Research Needs for Establishing Dietary Guidelines for the U.S. Population (1979)

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RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR THE U.S. POPULATION

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2 Assembly of Life Sciences
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NOTICE

The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the Councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the Committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

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GENERAL INTRODUCTION

The Food and Nutrition Board of the National Academy of Sciences was asked by the National Institute of Arthritis, Metabolism and Digestive Diseases, National Institutes of Health, to identify priorities for research in nutrition seeking new information needed for the development of dietary recommendations for the public.

It became clear in the early discussions that no single recommendation to the public regarding appropriate intakes of energy, protein, fat, carbohydrate. and sodium could be made. Needs for energy and essential nutrients vary with age, sex, physiological state, heredity, and physical activity. The nutritional needs of the young growing infant are distinctly different from those of the inactive octogenarian; those of the vigorous, active young person differ from those of the sedentary, obese person of middle age. Variations in needs due to age and sex are taken into account in the formulation of the Recommended Dietary Allowances. Guidelines for energy sources, protein, and sodium should also be specified by age and sex if they are to be useful. Therefore, many of the research needs identified in this report pertain to problems specific to subgroups of the population for whom unique guidelines are required.

Susceptibility to the chronic degenerative diseases that appear to have nutritional determinants is not uniform throughout the population. Even though heart disease and cancer are the major causes of death in the U.S., deaths have been occurring at increasingly older ages. The age of onset and death from these disabling diseases varies greatly among individuals. For those who are susceptible at an early age, determination of the basis for this susceptibility (hereditary or acquired) and the nature of the basic metabolic defect(s) is critical. The Board

therefore felt it appropriate to emphasize the need for research on the causes of the degenerative diseases as well as the role of nutrition in their possible prevention and treatment.

A major concern of the Board regarding dietary recommendations for the prevention of degenerative disease in the elderly is that relatively little is know about appropriate dietary recommendations for the healthy members of this large segment of the population. This group may be more heterogeneous in its needs than are other groups. Many suffer from deteriorating physiological function and a disproportionate prevalence of general illness, as well as chronic degenerative diseases. Frequently the aged suffer from not one, but several, debilitating conditions. For this group there is a need not only for general dietary information for maintenance of health and possible prevention of diseases but also for more knowledge about the aging process itself.

Although the Board felt that research required to improve the treatment of chronic degenerative diseases was important, this report does not consider such research in detail. Nonetheless, the potential for improving the health of those who are ill by giving careful attention to nutritional needs is emphasized. Many of the major health problems of the U.S. are known to be of multiple etiology. Even if dietary guidelines can be developed that will reduce the incidence, or delay onset, of some of these illnesses, there will be continued need for research to understand all of the causative factors. Recommendations for the possible prevention of diseases for which the cause is not known can rarely be made with confidence.

In seeking to identify research that could provide information needed to assess the validity of specific recommendations concerning diet and disease prevention, the Board recognized 1) that there already exists a considerable body of information about nutrition and maintenance of health; 2) that some recommendations of a general nature, and many that apply to specific population groups, can be made on the basis of currently available knowledge; 3) that nutrition research can provide information needed to enlarge the scope and specificity of such recommendations; 4) that current information about relationships between nutrition and prevention of degenerative diseases is controversial; and 5) that the information required to define better diets for the prevention of some of these chronic degenerative diseases will require additional time and research funds. It is essential to

establish accurately what can and cannot be accomplished through nutrition in the maintenance and improvement of health and in the prevention and treatment of disease.

The following sections deal with the state of knowledge and research needs pertinent to the establishment of dietary guidelines for intakes of total calories, sodium, carbohydrates, lipids, and proteins. The reports are presented separately. In each case research priorities are listed under topic headings. For convenience in use references are placed at the end of each section.

RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR ENERGY

All living organisms require a source of energy for survival. The biological functions for which food energy is needed include growth, maintenance, reproduction, lactation, physical activity and storage of energy as glycogen and fat. Energy is also needed for such metabolic functions as protein synthesis, organ secretions, preservation of ionic gradients and regulation of body pH and temperature. The essential nutrients, plus an adequate caloric intake, are required for an organism to reach its genetic potential with respect to size, weight and physiological function.

Energy may be derived from any reasonable combination of foodstuffs that includes carbohydrate, fat, protein, ethanol and other metabolizable sources of carbon and hydrogen. Although the amount of food energy available per capita to the U.S. population has declined only about 5 percent during this century, there has been some shift in the type of foods consumed[1]. The protein content of the food supply has remained constant at 12 percent of total calories. Total carbohydrate has decreased from 56 percent to 46 percent of calories whereas fat content has increased from 32 percent to 42 percent of calories. Vegetable oils have been the principal contributor to the increase in fat. These oils have displaced complex carbohydrates as a source of energy. Complex carbohydrates, in turn, have declined proportionately from 38 percent to 22 percent of total calories. Consumption of sugars increased during the first part of the century but has remained fairly stable at 24 percent of calories for the past 35 years. The 24 percent includes 6 percent sugars indigenuous to food and 18 percent refined (cane and beet) and processed (corn sugar, syrups, molasses and honey). At present, ethanol contributes an average of 7 percent of calories, although individual consumption varies greatly.

Energy expenditure may be considered either obligatory or discretionary. The obligatory expenditure is determined by the basal metabolic rate which is related to the surface area of the individual and amounts to about 1000 kcal/meter squared per day. This obligatory energy expenditure is equivalent to the amount of energy dissipated as heat due to the performance of internal work, both chemical and physical, and that lost from the body due to the requirement to maintain a temperature above that of the environment. Other less important non-discretionary sources of energy expenditure are associated with the specific dynamic action of food and climatic changes. The discretionary component of energy expenditure in man is that required for work and other activities. It can vary widely, being small for those engaged in light occupations but large for those who perform heavy manual labor[2].

Within each category of "overt work," the energy expenditure among individuals is also variable. Therefore, it would appear that different individuals perform work with different efficiencies. The question whether there is adaptation to increased and decreased energy intake through increased and descreased metabolism, respectively, remains unsettled[3]. With aging, energy requirements decrease because of a modest fall in basal metabolic rate and a general tendency toward less activity.

Modern life and work styles have reduced the amount of energy expended by most people. At the turn of the century, energy expenditure of a typical adult was 3,000 kcal/day. In the 1970s many adults require only 2,000 kcal--indeed, many sedentary females can maintain caloric balance on no more than 1,600 kcal/day. In fact, mechanized aids in the workplace and household, dependence on the automobile, addiction to television viewing and spectator sports have all contributed to the decline of energy expenditure in our country.

During pregnancy, energy requirements are increased to provide calories for the formation of placental and fetal tissues, the increase in maternal tissues, and to meeting the increased work load associated with nourishing and transporting nutrients to these tissues. It is estimated that the gross energy cost of a pregnancy is 80,000 kcal, or approximately 300 kcal/day on the average. Lactation also requires increased energy intake. Breast milk has an energy content of 70 kcal per 100 ml and requires approximately 90 kcal in food energy for its production. Total milk production can reach 1000 ml per day at a cost of 900 kcal, a third of which ordinarily comes from fat

stored during pregnancy. The remaining two-thirds, or about 600 calories per day, must come from increased food intake.

CONTROL OF FOOD INTAKE

Control of food intake is an important physiological process that is mediated by the central nervous system. Hunger and appetite sensations reflect the operation of a regulatory system associated with nerve tracts that pass through the hypothalamus. Many afferent and efferent stimuli are involved in adjustment of food intake to meet energy expenditure[4-6]. It seems likely that the adjustment of caloric intake to work and exercise is most competent in the middle range of energy expenditure and becomes sluggish both at the low and high ends of the scale[7]. It has been postulated that obesity occurs because of failure of this system to match energy intake to energy expenditure. Both chemical and physical signals are considered to activate this regulatory system, but there is no agreement on the identity or relative importance of the various postulated signals. Glucose, fatty acids, hormones, and neurotransmitters have all been suggested. Some investigators believe that body temperature is a critical regulatory factor. There is little agreement on either the nature of the central mechanism of food intake control or on the way in which body composition is controlled.

ALCOHOLISM

Intake of alcoholic beverages is increasing in this country. Although the average intake is about 7 percent of calories (equivalent to 3 ounces of whiskey per day), the range is high (0-40 percent of calories per day). It is estimated that 90 million Americans drink alcoholic beverages and that 9 million may be considered chronic alcoholics. Individuals with alcohol intakes in the range of 10-20 percent of calories on an adequate diet may become obese and very resistant to weight reduction. Severe alcoholics, on the other hand, on even higher intakes of ethanol (40-50 percent of calories), but with reduced food intake, may demonstrate weight loss and other complications of alcoholism.

OBESITY: A PUBLIC HEALTH PROBLEM

Obesity or excessive fatness is the most common form of malnutrition among the well-nourished peoples of the world. It is recognized as a major public health problem because of its association with many serious chronic degenerative diseases. Because depot fat is a physiological constituent of the body the clinical definition of obesity must necessarily be arbitrary. The percentage of depot fat in young adults in the third decade at "desired weight" is 12 percent in the male and 19 percent in the female[8]. Distribution of body weights in healthy adults over 30 is skewed toward the high side, the skewness becoming more prominent in older populations as the prevalence of obesity rises. Normal distributions, however, are approximated in young adults 20-29 years of age. For this age group a range that extended two standard deviations above the mean would include individuals who are 15 percent above desired body weight. The upper limit of this range would correspond to body fat content of 22 percent for males and 29 percent for females. If one accepts values that occur with a probability of only 5 percent as being significantly different from the mean, then these values for body weight and body fat content should be considered as the statistical line for defining obesity[9]. In a study of guinea pigs, Behnke[10] found that approximately 4 percent of lean body weight in males and 7 percent of lean body weight in females is fat-free adipose tissue. This normal complement of fat-free adipose tissue has a maximum capacity of lipid storage that is approximately 20 percent of the body weight in males and 28 percent of the body weight in females. These numbers are in remarkably good agreement with those deduced from statistical considerations of weight distribution among young men and women and provide additional evidence that a desirable population of fat cells might accomodate lipid in amounts equivalent to an increase of 15 percent above desired weight. Although the present state of knowledge of the prevalence of obesity is sketchy and incomplete in our country, data available from the National Center for Health Statistics suggest that the prevalence of obesity rises sharply after age 30, reaching a peak of 39 percent of men and 50 percent of women who are 10 percent or more overweight[11]. This incidence appears to be increasing.

The precise etiology of obesity in man is unknown. The often repeated statement that obesity is caused by the ingestion of more calories than are expended in the same

time period is factual, but is a statement of conditions required for deposition of lipid in fat cells rather than a statement of causes for the deposition. The causes of obesity are obscure. They are intertwined with neuro-humoral and metabolic factors that adjust appetite to energy expenditure as well as with psychological and sociological factors. Their understanding requires much additional research. In times of growth, convalescence, pregnancy and lactation a positive caloric balance is physiological and essential. In well nourished adults, positive caloric balance is unnecessary, undesirable, and can be hazardous. Because energy needs decrease with age and inactivity it is clear that for the elderly an increased fraction of the diet should be provided from foods with a high content of essential nutrients.

Over the past several years two forms of obesity have been described on the basis of adipocyte number and size. The first is of juvenile onset and tends to be characterized by both hyperplasia and hypertrophy of fat cells. The second is of adult onset and is presumably caused by hypertrophy of adipocytes[12]. It has been proposed that the juvenile type occurs in response to overfeeding in childhood[13]. Methodological difficulties have been cited as the basis for questioning the theory of hypercellularity as a cause of obesity[14]. Specific defects in the fat metabolism of adipocytes isolated from obese and nonobese persons have not been established with certainty. Endocrine factors in obesity also require further investigation[15].

It is recognized that obesity in man is associated with significant increases in morbidity and mortality from such chronic diseases as hypertension, diabetes, coronary artery disease, and gall bladder disease[16,17]. Further, insurance companies have established that death rates of the obese from these diseases are, on the average, above those of individuals with normal weights. In middle aged persons who are 25 percent or more above average weight (40 percent over desirable weight) the death rate is 1.7 times normal. In those 40 percent over average weight the death rate increases to 2.2 times normal[18]. Diabetes, hypertension and cardiovascular-renal disease contribute greatly to this increased mortality rate. In all overweight individuals the likelihood of developing diabetes is about 3 times that of normal weight individuals; in those 25 percent or more over average weight the likelihood is 8 times normal.

PREVENTION AND TREATMENT OF OBESITY

Prevention of obesity is more desirable, and maybe easier, than treatment. Unfortunately, there are no satisfactory longitudinal preventive trials. Successful treatment of obesity through diet therapy is achieved in only a low percentage of cases. Stunkard and McLaren-Hume[19] estimate that only 20 percent of obese individuals on diet therapy will lose 20 pounds and only 5 percent will lose 45 pounds. The success rates of organized weight-reducing clubs and exercise programs that attract a large number of persons are not documented by published data. Starvation, with or without protein supplements, may give massive weight loss. This loss is usually not sustained after discharge from a hospital and has, on occasion, brought on sudden death. Ileal-jejunal bypass for morbid obesity is fraught with complications. Schemes of behavior modification show promise but the number of patients studied is small and more information is needed[20].

Genetic and environmental factors interact in producing obesity;[19] the latter might therefore be altered to minimize the genetic predisposition. In view of the fact that a multiplicity of environmental factors may be causative and that individuals may respond variably to each factor, therapeutic programs should be diverse enough to recognize and approach individual needs. A broad-based, interdisciplinary program emphasizing repatterning of both dietary habits and physical activity patterns should be carried out.

It has been reported that blood pressures and plasma lipid concentrations decrease in response to weight reduction programs[17]. It has not been determined, however, whether such decreases reflect temporary restriction in salt, calories, and fat or are a result of the decrease in adipose mass *per se*. The few available studies of the hypotensive effect of weight loss[21,22,23] have failed to resolve the issue of the role of changes in total body sodium versus total body fat.

PUBLIC HEALTH EDUCATION

Obesity represents a personal indicator of an imbalance between energy intake and energy expenditure. Obesity becomes a segregating condition for many who are, or become with excess body fat, more susceptible to the chronic degenerative diseases. Public health education

addressed to weight control through diet and exercise programs may be an effective strategy for controlling the high incidence and mortality rates from these diseases. Although a recommendation for maintaining body weight appropriate for height and body build can be made without additional information, many research questions remain if programs for accomplishing this goal are to be effective.

RECOMMENDATIONS FOR RESEARCH

Obesity and Chronic Degenerative Disease

1. What are the health risks that can be independently attributed to obesity? At what degree of obesity are they sufficient to warrant intervention and to what extent does weight loss reduce the risks?
2. How are diabetes and impaired glucose tolerance related to weight gain and atherogenesis?
3. What is the effect of repeated periods of negative and positive caloric balance on the atherogenic process?
4. What are the mechanisms by which the reduction of weight in the obese individual improves his glucose tolerance?
5. What are the mechanisms underlying the decline of blood pressure that accompanies weight loss?
6. What is the basis of the relationship between obesity, hypertension, and cardiovascular renal disease?
7. Are there unique metabolic differences between the large number of obese persons who do not develop degenerative diseases early in life and those who do?
8. What is the influence of repeated periods of negative and positive caloric balance on calcium balance and the development of osteoporosis?

The Physiological Basis of Obesity

9. What is the true incidence and prevalence of obesity in children and adults?
10. Are there metabolic differences in the efficiency of energy utilization between lean and obese subjects?
11. What is the variability in the capacity of individuals to adapt metabolically to energy imbalance by conserving energy in a situation of caloric deprivation and by expending more in the presence of excess caloric intake?

12. Does the rate of energy expenditure during periods of excess caloric intake differentiate the lean from the obese individual?
13. Are there distinct populations of obese persons that permit classification of individuals according to age of onset of obesity and number of fat cells?
14. What are the mechanisms that control energy storage in the body and regulate food intake? Is thermogenesis important? What are the chemical signals and do they function differently in lean and obese individuals?
15. What is the stimulus for hyperplasia of fat cells as opposed to their hypertrophy?
16. What roles do heredity and nutrition play in determining adipocyte number in human subjects?
17. What is the relationship between maternal obesity and weight gain during pregnancy on fetal outcome and subsequent growth and development of the infant?
18. What is the relationship between total weight gain during pregnancy and postpartum obesity in the mother?
19. Do infant feeding practices influence fat cell number or contribute to later obesity?

Dietary Relationships in Treatment of Obesity

20. Is dietary composition, apart from calorie level, important for the treatment of obesity and how should it be tailored to the individual?
21. Should a comprehensive program for weight reduction employ behavior modification, diet therapy, and exercise? Is it possible to identify in advance those patients likely to be successful in long term weight control?

Relationship of Energy Intake to Other Nutrient Intakes

22. What is the effect of low food intake on the consumption of other nutrients, especially those which are not so widely distributed in foods, such as iron, other trace minerals, essential fatty acids, and vitamins?
23. To what extent does alcohol consumption reduce the intake of other nutrients by various population groups in the U.S.?

REFERENCES

1. Page, L. and B. Friend. 1978. The changing United States diet. BioScience 28:192-198.
2. Durnin, J. V. G. A. and R. Passmore. 1967. Energy, Work, and Leisure. London, Heinemann.
3. Garrow, J. S., ed. 1974. Energy Balance and Obesity in Man. American Elsevier Publ. Co., Inc., New York.
4. Friedman, U. I. and E. M. Stricker. 1976. The physiological psychology of hunger: A physiological perspective. Psychol. Rev. 83:409.
5. Lepkovsky, S. 1973. Newer concepts in the regulation of food intake. Am. J. Clin. Nutr. 26:271-284.
6. Novin, D., W. Wyrwicka, and G. A. Bray, eds. 1976. Hunger--Basic Mechanisms and Clinical Implications. Raven Press, New York. 494 p.
7. Mayer, J., ed. 1968. Overweight; Causes, Cost, and Control. Prentice-Hall, New Jersey. 213 p.
8. Keys, A. and J. Brozek. 1953. Body fat in adult man. Physiol. Rev. 33:245-325.
9. Olson, R. E. 1959. Obesity as a nutritional disorder. Proceedings of the Heinz International Symposium on Nutrition. Fed. Proc. (Symp.) 18:58-67.
10. Behnke, A. R. 1957. Conference on Overweight, National Health Council. Atlantic City, New Jersey, March 6-8, 1957.
11. Abraham, S. and C. L. Johnson. In preparation. Overweight Adults in the United States. Health Statistics. No. 51. National Center for Vital and Health Statistics. DHEW.
12. Hirsch, J. and J. L. Knittle. 1970. Cellularity of obese and non-obese human adipose tissue. Fed. Proc. (Symp.) 29:1516-1521.
13. Knittle, J. L. 1972. Obesity in childhood: A problem in adipose tissue cellular development. J. Ped. 81:1048-1059.
14. Björntorp, P. and L. Sjöstrom. 1971. Number and size of adipose tissue fat cells in relation to metabolism in human obesity. Metab. Clin. Exp. 20:703-713.
15. Albrink, M. J. 1974. Overnutrition and the fat cell. In: P. K. Bondy and L. E. Rosenberg, eds., pp. 417-443. Duncan's Diseases of Metabolism, 7th ed. W. B. Saunders Co., Philadelphia.
16. Keys, A. 1973. Overweight and the risk of heart attack and sudden death. In: G. A. Bray, ed. Obesity in Perspective. Fogarty International Center Series on Preventative Medicine Vol. II, Part II.

U.S. Gov. Print. Off., Washington, D.C. DHEW. NIH Publ.

17. Kannel, W. B. and T. Gordon. 1974. Obesity and cardiovascular disease: The Framingham Study. In: Burland, Samuel, and Yudkin, ed. Obesity. Churchill Livingstone, London.
18. Dublin, L. I. and H. H. Marks. 1952. Mortality among insured overweights in recent years. Transactions. Association of Life Insurance Medical Directors of America. 35:235-263.
19. Stunkard, A. and M. McLaren-Hume. 1959. The results of treatment for obesity. Arch. Int. Med. 103:79-85.
20. James, W. P. T., ed. 1976. Research in Obesity, A Report of DHSS/MRC Group. H. M. Stationary Office, Hillindon Press, Uxbridge, Middlesex.
21. Garn, S. M. and D. C. Clark. 1976. Trends in fatness and the origins of obesity. Pediatrics. 57:443-456.
22. Dahl, L. K. 1958. Salt intake and salt need. N. Engl. J. Med. 258:1152-1159, 1205-1208.
23. Reisin, E., R. Abel, M. Modan, D. S. Silverberg, H. E. Eliahou, B. Modan. 1978. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. N. Engl. J. Med. 298:1-6.

RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR SODIUM

Sodium and chloride ions in physiological concentration and balance are indispensable for many bodily processes including water balance, nerve conduction, heart action, and the function of certain enzyme systems.[1] Both sodium and chloride ions are therefore essential nutrients. The nutritional requirement for sodium for growth and for unavoidable losses from skin and in feces, as measured in metabolic balance studies, for both children and adults is in the range of 4-8 mEq or 100-200 mg of sodium per day. Although there is less information about chloride needs, conservation of sodium and chloride are partly linked. It is believed that chloride is required in more or less stoichiometric relationship to sodium and is conserved by the renal tubule in proportion to conservation of sodium. Average sodium chloride intake by the adult in the United States is estimated at about 10 g per day (range of 4-24 g), which is equivalent to an average daily intake of 4 g of sodium. The average adult intake of sodium ion in this country is therefore 20 to 40 times the nutritional requirement[2].

The relationship of salt to the pathogenesis of hypertension was emphasized as early as 1904 by Ambard and Beaujard[3] and later by Allen[4], Meneely and Dahl[5], and Dahl[2]. According to Freis[6], increased extracellular fluid volume is the initiating factor in a sequence of events leading to chronic hypertension. This has been documented by Ledingham[7], Borst[8], Goyton[9], and Tobian[10]. Likewise, the effectiveness of the rice diet developed by Kempner[11] in controlling hypertension appears to be related to a crucial role in reducing extracellular fluid volume[12,13]. The chlorothiazide diuretics appear to act in a similar manner in controlling hypertension[14,15].

Hypertension is essentially absent in several non-industrialized societies, as is increase in blood pressure

with increasing age. This situation appears to be related to a variety of environmental factors. The arguments favoring low salt intake in prevention of hypertension have been summarized by Freis[6] and Dahl[2]. Studies of hypertension have been made in societies in the Solomon Islands[16], the Amazon basin[17], and the Coco Islands[18]. On the Coco Islands, two groups of Polynesians, the Rarotongans and the Pukapukans, with similar genetic backgrounds were studied. Among the more westernized Rarotongans, sodium intake averaged about 120 mEq per day (2.6 g) and hypertension was common. Sodium intake among the Pukapukans averaged 60 mEq per day (1.3 g) and hypertension was rare. Blood pressure of 160/90 mm or higher was observed in 28 percent of adult Rarotongan males and only 3 percent of the Pukapukan males. When nonwesternized peoples free from hypertension adapt to modern ways of life, including increased salt intake, blood pressures rise and hypertension appears. Dahl[2] found a direct positive correlation between prevalence of hypertension and sodium intake.

There is evidence that sodium chloride intake above the nutritional requirement may permit the development of hypertension in persons with a genetic predisposition for this condition. Studies on animals show that genetically predisposed rats develop hypertension when fed high levels of sodium chloride[19]. Genetic variations in man are large, however, and it has not been possible to demonstrate an association between blood pressure and salt intake within selected populations. Customarily, the sodium excretions of groups of individuals in the U.S.A. with or without hypertension are found to be similar. A susceptible group, therefore, possesses other traits, presumably genetically based, which facilitate the development of hypertension in the presence of sodium. Available evidence suggests that 80 to 90 percent of the U.S. population can tolerate high salt intakes without developing hypertension.

There is a need for concern relative to maintenance of adequate intakes of sodium for growth of infants, and in pregnancy and lactation. In addition, the need for sodium may be particularly acute for persons who are physically active at high temperatures.

ADULTS

The Senate Select Committee on Nutrition and Human Needs[20] recommended that the discretionary adult intake of sodium

chloride in the U.S.A. be reduced to about 5 g per day (equivalent to 2 g of sodium). Since nondiscretionary intake of sodium chloride is about 3 g per day, the daily intake of sodium chloride would be 8 g per day. The Senate Committee recommendations were based on epidemiological observations, already cited, that various isolated populations with very low intakes of sodium have a negligible increase in blood pressure with age and an infrequent occurrence of hypertension among adults, and on the observation of Parijis *et al.*[21] that a reduction in sodium intake is associated with a reduction in blood pressure in some individuals with hypertension.

There has not been an adequately controlled study of adults to compare the effects of long-term daily intakes of 5, 10, or 15 g of salt on blood pressure. Available evidence indicates that sodium restriction to approximately 1.5 g per day (3.8 g salt) will effect a slight reduction (5 mm Hg) in blood pressure among moderately hypertensive adults[21]. Several very carefully controlled studies of severely hypertensive adults have shown that sodium must be restricted to 200 mg (0.5 g salt) per day in order to achieve a significant reduction in blood pressure[11].

It is doubtful that a reduction in discretionary intake of sodium chloride to 5 g per day (total of 8 g per day) will decrease the incidence of hypertension among U.S. adults. The reduction of total sodium chloride intake to 3 g per day or less, the level that may be necessary to prevent development of hypertension in the adult population may be unacceptable within the customary U.S. diet pattern. In addition, the assumption that restriction of salt intake to 3 g per day (equivalent to 1.2 g of sodium) will lead to reduction in incidence of hypertension is based largely on observations in nonwesternized cultures. Of course, the lack of increase in body weight (fatness) with increasing age, which is characteristic of these populations, may be a significant factor in limiting the increase in blood pressure with age and may contribute to the infrequent occurrence of hypertension among these adults.

Hypertension is known to be associated with obesity among adults in developed countries[22]. A number of studies have demonstrated that a decrease in blood pressure is associated with weight reduction, but critics have noted that the weight reduction resulting from calorie restriction was accompanied by sodium restriction. Recently, it was shown that weight loss achieved through diet (calorie restriction without sodium restriction) *was* associated with meaningful reduction in blood pressure[23].

CHILDREN

In a study of school children in Iowa, investigators found no relation between salt taste threshold and preference for salt, nor between threshold or preference for salt and blood pressure. Relative body weight (i.e. weight-for-height) was related to blood pressure and subjects in the highest blood pressure range had the highest relative weight[24].

It is not known whether sodium restriction in children would eventuate in any change in average blood pressure or distribution of population blood pressure levels. There is little information as to whether reduction in sodium intake in infancy and childhood will reduce the individual's chances for developing hypertension in adult life.

Current techniques do not permit identification of children destined to be hypertensive adults. Some preliminary data from longitudinal studies suggest that blood pressure tends to "track" from early childhood. If this were true, an individual child's blood pressure would tend to remain relatively fixed at a given position on the blood pressure distribution curve of the general population for age and sex[25]. One cannot safely conclude from all of the current data, however, that the child whose blood pressure is at the 90th percentile is at any greater risk of becoming hypertensive as an adult than another child of the same age and sex whose blood pressure is at the 10th percentile.

RECOMMENDATIONS FOR RESEARCH

Sodium and Essential Hypertension

1. Is there a level of sodium intake above the nutritional requirement that is *permissive* for the development of hypertension in man?
2. What is the relationship between sodium intake and blood pressure over the range of intakes from 500 mg to 5 gm for hypertensive and normotensive individuals?
3. What are the relative contributions of excess body fat and dietary salt to blood pressure in both normotensive and hypertensive persons?
4. What is the relationship between dietary sodium and potassium intakes in the regulation of blood pressure?

5. Is there a level of intake of sodium below which reduction of intake is without effect on blood pressure of normotensive individuals?
6. Once hypertension is established in the susceptible individual with an intake of about 10 g sodium per day, to what level must intake be limited to reduce mild, moderate, and severe hypertension? In what percent of individuals in the above three groups can salt restriction to the requirement level or any degree above it be therapeutic?
7. Does the amount of sodium intake in childhood affect the incidence of hypertension in adulthood?
8. What is the relationship between intake level of sodium and changes in extracellular fluid volume as a fraction of lean body weight? Is there a sodium threshold for the increase in volumes and, if so, does it vary with the individual?
9. Can a metabolic indicator of *proneness* to hypertension at given salt intakes be detected in children or in young adults?

Sodium and Other Physiological States

10. Can the taste of sodium chloride be altered by use of other flavoring agents?

REFERENCES

1. Select Committee on GRAS Substitutes. 1978. Tentative evaluation of the health aspects of sodium chloride and potassium chloride as food ingredients (Hearing Draft to FDA). Life Sciences Research Office, FASEB (SCOGS-102). 58 p.
2. Dahl, L. K. 1972. Salt and hypertension. Am. J. Clin. Nutr. 25:231-244.
3. Ambard, L. and E. Beaujard. 1904. Causes de hypertension arterielle. Arch. Gen. Med. 1:520-530.
4. Allen, F. M. 1925. Treatment of kidney diseases and high blood pressure. The Psychiatric Institute, Morristown.
5. Meneely, G. R. and L. K. Dahl. 1961. Electrolytes in hypertension. The effects of sodium chloride, evidence from animal and human studies. Hypertension and its treatment. Med. Clin. North Am. 45:271-285.
6. Freis, E. D. 1976. Salt, volume and the prevention of hypertension. Circulation 53:589-595.

7. Ledingham, J. M. 1953. Distribution of water, sodium and potassium in heart and skeletal muscle in experimental renal hypertension in rats. Clin. Sci. 12:337-345.
8. Borst, J. G. G. and A. Borst-de Geus. 1963. Hypertension explained by Starling's theory of circulatory hemeostasis. Lancet 1:677-682.
9. Goyton, A. C., T. G. Coleman, A. W. Cawley, R. D. Manning, Jr., R. A. Norman, Jr., and J. D. Ferguson. 1974. A systems analysis approach to understanding long-range arterial blood pressure control and hypertension. Circ. Res. 35:159-176.
10. Tobian, L., Jr. 1972. A viewpoint concerning the enigma of hypertension. Am. J. Med. 52:595-602.
11. Kempner, W. 1948. Treatment of hypertensive vascular disease with rice diet. Am. J. Med. 4:545-577.
12. Murphy, R. J. F. 1950. The effect of "rice diet" on plasma volume and extracellular fluid space in hypertensive subjects. J. Clin. Invest. 29:912-917.
13. Watkin, D. M., H. F. Fraub, F. T. Hatch, and A. B. Gutman. 1950. Effects of diet in essential hypertension. II. Results with unmodified Kempner rice diet in fifty hospitalized patients. Am. J. Med. 9:441-493.
14. Dustan, H. P., G. R. Cumming, A. C. Corcoran, and I. H. Page. 1959. A mechanism of chlorothiazide enhanced effectiveness of antihypertensive ganglioplegic drugs. Circulation 19:360-365.
15. Wilson, I. M., and E. D. Fries. 1959. Relationship between plasma and extracellular fluid volume depletion and antihypertensive effect of chlorothiazide. Circulation 20:1028-1036.
16. Page, L. B., A. Damon, and R. C. Moellering, Jr. 1974. Antecedents of cardiovascular disease in six Soloman Island societies. Circulation 49:1132-1146.
17. Lowenstein, F. W. 1961. Blood pressure in relation to age and sex in the tropics and subtropics. A review of the literature and an investigation of two tribes of Brazil Indians. Lancet 1:389-392.
18. Prior, A. M., J. G. Evans, H. P. B. Harvey, F. Davidson, and M. Lindsey. 1968. Sodium intake and blood pressure in two Polynesian populations. N. Eng. J. Med. 279:515-520.
19. Meneely, G. R. and H. D. Battarbee. 1976. Sodium and potassium. Nutr. Rev. 34:225-235.

20. Select Committee on Nutrition and Human Needs, United States Senate. 1977. Dietary Goals for the United States, 2nd ed. U.S. Government Printing Office, Washington, D.C. 83 p.
21. Parijis, J., J. V. Joosens, L. VanderLinden, G. Versdreken, and H. K. P. C. Amenj. 1973. Moderate sodium restriction and diuretics in treatment of hypertension. Am. Heart J. 85:22-34.
22. Chiang, B. N., L. V. Perlman, and F. H. Epstein. 1969. Overweight and hypertension: a review. Circulation 39:403-421.
23. Reisin, E., R. Abel, M. Modan, D. S. Silverberg, H. E. Eliahon, and B. Modan. 1978. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. N. Eng. J. Med. 298:1-6.
24. Lauer, R. M., L. J. Filer, M. A. Reiter, and W. R. Clarke. 1976. Blood pressure, salt preference, salt threshold and relative weight. Am. J. Dis. Child. 130:493-497.
25. Task Force on Blood Pressure Control in Children (National Heart, Lung, and Blood Institute). 1977. Report of the Task Force on Blood Pressure Control in Children. Pediatrics (Suppl.) 59:797-820.

RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR LIPIDS

Dietary fat, which is present in foods mostly as triglycerides, is an important source of energy in human diets. Studies of patterns of food consumption in the U.S. since 1900 have shown that whereas calorie availability has declined slightly from about 3500 to 3200 per day per capita, fat intake has increased from 32 percent of calories in 1900 to approximately 42 percent of calories at the present time[1,2]. Large increases in the use of vegetable fats and oils over this period have been responsible for the increase in total fat intake and in fat as percent of calories in the diet. The intake of saturated fat over the 70-year period has been relatively constant, whereas unsaturated fatty acid intake has increased. Oleic acid intake has increased somewhat, but linoleic acid intake has increased most, from 10 grams to approximately 20 grams per day. This shift has resulted in an increase in the ratio of polyunsaturated to saturated fatty acids (P/S ratio) from 0.2 in 1900 to 0.4 in 1970. Other lipids contained in foods in lesser amounts include phospholipids, animal and plant sterols, sterol esters, carotenoids, and fat-soluble vitamins.

The two polyunsaturated fatty acids, linoleic acid and arachidonic acid, which are present in food as esters of glycerol, are known to be required for nutritional well-being. Linoleic acid is the single essential fatty acid for man because arachidonic acid can be formed from linoleic acid in the human body. Linoleic acid occurs in high concentrations in the various edible vegetable oils, e.g., corn, cottonseed, peanut, safflower, and soybean. Linoleate and arachidonate are essential components of animal membranes. Besides being essential for normal membrane synthesis, arachidonic acid is also required for the synthesis of prostaglandins, which regulate platelet activity and

clotting, as well as other physiological activities in kidney, heart, and vascular tissue.

As already indicated, the intake of linoleate by the American population over the past 70 years has doubled. Studies in both human subjects and animals indicate that the required intake of essential fatty acid lies within the range of 1-2 percent of total calories, which is approximately one-fifth of the present dietary intake of linoleic acid by the average American[3].

DIETARY LIPIDS AND DISEASE

Dietary cholesterol has been of particular interest to physicians and epidemiologists because of the demonstration by Anitschkow and Chalatow[4] that feeding cholesterol-enriched diets to rabbits brought on experimental atherosclerosis. The intake of cholesterol by the American population, however, is relatively low compared to the 1.0 percent required to induce experimental atherogenesis in animals. The cholesterol content of the average American diet is about 0.06 percent of the dry weight, which provides about 500 mg of cholesterol per day per capita. Nonetheless, interest in dietary cholesterol as a possible cause of hypercholesteremia and atherosclerosis in man continues. Although the incidence of coronary disease increased from 1930-1955 and is now decreasing, the intake of dietary cholesterol by Americans has not changed appreciably since 1909[2]. In 1909 the average amount of cholesterol in the U.S. food supply was 509 mg per day; in 1950 it was 577, and in 1975 it was 556.

Concern about the relatively high fat content of the American diet has arisen largely from epidemiologic studies in a number of countries. These studies have shown, in general, that rates of atherosclerosis, in particular coronary artery disease, correlate statistically with the levels of dietary fat and cholesterol in the national diets[5]. The significance of the correlation is not clear because of the high incidence of undernutrition and other diseases in many of these countries. The "Lipid Hypothesis," which is one of many hypotheses for the causation of atherosclerosis, states that high dietary lipids lead to high plasma lipids, which in turn initiate and foster atheroma formation in the arterial wall. According to this hypothesis, the strategy for prevention of atherosclerosis would be to lower dietary lipid in the hope of thus lowering plasma lipids sufficiently to retard the generation of atheroma or even enhance its regression.

Three lines of evidence are cited[6] in support of the "Lipid Hypothesis" namely animal experiments, metabolic studies in limited numbers of human subjects, and epidemiological data. Rabbits, hypothyroid rats, and subhuman primates fed diets low in protein and rich in cholesterol and saturated fat develop hypercholesteremia and some manifestations of atherosclerosis. In human subjects on a metabolic unit, it is possible to show that the levels of saturated fat, polyunsaturated fat, and cholesterol in the diet can all affect serum cholesterol levels. Saturated fat in the diet is quantitatively the most important, polyunsaturated fat next important, and cholesterol content least important in this context[7].

Although it has been observed in many studies that plasma total cholesterol is positively related to the risk of subsequent coronary heart disease, the relationship between diet and serum cholesterol in given populations is not precise. With respect to dietary cholesterol, the Framingham study[8] showed no relationship between dietary fat and cholesterol intakes and serum cholesterol levels of 1000 persons whose dietary cholesterol intakes ranged from about 200 to 1500 mg per day. The population, examined by tertiles, shows no correlation between cholesterol intake and serum cholesterol values. Nichols *et al.*[9] observed the same lack of correlation between dietary habits and serum cholesterol in a population of 2000 persons studied in Tecumseh, Michigan.

Much is made of migrant populations that move from an area characterized by one cardiovascular disease risk to an area with a different risk pattern. Keys *et al.*[10] studied indigenous Japanese men in Japan, Hawaii, and Los Angeles, and Caucasians in Hawaii and Minnesota. The three variables--dietary saturated fat, blood lipids, and coronary disease rate--increased from indigenous Japanese to migrant Japanese to the Caucasian group. This evidence argues for the importance of environmental factors, but to what extent these factors are dietary remains to be proven.

INTERVENTION STUDIES

If a given hypothesis of disease causation is sound, a change in putative cause should result in a change in disease rate. Twelve intervention trials have been carried out to test the effect on coronary artery disease rate of modifying serum cholesterol levels by diet or drugs in populations in London, New York City, Oslo, New Jersey,

Helsinki, Los Angeles, and Edinburgh[11-15]. These data have been discussed by Ahrens[16]. These studies, carried out under somewhat different conditions in men ranging from 30 to 60 years of age, for 5-10 years, involving 40,000 man years of study, have shown no effect upon overall mortality. In Los Angeles and Helsinki there was a slight decrease in mortality due to coronary artery disease in the experimental group, but this was offset by an increase in deaths from other causes, particularly cancer. Lowering the serum cholesterol by 6-10 percent by drug therapy for 5 years likewise had no effect upon mortality rate. In a recent World Health Organization trial of clofibrate[17], a drug that reduces lipid synthesis by the liver, involving 30,000 healthy male volunteers, 30-59 years of age, there was an increased mortality from all causes in the hypercholesteremic, clofibrate-treated group. Incidence of nonfatal myocardial infarctions decreased 20 percent in the treated group, but there was a large increase in fatal gastrointestinal disorders and cancer.

LIPOPROTEINS AS RISK FACTORS

Studies of plasma lipoproteins as risk factors have shown that low density lipoprotein levels (LDL) correlate with increased risk but that increased high density lipoprotein (HDL) levels correlate with decreased risk[18]. Very low density lipoprotein and chylomicron levels do not appear to correlate with risk for coronary heart disease.

RECOMMENDATIONS FOR RESEARCH

Diet-Heart Study

1. Will dietary intervention designed to lower serum lipids reduce the morbidity and mortality of coronary artery disease?
2. What is the relative contribution of genetic and environmental variables in determining morbidity and mortality from coronary artery disease?
3. Is there basis for diet intervention in childhood designed to prevent later atherosclerosis?

Lipoproteins as Risk Factors

4. Does the LDL/HDL ratio provide a better indication of risk than the concentration of each alone?
5. Does diet affect HDL concentrations?
6. Do exercise, alcohol, and estrogen reproducibly elevate HDL concentration?
7. Since HDL is a heterogeneous protein composed of HDL_1, (HDL_C), HDL_2, and HDL_3, what is the effect of environment and endocrine factors on the distribution of lipids in these sub-fractions?

Role of Polyunsaturated Fatty Acids (PUFA)

8. What is the biochemical mechanism by which polyunsaturated fatty acids lower serum cholesterol and lipoprotein levels?
9. What are the biochemical effects of trans-fatty acids generated in the hydrogenation of PUFA on serum cholesterol levels, metabolism and atherogenesis?
10. Does total dietary fat intake or the PUFA subfraction of the fat increase the potential of animals or man to develop cancer?
11. Are PUFA toxic to animals or man above a given dietary level? (pro-oxidation and other actions?)
12. Does the polyunsaturated/saturated ratio in the diet affect the influence of dietary cholesterol on serum cholesterol, LDL, and HDL levels?
13. Does the specific composition of dietary PUFA--e.g., eicosapentenoic acid content--consistently affect prostaglandin, thromboxane, and prostacyclin synthesis and influence platelet aggregation and clotting?

Role of Other Dietary Factors

14. Does the amount and type of dietary fiber affect biliary cholesterol, serum cholesterol, LDL, and HDL levels?
15. Are there significant interactions between dietary lipids, vegetables proteins, and fiber in the regulation of serum cholesterol levels?
16. Does amount and type of dietary protein play a significant role in the regulation of the cholesterol levels in man?

17. Does dietary trace mineral intake or specific ratios of these minerals (e.g. zinc/copper) affect serum cholesterol levels in man?

REFERENCES

1. Friend, B. 1967. Nutrients in United States food supply. A review of trends, 1909-1913 to 1965. Am. J. Clin. Nutr. 20:907-914.
2. Gortner, W. A. 1975. Nutrition in the United States, 1900-1974. Cancer Res. 35:3246-3253.
3. Food and Nutrition Board (National Research Council). 1974. Recommended Dietary Allowances, 8th ed. National Academy of Sciences, Washington, D.C., 129 p.
4. Anitschkow, N. and S. Chalatow. 1913. Über experimentelle Cholesterinsteatose und ihre Bedentung für die Enstehung einiger pathologische Prozese. Centraul. Allg. Pathol. U. Pathol. Anat. 24:1.
5. Stamler, J., R. R. Beard, W. E. Conner, V. G. de Wolfe, J. Stokes III, P. W. Willis III, A. M. Lilienfeld, T. R. Dawber, J. T. Doyle, F. H. Epstein, L. H. Kuller, and W. Winkelstein, Jr. 1970. Primary prevention of the atherosclerotic diseases. Circulation XLII, A-55 to A-95.
6. Glueck, C. J. and W. E. Connor. 1978. Diet--coronary heart disease relationships reconnoitered. Am. J. Clin. Nutr. 31:727-737.
7. Keys, A., J. T. Anderson, F. Grande. 1965. Serum cholesterol response to changes in the diet. II. The effect of cholesterol in the diet. Metab. Clin. Exp. 14:759-765.
8. Kannel, W. B., and T. Gordon. 1970. The Framingham Diet study: diet and the regulation of serum cholesterol. DHEW Report, Section 24, Washington, D.C.
9. Nichols, A. B., C. Ravenscroft, D. E. Lanphier, and L. D. Ostrander. 1976. Daily nutritional intake and serum lipid levels. The Tecumseh study. Am. J. Clin. Nutr. 29:1384-1392.
10. Keys, A., N. Kimura, A. Kusukawa, B. Braite-Stewart, N. Larsen and M. H. Keys. 1958. Lessons from serum cholesterol studies in Japan, Hawaii, and Los Angeles. Ann. Intern. Med. 48:83-88.
11. Dayton, S., M. L. Pearce, S. Hashimoto, W. J. Dixon, and U. Tomiyasu. 1969. A controlled clinical trial of a diet high in unsaturated fat in preventing complications of atherosclerosis. Monograph No. 25, American Heart Association, New York.

12. Dewar, H. 1971. A trial of clofibrate in the treatment of ischemic heart disease (five-year study by a group of physicians of the Newcastle upon Tyne Region). Br. Med. J. 4:767-775.
13. Oliver, F. J. 1971. Ischemic heart disease: a secondary prevention trial using clofibrate (report by a research committee of the Scottish Society of Physicians). Br. Med. J. 4:775-784.
14. Stamler, J. 1975. The coronary drug project (clofibrate and niacin in coronary heart disease). J. Am. Med. Assoc. 231:360-381.
15. Turpeinen, O., M. Miettinen, and M. J. Karvanen. 1968. Dietary prevention of coronary heart disease. Am. J. Clin. Nutr. 21:255-276.
16. Ahrens, E. H. 1976. The management of hyperlipidemias: whether, rather than how. Ann. Intern. Med. 85:87-92.
17. Committee of Principal Investigators. 1978. A cooperative trial in primary prevention of ischemic heart disease using clofibrate. Br. Heart J. 40: 1069-1085.
18. Gordon, T., W. P. Castelli, M. C. Hjortland, W. B. Kannel, and T. R. Dawber. 1977. High density lipoprotein as a protective factor against coronary artery disease. Am. J. Med. 62:707-714.

RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR CARBOHYDRATES AND FIBER

Dietary carbohydrates are heterogeneous and consist of polysaccharides, oligosaccharides, disaccharides, and monosaccharides. Starch is the primary carbohydrate and the primary source of energy. The most prevalent sugars are sucrose, glucose, fructose, and lactose. Although the constituent sugars provide the same number of calories per gram as do polysaccharides, the physiologic and metabolic effects of the various carbohydrates can be different. Plant foods also provide fiber--a rather ill-defined group of polysaccharides, pectins and gums. They differ greatly in the extent to which they are degraded in the digestive tract and contribute to the indigestable residue excreted in the stool.

Since 1910 the amount of carbohydrate in the U.S. food supply has fallen from about 500 g to about 380 g per person per day. In 1910, carbohydrates made up 56 percent of the available food energy; today they provide about 46 percent. This decline has been accompanied by a reciprocal increase from 32 percent to 42 percent in the proportion of food energy available as fat. The shift toward less carbohydrate and more fat is the result of reductions in the use of cereal grains and potatoes and an increase in the use of vegetable oils and products[1].

The use of sugars (refined plus naturally occurring) has changed little since the 1920's. By 1976 these sugars represented about half of the total food carbohydrates, whereas in 1909-1913 they represented only about one-third of the then larger total quantity of carbohydrate in the food supply. During the past few years utilization of corn syrups, some of them high in fructose, has been increasing. The syrups are used largely as substitutes for sucrose. As complex carbohydrates have decreased so has dietary fiber in the U.S. food supply. One estimate

suggests that the fiber intake has fallen from about 6 g in 1910 to just over 4 g per person per day in 1974[2].

High sucrose intake has been suggested as a factor in the etiology of diabetes and cardiovascular disease, and diabetes and has long been recognized as a factor in the development of dental caries.

Diabetes mellitus is not a single disease but rather a syndrome with different causes and mechanisms of inheritance[3]. Neither the nature of its inheritance nor the nature of environmental interactions leading to onset of the disease has been clearly established[4]. The type of diabetes mellitus that occurs primarily in adults after age 40 and is not insulin-dependent is strongly associated with obesity and appears to result from alteration in insulin receptors.

It has been mistakenly thought that high sugar intake leads to development of this latter form of the disease. Studies of the relationship between sugar consumption and the incidence of diabetes mellitus have not been convincing. Cohen and associates[5] have selected a strain of rats that develops permanent hyperglycemia after consuming, for a prolonged time, a diet in which over 70 percent of the calories are from sucrose. Experimental diabetes, however, has also been produced in laboratory animals by feeding them diets high in fat and even in protein[6]. Epidemiological studies are not consistent with the hypothesis that high sugar consumption is a cause of the disease. Medalie *et al.*, on the basis of an extensive multivariate analysis, concluded that diet composition was not an important factor in the development of diabetes mellitus[7]. The Pima Indians of Arizona, for example, have a prevalence of diabetes that is among the highest in the world but their intake of sugar is considerably lower than that of the average American[8]. There is evidence that genetic factors are important in the development of diabetes in the Pimas. There is a question about the role of chromium as an etiologic agent in the development of late onset diabetes[9]. The American Diabetes Association places great emphasis on control of diabetes by achieving and maintaining appropriate body weight. This requires avoidance of excessive caloric intake and maintenance of an adequate level of physical activity.

Sugars have also been implicated in development of hyperlipidemia[10], although the triglyceridemias induced by a high sugar intake tend to be transitory. Increased sucrose intake has been associated epidemiologically with the development of heart disease in man, although not as an

independent factor[11]. A diet containing 75 percent of calories as carbohydrate (one-fourth as simple sugar) will lower serum cholesterol and normalize blood glucose levels in some diabetic men[12]. Diets containing sucrose or fructose are more atherogenic for rabbits[13] and baboons[14] than diets containing glucose or starch. Wheat starch is not hypocholesteremic for rabbits but potato, rice, and corn starch are[15]. These observations raise questions about differences among complex carbohydrates.

The subject of sugars in cardiovascular disease has been reviewed in detail by Grande[16], who concluded that the weight of evidence fails to support any direct association between high sucrose intake and the development of coronary heart disease. Connor and Connor, in another review of the subject, concluded "on the basis of the evidence to date--sucrose in the amount usually consumed has no discrete, untoward effects upon hyperlipidemia, atherosclerosis and coronary heart disease"[17].

Since dietary components often interact, the type and amount of fat or protein in the diet may affect utilization of simple or complex carbohydrates. There may be interactions among many dietary components that affect serum lipid concentrations and other metabolic responses.

Dental caries is most prevalent among populations that have the greatest amount of sucrose in their diets. Experimental studies in both man and animals have demonstrated that sucrose is highly cariogenic[18]. Nevertheless, sucrose is not the only cariogenic carbohydrate nor does dental caries have a simple etiology. Not only does dental caries occur in populations that have not consumed sugar but the influence of sugar on the development of the disease depends upon the form in which sugar is fed, the frequency of consumption, and the duration of exposure of the tooth surface. Sticky carbohydrates, in particular, promote dental caries[19].

Resistance of the tooth to invasion by cariogenic bacteria depends on many factors. Total nutritional adequacy is essential for development of sound teeth. Fluoride is important in increasing resistance to dental caries. Crowding of the teeth (more prevalent in Caucasians than in Orientals) may be a factor in that it increases the potential area for lodging and stagnation of food particles. Oral hygiene is also critical in reducing incidence of dental caries. Although elimination of sugar from the diet greatly reduces the incidence of caries, it remains to be established whether moderate reduction in the proportion of sugar in the diet is an effective control measure.

Low fiber intake has been implicated as an etiologic agent in a number of chronic diseases, particularly those of the bowel. Diets throughout the world differ greatly in the amounts and types of fiber they contain. Some populations in which the incidence of coronary disease is low subsist on diets with a high fiber content. Coronary-prone and coronary-free populations, however, differ in many ways other than diet--in life expectancy, level of exercise, stress, smoking, and genetics. Populations whose diets contain relatively large amounts of fiber also have a low incidence of colon cancer, but statistics from the American Cancer Society show that the incidence of colon cancer in the U.S. has been virtually unchanged since 1940. Blot *et al.*,[20] have reported that the Northeast and Midwest are the areas in the U.S. which show the highest incidence of colon cancer. In view of the fact that Americans eat a relatively uniform diet, the data would appear to indict other regional environmental factors.

Much remains to be learned about fiber analysis, fiber composition, and how different fiber components affect nutrient utilization, gastrointestinal function, and metabolic processes. Fiber contains phytates, saponins, and other compounds that may interfere with micronutrient utilization. High fiber diets are reported to reduce the absorption of calcium, magnesium, zinc, and phosphorus[21].

RECOMMENDATIONS FOR RESEARCH

Carbohydrates and Dental Caries

1. What is the effect of a 25 to 50 percent reduction of sugar consumption on the incidence of dental caries?
2. What is the influence of sucrose on incidence of dental caries in relation to local fluoridation of water?
3. What is the influence of tooth crowding and other factors on incidence of caries in relation to sucrose intake?
4. What differences are there between the oral flora of persons who are highly susceptible to dental caries and those who are caries-free?
5. Do carbohydrates differ in their ability to support populations of microorganisms that cause periodontal disease?

Fiber

6. How can the methodologies for analysis of fiber be improved to permit accurate characterization of different fiber components?
7. Does a high fiber intake suppress food intake? At what level is it effective?
8. Do complex carbohydrates and various fibers differ in their effects on serum lipids and lipid metabolism?
9. How do individual types of fiber influence lipid metabolism and blood lipid concentrations?
10. How consistent is the relationship between dietary fiber intake and colon cancer?
11. What environmental factors are associated with a high incidence of colon cancer?
12. How do the type and amount of fiber affect trace mineral balance?

Sucrose and Food Consumption

13. Does early exposure of children to sucrose affect their eating habits as adults?
14. Is sweetness a stimulant for excessive food intake in man? Are food intake responses to sweeteners different in obese and nonobese subjects?

Carbohydrates, Diabetes and Lipid Metabolism

15. Are there differences in insulin response in man and animals to fructose, sucrose, sugar alcohols, and other carbohydrates?
16. What quantities of simple sugars, especially of fructose, are required to produce a significant effect on serum lipids in young and old animals and in human subjects?
17. Do simple and complex carbohydrates induce lipemia to the same extent in normal persons as they do in those with hereditary hyperlipoprotinemias, especially Type IV?
18. Can effects on serum lipids observed with extremely high sucrose diets be reproduced when the diet contains 30 percent or more of mixed fats? When sucrose represents only 15 to 20 percent of calories?
19. What proportion of the population has hyperlipemias (Type III and IV) that are responsive to high sucrose

feeding? Is this condition associated with an abnormally high insulin response to various carbohydrates?

20. What proportion of diabetics respond with improved glucose tolerance to chromium supplements?

REFERENCES

1. Celendar, I. M. 1978. Dietary trends and nutritional status in the United States. Food Techn. 32:39-41.
2. Gortner, W. A. 1975. Nutrition in the United States, 1900-1974. Cancer Res. 35:3246-3253.
3. Craighead, V. E. 1978. Current view on the etiology of insulin-dependent mellitis. N. Engl. J. Med. 229:1439-1445.
4. Cohen, A. M., A. Teitelbaum, S. Briller, L. Yanko, E. Rosenmann, and E. Shafrir. 1974. Experimental models of diabetes. In: H. L. Sipple and K. W. McNutt, eds., pp. 483-511. Sugars In Nutrition. Academic Press, New York.
5. Cohen, A. M., A. Teitelbaum, and R. Saliternik. 1972. Genetics and diet as factors in the development of diabetes mellitus. Metab. Clin. Exp. 21:235-240.
6. West, U. M. 1976. Prevention and therapy of diabetes mellitis. In: D. M. Hegsted *et al.*, eds., pp. 356-364. Present Knowledge In Nutrition. The Nutrition Foundation, Inc., New York.
7. Medalie, J. H., C. M. Papier, U. Goldbourt, and J. B. Herman. 1975. Major factors in the development of diabetes mellitus in 10,000 men. Arch. Intern. Med. 135:811-817.
8. Bennett, P. H., N. B. Rushforth, M. Miller, and P. M. LeCompte. 1976. Epidemiologic studies of diabetes in the Pima Indians. Recent Prog. Horm. Res. 32:333-376.
9. Kiu, V. J. K. and J. S. Morris. 1978. Relative chromium response as an indicator of chromium status. Am. J. Clin. Nutr. 31:972-976.
10. Macdonald, I. 1966. Lipid responses to dietary carbohydrates. Adv. Lipid Res. 4:39-67.
11. Yudkin, J. and J. Morland. 1967. Sugar intake and myocardial infarction. Am. J. Clin. Nutr. 20:503-506.
12. Kiehm, T. G., J. W. Anderson, and K. Ward. 1976. Beneficial effects of a high carbohydrate, high fiber diet on hyperglycemic diabetic men. Am. J. Clin. Nutr. 29:895-899.

13. Kritchevsky, D., P. Sallata, and S. A. Tepper. 1968. Experimental atherosclerosis in rabbits fed cholesterol-free diets. II. Influence of various carbohydrates. J. Atheroscler. Res. 8:697-703.
14. Kritchevsky, D., L. M. Davidson, I. L. Shapiro, H. K. Kim, M. Kitagawa, S. Malhotra, P. P. Nair, T. B. Clarkson, I. Bersohn, and P. A. D. Winter. 1974. Lipid metabolism and experimental atherosclerosis in baboons: influence of cholesterol-free, semi-synthetic diets. Am. J. Clin. Nutr. 27:29-50.
15. Hamilton, R. M. G. and K. K. Carroll. 1976. Plasma cholesterol levels in rabbits fed low fat, low cholesterol diets. Effects of dietary proteins, carbohydrates, and fibre from different sources. Atherosclerosis 24:47-62.
16. Grande, F. 1974. Sugars in cardiovascular disease. In: H. L. Sipple and K. W. McNutt, eds., pp. 401-437. Sugars in Nutrition. Academic Press, New York.
17. Conner, W. E. and S. L. Conner. 1976. Sucrose and carbohydrate. In: D. M. Hegsted, *et al.*, eds., pp. 33-42. Present Knowledge In Nutrition. The Nutrition Foundation, Inc., New York.
18. Select Committee on GRAS Substances. 1976. Evaluation of the health aspects of sucrose as a food ingredient. Life Sciences Research Office, FASEB (SCOGS-69) (NTIS No. PB262), 30 p.
19. Gustafsson, B. E., *et al.*, 1954. Vipeholm dental caries study; effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for 5 years. Acta Odontol. Scand. 11:232.
20. Blot, W. J., J. F. Fraumeni, Jr., B. J. Stone, and F. W. McKay. 1976. Geographic patterns of large bowel cancer in the United States. J. Nat. Cancer Inst. 57:1225-1231.
21. Reinhold, J. G., B. Faradji, P. Abadi, and F. Ismail-Beigi. 1976. Decreased absorption of calcium, magnesium, zinc and phosphorus by humans due to increased fiber and phosphorus consumption as wheat bread. J. Nutr. 106:493-503.

RESEARCH NEEDS FOR ESTABLISHING DIETARY GUIDELINES FOR PROTEIN

Protein requirements of adults have been investigated extensively over many decades. A basic problem in establishing recommended dietary allowances for protein is that, owing to differences in their amino acid compositions, food proteins differ in the efficiency with which they are utilized by the body. The subject has been reviewed in detail by a Committee on Amino Acids of the Food and Nutrition Board[1] by an Expert Panel of the Food and Agriculture and World Health Organizations (FAO/WHO)[2] and by the Committee on Dietary Allowances of the Food and Nutrition Board[3]. These committees concluded that not more than 0.8 g of protein of good quality per kg of body weight was required to meet the daily protein needs of those healthy adults with the highest requirements. Protein needs of infants have also been studied extensively. Recommended intakes of protein of the quality of milk proteins of 2 to 2.5 g/kg of body weight at birth, falling to about 1.5 g/kg by one year of age, are considered soundly based and well-established. Recommended intakes for intermediate age groups, for pregnancy, and for lactation have been estimated by interpolation from these values and from knowledge of growth rates, of fetal development, and of milk production and composition.

It has been recommended in the United Kingdom for many years that dietary protein should be obtained from a mixture of animal and vegetable sources that will ensure good quality and in an amount that represents 10 percent of total calories[4]. The figure of 12 percent of caloric intake, suggested as appropriate in the Senate Select Committee's Dietary Goals[5], is closely in line with the amount present, on the average, in U.S. diets. If the protein in the diet is of good quality and food intake is adequate to meet energy needs, 12 percent of calories

(about 90 gms) from protein will considerably exceed protein requirements. In fact, average intakes of protein in the United States are well above amounts considered adequate to meet nitrogen and amino acid needs[1].

There is no evidence to suggest that either inadequate or excessive intake of protein is a general public health problem in the United States, even though the consumption of protein by individuals can range widely (40-150 gm per day). Low intakes of protein are usually associated with low caloric intake, which may result from adherence to weight reduction regimens, illness, neglect, or social maladjustment.

Although there is little difficulty in making generally acceptable recommendations for dietary guidelines for protein intake for various age groups, many questions about protein nutrition remain inadequately investigated. Questions about tolerance of high protein intake persist. Evidence that a high protein intake leads to an increase in urinary calcium excretion, without a corresponding increase in calcium absorption, has led to concern that a high protein intake may result in negative calcium balance in some adults in the U.S.[3]. Loss of renal function associated with increasing age has been observed[6] and raises a question as to the optimum protein intake for the elderly. Observations that certain plant proteins have a serum cholesterol-lowering effect merit further investigation[7].

Protein-trace nutrient relationships have received some attention in the past but have not been thoroughly investigated. The influence of different food protein sources on availability of essential nutrients from other foods is of interest, particularly in relation to iron absorption[5]. Evidence that brain concentrations of neurotransmitters can be influenced by blood amino acid patterns and concentrations, which in turn can be influenced by amino acid intake[8], has opened new avenues for investigation of relationships between diet and behavior.

Many people in the U.S. follow weight-reduction regimens and, for considerable periods of time, are in a state of energy deficit. This raises concern about nitrogen retention. There is a need to know whether the proportion of calories from protein should be increased as caloric intake falls. There is also a need to know whether or not protein utilization is less efficient when caloric intake decreases during aging[9].

The proportion of animal to vegetable protein in the U.S. food supply has increased significantly during the

past 70 years. Animal protein is of higher quality and more readily digestible than plant protein. Also, sources of animal protein provide vitamin B_{12}, trace minerals, and iron in highly available forms[5]. Currently, many new food products are entering the market place. It is not known whether digestibility of the proteins in these products and the biological availability of the trace nutrients they contain are as high as those from more traditional sources.

Whereas protein research is not necessary for developing general dietary guidelines, a number of issues relative to protein nutrition for the population as a whole or for unique groups within the population merit investigation. These issues are listed below.

RECOMMENDATIONS FOR RESEARCH

Proteins and Serum Cholesterol

1. Do certain amino acid patterns in dietary proteins alter plasma lipoprotein synthesis?
2. Does the type of protein consumed affect the concentrations of blood lipids?
3. Are there unrecognized constituents associated with plant proteins that affect cholesterol transport or synthesis?
4. How significant are the effects of plant proteins, as a component of a complex mixed diet, on plasma cholesterol concentrations and on lipoprotein patterns?

Interactions between Protein and Trace Minerals

5. How significant is the effect of animal proteins in increasing absorption of iron from other foodstuffs?
6. Do animal proteins affect the absorption of trace minerals other than iron from foodstuffs?
7. What is the relationship between protein intake and the utilization of magnesium and trace minerals?

Protein Needs and Urinary Calcium Excretion

8. What is the underlying basis for the increased urinary calcium loss associated with high protein intake as noted in short term experiments?

9. What factors may alter this loss?
10. Do people adapt to high protein intakes, such that the excess urinary calcium loss is only transitory?

Protein Needs of the Elderly

11. What are the effects on nitrogen retention of protein intakes in excess of protein needs or of low protein and calorie intakes?
12. How much do disease, immobility, and deterioration of body function influence protein requirements?
13. Does increased protein intake influence rate of recovery from illness?

Amino Acids and Brain Function

14. Do marginal intakes of amino acids affect mental development and behavior?
15. Over what range of protein intakes can brain neurotransmitter concentrations be modified?
16. Can specific behavioral changes be related to changes in neurotransmitter concentrations?
17. Do changes in brain amino acid concentrations affect behavior directly instead of through mechanisms not related to changes in neurotransmitter concentrations?

Assessment of Protein Quality

18. Is it possible to develop reliable and acceptable methods for assessing protein quality from the amino acid composition of proteins that will predict accurately the amount of protein needed to meet human protein requirements?
19. Can simple but satisfactory methods be developed for measuring the biological availability of amino acids in proteins that have been processed in various ways in order to predict the efficiency of protein utilization by amino acid scoring?
20. Can protein adequacy be estimated accurately from knowledge only of lysine, sulfur-containing amino acids, and tryptophan intakes?
21. Do the quantities of hemaglutinens, protease inhibitors and similar substances present in many high protein foods (particularly legumes) cause significant adverse effects in humans?

Energy Intake and Protein Utilization

22. How far can caloric intake fall below the requirements for maintenance or growth without affecting amino acid utilization? Before impairing utilization?
23. What is the extent of retention of nitrogen from protein consumed with low calorie reducing diets? To what extend does increasing the protein content of reducing diets reduce body protein wastage?
24. What is the upper limit of protein intake beyond which no further improvement of protein utilization occurs when caloric intake is restricted by 10 to 50 percent? Is this limit the same for chronically obese individuals as for individuals who are only moderately overweight?

Protein Needs During Pregnancy

25. How well defined are the protein and energy needs of the young, pregnant girl who is still growing?
26. Is some of the protein consumed in excess of maintenance needs early during gestation stored and used later when protein demands of the fetus are high?
27. Is there a relationship between protein intake and the toxemia of pregnancy?

Adequacy of Protein Intake of the U.S. Population

28. Are there groups of people within the population who have habitually low or inadequate intakes of protein? Are there groups with habitually very high intakes of protein?
29. What is the range of protein intake of those over 65?
30. Do chronic illnesses that do not require hospitalization result in inadequate protein intakes?
31. Are there significant numbers of people in the U.S. who have adequate energy intakes and inadequate protein intakes?
32. To what extent do vegetarians and others who restrict their consumption of animal products have inadequate protein intakes?

Protein Needs and Serious Illness or Malnutrition

33. There are a number of questions concerning the treatment of patients with serious diseases and the worldwide problem of malnutrition. Answers to these questions may eventually have important implications for dealing with public health issues:

- Is modification of protein intake of value in the treatment of cancer and other major degenerative diseases?
- Can the amino acid composition of solutions for total parenteral nutrition be modified to alter substantially the efficiency of amino acid utilization?
- Is the tolerance for protein exceeded if large amounts of protein are provided in an effort to reduce body wasting that occurs as the result of trauma and infection?
- How do marginally inadequate protein and energy intakes during infancy and early childhood affect immunocompetence and mental function?

REFERENCES:

1. Food and Nutrition Board (National Research Council). 1959. Evaluation of protein nutrition. National Academy of Sciences, Washington, D.C. (Pub. No. 711) 61 p.
2. Food and Agriculture Organization/World Health Organization. 1973. Energy and Protein Requirements. WHO Tech. Rep. Ser. No. 522, FAO Nutr. Meetings Rep. Ser. No. 52, FAO/WHO Geneva.
3. Food and Nutrition Board (National Research Council). 1974. Recommended dietary allowances, 8th ed. National Academy of Sciences, Washington, D.C., 129 p.
4. Department of Health and Social Security. 1969. Recommended intakes of nutrients for the United Kingdom. Reports on Public Health and Medical Subjects, No. 120. Her Majesty's Stationery Office, London.
5. Senate Select Committee on Nutrition and Human Needs, United States Senate. 1977. Dietary Goals for the United States, 2nd ed. U.S. Government Printing Office, Washington, D.C. 83 p.
6. Shank, R. C. 1976. Nutritional characteristics of the elderly--an overview. In: M. Rockstein and M. L.

Sussman, eds., pp. 9-28. Nutrition, Longevity and Aging. Academic Press, Inc., New York.

7. Kritchevsky, D. 1978. Vegetable protein and atherosclerosis. J. Am. Oil Chem. Soc. 56:135-146.
8. Fernstrom, J. D. 1977. Effects of the diet on brain neurotransmitters. Metab. Clin. Exp. 26:207-223.
9. Harper, A. E. 1978. Recommended dietary allowances for the elderly. Geriatrics 33:73-80.