels, plus high cholesterol levels, cause increased chances of heart disease.

WE KNOW THAT

Diabetes and high TRIGLYCERIDES are related. High TRIGLYCERIDES alone do not cause diabetes.

WE SUSPECT THAT

High TRIGLYCERIDES alone may increase chances of heart disease.

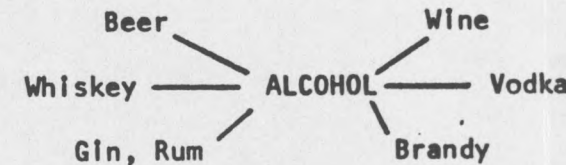
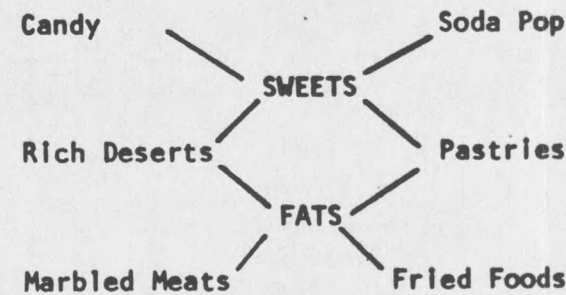
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WHERE DO THEY COME FROM?

Research has shown two groups of people have higher TRIGLYCERIDES than normal. One group comes from families who have inherited higher TRIGLYCERIDES than normal. One group eats more calories than needed for energy. You cannot choose your family, but you can choose what you eat and what you weigh. Most people can lower TRIGLYCERIDES by losing weight. (See the table on the back for YOUR ideal body weight.)

HIGH CALORIE FOODS



WHAT CAN I DO?

DO

Eat a variety of basic foods. Be sure that most of your calories come from grains (bread and cereals), vegetables, fruits, meat, fish and milk. Read labels to find out what you are eating.

DO

Limit ALCOHOL to no more than two servings in one day. One serving means:

- 1 ounce gin, rum, vodka, whiskey OR
- 1 1/2 oz. sweet wine OR
- 2 1/2 oz. dry table wine OR
- 5 ounces beer

DO

Use fish, chicken and turkey as often as you do other meats. They contain less fat and fewer calories.

The fats you eat are of more concern if your CHOLESTEROL level is also high. If this is so, ask your Heart Association for more information.

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Research
Education
Community
Programs



chicago
heart
association

Serving Cook, DuPage and Lake Counties

EXHIBIT L

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1978-1979

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Chicago Heart Association,
incorporated "Not For Profit"
in 1922, is an affiliate of
The American Heart Association.

December 7, 1978

American Heart Association National Center
7320 Greenville Avenue
Dallas, Texas 75231

Attention: Nutrition Committee

Dear Members of the American Heart Association Nutrition Committee,

At a recent Preventive Cardiology meeting held here at the Chicago Heart Association on September 29, 1978, a discussion took place regarding the need for improvement of meals served on airplanes. Many of those who attended this meeting agreed that more airlines should offer foods which are considered "Heart Healthy".

The Chicago Heart Association's Nutrition Subcommittee recommends that American Heart Association's Nutrition Committee investigate possible ways of influencing the airlines to make heart healthy meals available to all passengers. We are sure you will agree that many people would benefit from this worthwhile project.

Thank you for your attention in this matter.

Sincerely,

Therese A. Dolecek

Therese Ann Dolecek, R.D., M.S.
Chairman
Nutrition Subcommittee
Chicago Heart Association

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12-11-78

AHA NATIONAL POLICY GUIDELINES
WITH RELATIONSHIP TO
OTHER AGENCIES IN THE CO-SPONSORSHIP OF PROGRAMS

1. "Co-sponsorship": Support of this degree will require that the Heart Association be involved in the initial planning and development of the Program and will have the full right of approval of the finalized program which is to be implemented. Co-sponsorship may or may not involve budgetary support and/or staff support.
2. "In Cooperation With": Programs which will be listed as "in cooperation with the Heart Association" will also require involvement of the Heart Association in the initial planning and in the final approval of the Program, but will not involve budgetary or staff support.
3. "Endorsement": Programs which are endorsed by the Heart Association will be those which meet the criteria and guidelines established by the appropriate Heart Association Committee for the activity planned, e.g., hypertension screening, but will not involve the Heart Association in the planning or implementation of the program and will not include budgetary or staff support.
4. "Support in Principle": Programs sponsored by outside agencies which do not correspond to ongoing programs in the Heart Association and for which no guidelines have been established, yet which are, in general, in support of the broad objectives of the Heart Association will be designated as supported in principle.

Approved by the Board of Directors - January 28, 1977

NUTRITION WORKING GROUP

PROPOSED NEW INITIATIVE

Title - Dietary Sodium and Its Role in the Occurrence, Management and Prevention of Hypertension

Objectives:

To encourage and support a broad range of investigations including basic, clinical, epidemiological, preventive and therapeutic studies on the role of sodium and related ions in hypertension.

The types of studies which are sought would include:

- 1) The development of improved methodology for determining sodium intake practical for short term and long term studies of hypertension in free living populations. (Included should be consideration of potassium intakes and other related parameters of potential relevance for studies of hypertension. The identification of body tissues which might reflect recent or long term intake should be considered.)
- 2) Metabolic and clinical studies of sodium intake and its relation to blood pressure over a broad range of intake are needed to determine whether there is dose related response or threshold effect.
- 3) Characterization in physiologic and metabolic terms of salt sensitivity, i.e. blood pressure change in response to salt intake. (Included should be studies of potassium, renin-angiotensin, aldosterone and extracellular water.) In addition there should be determination of the prevalence of salt sensitivity in selected U.S. population.
- 4) Studies of the effects of sodium restriction on blood pressure in salt sensitive hypertensive individuals.

5) Clinical studies attempting to characterize the separate and distinct therapeutic effects on hypertension of weight reduction, sodium restriction and hypertensive drugs.

6) Study of the feasibility of achieving practical reductions of blood pressure through moderate restriction of sodium intake in free-living group of individuals with mild and moderate hypertension and not treated with anti-hypertensive drugs.

7) An opportunity is sought to observe a previously isolated unacculturated population with demonstrated low salt intake and to describe changes in prevalence of hypertension which accompany industrialization, modification of conditions of living and increasing consumption of salt in the diet.