

SHOULD THERE BE INTERVENTION TO ALTER SERUM LIPIDS IN CHILDREN?

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In recent years considerable study and discussion has focused on the role of diet in the pathogenesis of atherosclerosis. In 1984 a consensus development panel was convened by the National Institutes of Health in an effort to bring together available information on this topic and to resolve some of the controversial issues. The findings of the panel and its recommendations were published in 1985 (14). The panel concluded that sufficient evidence from several types of studies linked dietary fat and cholesterol to the risk of atherosclerosis. They therefore made dietary recommendations that are appli-

cable to all of the American public over the age of two years. These recommendations include limiting fat intake to 30% of total calories with 10% or less from saturated fat, 10% from monounsaturated fats and less than 10% from polyunsaturated fats. The total daily cholesterol intake should be limited to 100 mg per 1000 calories, not to exceed 300 mg as a daily total. The conclusions of the panel did not meet with universal acceptance. In particular, reservations were expressed concerning the application of the dietary recommendations to children and adolescents (3, 86).

The purpose of this paper is to review the evidence for and against the need for dietary intervention in children and adolescents to reduce the risk of atherosclerosis and its consequences for adults. Because these dietary interventions are designed to have their beneficial effects in adulthood, it is particularly important that they not have negative effects for the growing child and developing adolescent. The various studies on the role of children's diets in predisposing to the risk of atherosclerosis in adulthood are evaluated. It is important to assess the information available concerning current dietary practices in children. The available information concerning blood lipid values in American children and adolescents, the relationship of these values to dietary patterns, and their utility in predicting adult lipid patterns is reviewed. Studies on dietary interventions in children and adolescents are few in number. Available information is assessed and interventions are evaluated for efficacy and potential harmful effects. Potential areas for future research are discussed.

Background Information Supporting Intervention

The role of serum lipids and cholesterol as risk factors in the pathogenesis of atherosclerosis and coronary heart disease has been extensively explored in recent reviews (35, 43, 79) and position papers (38, 45). In the United States the results of three large clinical studies are the primary basis for the recommendation of dietary intervention to reduce the risk of morbidity and mortality from coronary artery disease (4, 50, 51, 63). Based on the results of the consensus development panel mentioned above (14), a national cholesterol education program was begun (90b) with the intent of changing the perspectives of both physicians (83) and the public (82) on cholesterol and heart disease. A continuing series of articles, commentaries, and editorials make it clear, however, that a true "consensus" on the matter of dietary and even drug intervention in adults to reduce the incidence of coronary artery disease has not yet been achieved (2, 3, 6, 18, 19, 26, 37, 41, 47, 59, 70, 72, 74, 88, 89, 93). It is not the purpose of this review to explore the issues involved in recommendations of dietary and drug intervention in adult atherosclerosis. The various aspects of this issue are well developed in the references listed above.

The risk of developing atherosclerosis in adulthood is thought to be

influenced by diet and serum lipid patterns in early life. Thus the recommendations for dietary intervention in all Americans over the age of two (14, 92, 96). The data supporting the role of diet and serum lipids in childhood as risk factors for adult atherosclerosis have been recently and extensively reviewed (29, 44, 58, 67, 69, 80, 95). Support for the contention that the risk of adult atherosclerosis begins in childhood has been drawn from a variety of sources including comparative population studies, evaluation of arterial changes in children and young adults, the relationship of diet and serum lipids, family studies, and evidence that serum lipid patterns in children predict adult values.

Population Studies

Knuiman and coworkers (40) have reported a population study measuring serum total and HDL cholesterol concentrations in seven- and eight-year-old boys from sixteen countries. The determinations were done in one laboratory and a standardized protocol was used for the drawing of blood and the preparation, storage, and transport of the serum. The sixteen countries were selected from Africa, America, Asia, and Europe. They were chosen because of different rates of mortality from coronary heart disease for men aged 55–64. The coronary heart disease data were obtained for 1974 and 1975. The cholesterol determinations were done on blood drawn in 1978 and 1979. The results of this study indicated that serum total cholesterol concentration is lower in developing countries of Africa and Asia and higher in the developed countries of Europe and the United States. The lowest values were found in three West African countries (Ghana, Ivory Coast, and Nigeria), intermediate values were found in the Philippines, Greece, Portugal, and Hungary, and the highest total serum cholesterol concentrations were found in the United States and other European countries. The HDL cholesterol accounted for about one third of the total cholesterol values except in boys from Pakistan and the Philippines in whom it represented one fourth of the total value. The cholesterol values were also directly related to the availability in the various countries of animal product food expressed as a percentage of the total energy supply per capita.

It is tempting to conclude from this and similar population studies that an association between diet and serum cholesterol values in children is reflected in the rates of mortality from coronary heart disease in adult men. Conclusions from such population studies, even those as well controlled as the one mentioned above, must be tempered with several reservations. The mortality figures for coronary heart disease were derived from the years 1974–1975, and the serum cholesterol values and inferred dietary information were obtained from seven- and eight-year-old boys in 1978–1979. It is impossible to know the comparability of diets during childhood for men at risk of coronary heart disease to the diets of boys growing up during the

1970s. In some countries, war-related deprivation could have significantly influenced the diet of men who would have been ages 55–64 during the years 1974–1975. Therefore it is difficult to know the degree to which current dietary patterns and serum cholesterol values can be used to make an association with adult coronary heart disease. Furthermore, there are other reasons that changes in nutritional status among children in these countries are likely to have taken place. In the United States secular trends in growth and maturation have occurred (77) as a consequence of improved nutritional status (52). Similar secular trends had been noted in other countries as well (42). The secular trends in the United States have occurred during a time of progressive decrease in atherosclerotic heart disease (90a). Furthermore, in these population studies no data are available relating the diets of the individuals in the study to their serum cholesterol values, which show considerable variation within the population (40). For all of these reasons the population studies can be regarded as suggestive but far from conclusive in attempting to associate serum cholesterol values, diet during childhood, and the risk of coronary heart disease during adulthood.

Other potential confounding factors are illustrated by a recent description of a correlation between weight in infancy and death from coronary heart disease (5). In this study, the relationship between weight at birth and at one year of age to the risk of death from ischemic heart disease was evaluated in 5,654 men, and an inverse relationship between weight in infancy and the risk of death was found. The authors conclude that impaired growth and development in prenatal and early postnatal life maybe an important risk factor for ischemic heart disease. The study was done in part to explore the reason for the observation that in Britain the highest rates of coronary artery disease are in the poorest areas and lowest income groups. The study does point out the difficulty in drawing conclusions from population studies in which all of the potentially variable environmental factors during childhood that influence the risk of subsequent coronary artery disease as an adult cannot be identified and controlled.

Vascular Lesions During Childhood and Adolescence

Various observations have been interpreted as indicating that atherosclerosis has its onset in childhood (29, 54, 67, 78, 79, 85). Fatty streaks have been found in arterial vessels of children older than age ten years and are thought to be precursor lesions for atherosclerosis (54, 78). Fatty streaks are present, however, in the aorta of virtually every child by the age of ten years regardless of race, sex, or environment (79). The relationship of these fatty streaks to the formation of fibrous plaques, which is the most characteristic lesion of advancing atherosclerosis, remains uncertain and a subject of controversy (85). Although the distribution of fatty streaks in children is ubiquitous among all the world's population, a similar distribution of fibrous plaques has not

been observed. Therefore, the contention that fatty streaks in arterial vessels are evidence for the childhood origin of atherosclerosis is difficult to accept. Certainly, no evidence indicates that diet is an associated factor or that the onset or extent of fatty streaks can be ameliorated by dietary intervention. Tejada et al (88a) found that fatty streaks were universal in aortas of children from Guatemala, Costa Rica, and Louisiana. Plaques did not appear in children until age 15, and no differences were seen in the degree of atherosclerosis until age 30.

Newman and his coworkers (67) evaluated the relationship of serum lipoproteins and systolic blood pressure to early atherosclerosis in thirty five individuals who died at a mean age of eighteen years. Fatty streaks in the aorta were related to antemortem levels of both total and low density lipoprotein cholesterol; coronary artery fatty streaks were correlated only with the very low density lipoprotein cholesterol. However, no correlation was found between serum lipoprotein levels and the presence of fibrous plaques in the coronary arteries; these plaques showed a weak relationship (p-value of 0.09) only to systolic blood pressure. No information is available concerning dietary patterns in this group of individuals. In other studies no correlation between diet and cardiovascular disease risk factor variables has been found (22, 91).

Atherosclerotic coronary artery disease has been found in apparently healthy young American men dying in battle during the Korean (17) and Vietnam (56) wars. This is to be expected if atherosclerotic plaques begin to form in males at puberty (88a). No information is available concerning the dietary patterns of those with or without coronary artery lesions. Furthermore, there is no additional information concerning other possible clinical contributing factors such as genetic predisposition, smoking habits, serum lipid patterns, and so forth. It is of interest that the frequency of atherosclerotic lesions in servicemen decreased from a value of 77.3% in the Korean experience (17) to 45% in the Vietnam study (56). This finding is consistent with the steady decrease in number of deaths due to heart disease and stroke that has been observed in the United States since 1950 (90a).

In summary, the evidence for atherosclerotic heart disease beginning in childhood is inconclusive. The significance of fatty streaks as a potential precursor lesion for fibrous plaques remains uncertain. The relationship between coronary artery fibrous plaques, diet, and serum lipid patterns is yet to be established.

Diet and Serum Lipid Relationships

As noted above, serum lipid patterns in children vary among different populations with presumably different dietary patterns. If dietary interventions are to be proposed in an effort to alter serum lipids in children, it will be important to determine the degree to which a relationship exists. Several

studies concerning the relationship are available for school age children (22, 24, 32, 62). One group has studied serum lipids, lipoproteins (24) and dietary intakes (68) in preschool children, but unfortunately no attempts at any correlations were reported. In the studies by Weidman and his coworkers (91), 103 healthy white school children between the ages of 6 and 16 were evaluated, and no correlation was found between serum cholesterol levels and the daily amount of total calories, cholesterol, fat, saturated fat, or sugar in the diet. Frank and coworkers (22) studied food intakes and eating patterns in 185 ten-year-old children to evaluate their relationship to arteriosclerosis risk factor variables including total cholesterol and lipoprotein values. Correlations could not be established in individual children but, similar to population studies, when serum cholesterol values were grouped as low, intermediate, or high correlations showed that the children who were below the twenty-fifth percentile for serum cholesterol consumed less fat. In this small sample, therefore, these results resemble the larger population studies, according to countries, reported above. Two large surveys based on lipid research clinics (32, 62) reported results in a total of 2,903 children, ages 6 to 19 years. Dietary intake was assessed by random recall and compared with serum cholesterol, triglyceride HDL, LDL, and VLDL cholesterol values. Negative or inconsistent weak associations were found in these two studies.

The difficulty in associating diet with serum lipid patterns in individuals even in these large studies may in part be related to the 24-hour dietary recall model. A high level of variability from day to day in dietary intake is likely. Statistical associations may be difficult to make because of multiple independent variables, day-to-day variance in lipoprotein levels, and problems in obtaining accurate nutrient histories. (68) Another important factor, however, may very well be the individual variation in serum lipid responses to such dietary components as cholesterol (8, 55).

Genetic Predisposition

Children of parents who have significantly increased or decreased levels of serum cholesterol and triglyceride are themselves more likely to have similar serum lipid value distributions (61). Children born to fathers who have had coronary artery disease at a young age are likewise at increased risk for having abnormally increased serum lipoproteins (48). These are children without definable genetically determined lipoprotein disorders. This aspect of familial associations of lipoprotein serum patterns obviously is a confounding factor in attempting to determine the association in healthy children of diet and serum lipids.

"Tracking"

Several investigators have explored the likelihood that serum lipid values found to be increased early in life will be maintained during childhood,

adolescence, and into adulthood (25, 46, 71). Increased levels of cholesterol during childhood do predict for increased levels in the top twentieth percentile as a young adult. However, only about one half of the children found to have increased levels during childhood will have such persistently high levels as young adults (25, 46). The degree to which genetic factors described above played a role in these observations is unknown. Other factors such as obesity acquired in adolescence, oral contraceptive use, and cigarette smoking were found to have deleterious effects on adult cholesterol levels (25). In these studies no attempt was made to correlate dietary patterns with serum lipid levels. No information is available concerning the possible role of dietary alterations in those children in whom decreased values were found subsequently when they became young adults.

Current Serum Values in US Children

During the past two decades several studies have evaluated serum lipid values in children of the United States (12, 16, 24, 27, 75). These studies have reported total serum cholesterol only (12, 16, 27) or total serum cholesterol and the lipoprotein fractions (24, 75). Measurement of total serum cholesterol alone is inadequate with respect to sensitivity and specificity for predicting the low density lipoprotein cholesterol value (16).

The total serum cholesterol value increases rapidly during the first year of life and then demonstrates a much slower increase after the age of two until the age of ten. During the second decade of life a decrement in total serum cholesterol values is noted (12, 16, 27). The total serum cholesterol values do not appear to have changed significantly during the past two decades (75). In addition to these age differences, slight differences according to race and sex have been observed (12, 16, 27). Whether these slight differences are due to genetic patterns or to environmental factors is not known. The biological significance of the differences likewise is unknown. A table of total serum cholesterol values for children that represents a composite of these references is given in Table 1. No data are available that would allow use of total serum cholesterol values in assigning children to risk categories as has been proposed for adults (90b). An attempt to set an arbitrary upper limit of acceptabil-

Table 1 Average total plasma cholesterol values by age, sex, and race (mg/dl)^a

Age	White		Black	
	Male	Female	Male	Female
5-9	160	163	166	175
10-14	157	159	164	165
15-19	151	157	158	165

^a Values derived from References 12, 16, 27, 44.

ity (16, 27, 75) for purposes of intervention presents a difficulty because only 50% of these individuals will be found to have values in the upper ranges when they reach adulthood (46, 71).

Current Dietary Patterns in US Children

Several studies have also reported dietary patterns of American children and adolescents during the past three decades (1, 8, 10, 15, 23, 33, 57, 81, 90, 91). During this period of observation the percent of calories derived from fat has decreased from about 41% in the 1960s (1) to 37 or 38% in the 1970s (10, 15, 23) to 36% in the 1980s (81, 90). A recent report indicates that the percent of food energy from fat in children 1 to 5 years of age is about 34% (90). The percent of total calories from saturated fat sources remains at about 14–15%.

The dietary intake of cholesterol has also shown a similar decrease during this period of time. Daily intake in milligrams has been reduced from an average of 315 in the early 1970s (10) to 280 in the late 1970s (81) to 266 in the early 1980s (57) to values of 245 for girls and 317 for boys in the late 1980s (57).

It must be remembered that these trends represent averages with great variability from individual to individual, from day to day, and reflect differences in racial, regional, and ethnic backgrounds. Clearly, however, within the population of children in the United States significant changes have taken place in dietary patterns during the past three decades.

Response to Dietary Intervention

Several types of studies can be used to assess dietary intervention in children with respect to feasibility, safety, and effectiveness in modifying serum lipid patterns. These include population studies (39) therapeutic attempts for children with hyperlipidemia (28, 31, 94), and interventions that are community (36) or school (76) based programs designed for a general population.

As already noted (40), population studies have demonstrated considerable differences in cholesterol concentrations in children in different countries with different rates of mortality from coronary heart disease for men aged 55–64. No description of dietary patterns among the children studied nor among the adults having different risks for coronary artery disease was included in the report by Knuiman et al. Kaufman and coworkers (39) have compared the nutrient intakes of selective populations in the United States and Israel. Differences were found in total energy intake and the percent of calories coming from fat sources. In Jerusalem the intake of total fat ranged between 32 and 34% of calories, and saturated fatty acids represented about 10% of the calorie intake. In the United States about 39 to 41% of the calories came from fat, and saturated fatty acids represented 14–16% of the total. The data in the United States were obtained from the years 1972 to 1978 and in Jerusalem

from 1976 to 1980. It is important to remember that among children in the United States the percent of calories coming from fat has been steadily decreasing (1, 10, 15, 23, 57, 81, 90). Thus it is difficult to know what the comparable figures would be at this time. The dietary cholesterol intakes were somewhat higher in Jerusalem (184 and 208 mg per 1000 kilocalories for boys and girls respectively) than in the United States (157 and 140 mg per 1000 kilocalories for boys and girls respectively). An attempt was made to evaluate height and weight in relationship to the nutrient patterns. This analysis was confounded by differences among individuals of different origins in Jerusalem, but in general no striking differences in height or body mass was found. The authors concluded that the implementation of a fat-restricted diet was feasible in Western free-living populations that exercised a range of dietary and marketing operations.

The familial hyperlipoproteinemias represent several metabolic defects. The results of therapy for these children have only limited applicability to considerations of dietary interventions in normal children. Interventions by dietary modifications only (28, 31, 94) or dietary modification and the administration of bile acid-binding resins (31, 94) are not consistently effective in reducing increased lipid levels toward normal. Obviously there is great variability in response depending on the nature of the metabolic defect. Glueck and coworkers (31) reported the results of a study evaluating the safety and efficacy of long-term diet or diet and bile acid-binding resin cholesterol lowering therapy in 73 children who were heterozygous for familial hypercholesterolemia. Although no effect on normal growth was observed in these children and no apparent deleterious effects were noted, this report subsequently had to be withdrawn because it was "seriously flawed by unintentional errors" (30). Studies of dietary interventions in children with metabolic defects are not likely to be useful in assessing efficacy in normal children.

The feasibility of implementing a community-based dietary intervention program has been reported by Hollis and coworkers (36). Despite a fairly active recruitment program, only about one half of the families invited to participate actually signed up for the intervention program. In this preliminary report the proportion of families electing intervention that complied fully with the proposed five-year program is not mentioned. The recruitment effort, however, was carried out from May 1978 to May 1979. Possibly, improved results could now be obtained as a result of the change in public perspective on cholesterol and heart disease that has taken place since then (82). A school-based cholesterol reduction intervention program was offered to primary grade students as part of a "know your body" school health program (76). This program includes an annual cholesterol screening for all students. Those students with a total serum cholesterol value greater than 170 mg per dl

were eligible for a workshop designed to teach students about food composition and to modify behavior with respect to diet. Only 34 students in the intervention program demonstrated a decrease in mean cholesterol values from 197 to 179 mg per dl. In a reference group of 118 children who did not participate in the more intensive program, a reduction in cholesterol values from 197 to 184 occurred during the same period of time. This reference group was selected because they were in the upper fiftieth percentile for cholesterol values. The duration of dietary intervention effect and the pattern of subsequent compliance in this study is not known. It would seem, however, that a school-based program might be an effective method of modifying dietary behavior among children. In this study no data are available to describe what changes might actually have taken place and no comparisons of the dietary patterns of the study group and the control group were carried out.

Dietary interventions may carry some risks for children. For example, growth velocities in preschool children given vegetarian diets are decreased, particularly under the age of two years (84). More recently, growth failure in infants (73) and children (49) has occurred as a consequence of parental or the child's concern about dietary excess and its relation to adult chronic diseases. Finberg (20) in an editorial points out the clear need for adequate follow-up and competent counseling concerning dietary intervention when these recommendations are made. In addition to paying attention to total energy intake, care has to be taken to make sure that specific nutrients are provided. For example, iron is an important nutrient for growing infants, children, and adolescents. The bioavailability of dietary iron is critical, and the amount absorbed from food depends on the amount of heme iron available (60). Unmonitored attempts to reduce saturated fats from animal sources could reduce iron intake from meat and calcium intake from dairy products (65).

Future Studies

From the foregoing discussion it is clear that we are missing many elements in our understanding of the role of children's diet as an influential factor in the development of adult atherosclerosis. Certainly, further studies are needed on the evolution of fibrous plaques and their relationship to fatty streaks, which are ubiquitous in children. Much more needs to be known about individual factors that influence the relationship between diet and serum lipid patterns. Longitudinal studies are needed to determine if there is a relationship among children's dietary patterns, serum lipid values, and the risk of adult coronary artery disease. Other contributing factors such as high blood pressure and smoking need to be identified to determine the priorities for intervention. Clearly the role of genetic factors needs to be determined.

If dietary intervention is to be proposed for children and adolescents, well-controlled perspective intervention studies are needed to determine

feasibility, safety, and effectiveness. These studies optimally should include not only short-term studies dealing with the child and adolescent but studies of sufficient duration so that the effect on the adult experience can be determined. At a minimum, however, the studies should be done in children and adolescents so that dietary composition can be assessed in relationship to its feasibility and application for growing children and adolescents.

Conclusions and Recommendations

Many recommendations have been made for modifying the diet of children and adolescents to reduce the risk of coronary artery disease in adults. Some of these recommendations for children and adolescents conform to the limitations of fat and cholesterol published by the consensus development panel (14, 92, 96). Others suggest that dietary intervention be delayed until age five (86, 87) and then carried out with less stringent limitations on fat amounting to about 35% of total calories (3, 7, 86, 87) and no limitations on dietary cholesterol within the ranges usually found in the normal diets of children and adolescents. A recent survey of the practices and attitudes of pediatricians with respect to adult heart disease prevention in childhood (64) indicated that a majority of pediatricians take a family history of cardiovascular diseases, measure blood pressure, recommend exercise to school age children, and advise patients and parents against smoking. A relatively low level of dietary advice is provided, and most pediatricians do not measure serum cholesterol levels except in high risk older children. In this survey (64) few pediatricians expressed confidence in their ability to affect change in patients' life-styles.

Routine measurement of cholesterol levels is consistent with the recommendations of the Committee on Nutrition of the American Academy of Pediatrics (13). The committee has recommended regular elective testing of children over two years of age who have a family history (parent, sibling, grandparent, uncle, aunt) of hyperlipidemia or early myocardial infarction (under fifty years of age in men or under sixty years of age in women). If borderline or increased values are found in the children, several tests in the fasting stage should be obtained including total cholesterol, triglyceride, and density lipoprotein cholesterol determinations. If values persistently exceed the seventy-fifth percentile, then dietary counseling is recommended. This dietary intervention requires many months of nutritional counseling by experienced registered dietitians. The potential hazards of dietary recommendations without careful follow-up have already been noted (20, 49). Sometimes a child or adolescent will be brought to the physician because of findings from a public cholesterol screening program. Because of the inaccuracy of portable cholesterol analyzers used in screening programs, any value must be rechecked (66).

Almost everyone agrees that cholesterol should not be the sole focus of

health-related recommendations for children and adolescents that seek to reduce the risk of adult atherosclerotic heart disease. Blood pressure, smoking, and physical fitness (9) are also important considerations in programs designed to reduce heart disease mortality. The American Academy of Pediatrics has taken all of these factors into account in making recommendations for prudent life-styles (49). These recommendations are as follows:

1. When breast feeding is unsuccessful, inappropriate, or stopped early, infant formulas provide the best alternative for meeting nutritional needs during the first six to twelve months of life. During the second six months of life, whole cow's milk may be introduced to infants who are consuming one third of their calories from a supplemental balanced mixture of cereals, vegetables, fruits, and other foods. Supplementary foods are recommended beginning at four to six months of age. Dietary fat should not be restricted in this age group.
2. After one year of age, infants should receive a varied diet including each of the major food groups. A balanced and varied diet provides the best assurance of nutritional adequacy.
3. Detection of obesity by measuring height and weight and detection of hypertension by measuring blood pressure according to the schedules published by the Academy will permit the early recognition and treatment of obesity and hypertension.
4. Counseling on the maintenance of ideal body weight and a regular exercise program and, in teenagers, counseling concerning the dangers of both smoking and the use of smokeless tobacco should be a routine part of all health provision visits.
5. Family history for each patient should include information about family members who have had a premature heart attack or stroke, hypertension, obesity, diabetes mellitus and/or hyperlipidemia.
6. Screening of children more than two years old who are at risk because of family history should consist of at least two serum cholesterol measurements. High density lipoprotein cholesterol level should be measured in those who consistently have levels above the ninety-fifth percentile for age and sex. If high density lipoprotein cholesterol is not the cause of the hypocholesterolemia, the child should be treated with the appropriate diet and/or medication.
7. Current dietary trends in the US—decreased consumption of saturated fats, cholesterol, and salt and an increased intake of polyunsaturated fats—should be followed with moderation. The optimal total fat intake cannot be determined, but 30 to 40% of calories seems a sensible intake for adequate growth and development. Diets that avoid extremes are safe for children for whom there is no evidence of special vulnerability.

We need to recognize that these recommendations cannot be made in a societal vacuum. Children are not at risk for atherosclerosis, but adolescents may be, particularly boys. Much of the food intake, especially for adolescents, is not from home sources but is obtained in school lunch programs and in fast food restaurants. Many of the recommendations therefore will necessitate a community effort to modify food sources not only in the home but in those restaurants frequented by adolescents as well (76a). Health professionals must become involved in community efforts to improve the diets of adolescent boys with the hope of reducing the development of chronic diseases upon reaching adulthood.

In summary, the present data, in my mind, resolved no issues regarding diets to be recommended during childhood and adolescence. Data are urgently needed to assess the effects of restricting fat and cholesterol and of increasing the proportion of polyunsaturated fats on the growth and health of individuals during the first two decades of life. Final recommendations must take into consideration eating patterns and dietary preferences. In order to be valuable, a nutritional prescription must also be effective and acceptable.

Literature Cited

1. Abraham, S. C., Johnson, C. L., Dresser, C. M. 1979. Caloric and selected nutrient values for persons 1-74 years of age: First health and nutrition examination survey, United States, 1971-1974. *Vital Health Stat. Ser. 11, No. 209, DHEW Publ. No. (PHS) 79-1657. US Dept. Health, Educ., Welfare, Public Health Serv., Natl. Cent. Health Stat.*
2. Ahrens, E. H. Jr. 1985. The diet-heart question in 1985: Has it really been settled? *Lancet* 1(8437):1085
3. American Academy of Pediatrics: Committee on Nutrition. 1986. Prudent lifestyle for children: dietary fat and cholesterol. *Pediatrics* 78(3):521-25
4. Anderson, K. M., Castelli, W. P., Levy, D. 1987. Cholesterol and mortality, 30 years of follow-up from the Framingham study. *J. Am. Med. Assoc.* 257(16):2176-80
5. Barker, K. J. P., Margetts, B., Osmond, C., Simmonds, S. J., Winter, P. D. 1989. Weight in infancy and death from ischaemic heart disease *Lancet* 2(8663):577
6. Becker, M. H. 1987. The cholesterol saga: whither health promotion? *Ann. Intern. Med.* 106(4):623
7. Belmaker, E., Cohen, J. D. 1985. The advisability of the prudent diet in adolescence. *J. Adolesc. Health Care* 6(3):224-32
8. Beynen, A. C., Katan, M. B. 1985. Inter-individual variation in the cholesterolic response to dietary cholesterol. *Prog. Clin. Biol. Res.* 188:195-207
9. Blair, S. N., Kohl, H. W. III, Paffenbarger, R. S. Jr., Clark, D. G., Cooper, K. H., Gibbons, L. W. 1989. Physical fitness and all-cause mortality: A prospective study of healthy men and women. *J. Am. Med. Assoc.* 262(17):2395-2401
10. Carroll, M. D., Abraham, S., Dresser, C. M. 1983. Dietary intake source data: United States, 1976-80. *Vital Health Stat. Ser. 11, No. 231, DHHS Publ. No. (PHS) 83-1681. Washington, DC: US Dept. Health Hum. Serv. Public Health Serv., Natl. Cent. Health Stat.*
11. Deleted in proof
12. Christensen, B., Glueck, C. J., Kwiterovich, P., deGroot, I., Chase, B., et al. 1980. Plasma cholesterol and triglyceride distributions in 13,665 children and adolescents: The prevalence study of the Lipid Research Clinics Program. *Pediatr. Res.* 14(3):194-202
13. Committee on Nutrition. 1989. Indications for cholesterol testing in children. *Pediatrics* 83:141
14. Consensus Conference. 1985. Lowering blood cholesterol to prevent heart dis-

- ease. *J. Am. Med. Assoc.* 253(14):2080-86
15. Cresanta, J. L., Farris, R. P., Croft, J. B., Webber, L. S., Frank, G. C., Berenson, G. S. 1988. Trends in fatty acid intakes of 10-year-old children, 1973 to 1982. *J. Am. Diet. Assoc.* 88 (84):178
16. Dennison, B. A., Kikuchi, D. A., Srinivasan, S. R., Webber, L. S., Berenson, G. S. 1990. Serum total cholesterol screening for the detection of elevated low-density lipoprotein in children and adolescents: the Bogalusa Heart Study. *Pediatrics* 85(4):472-79
17. Enos, W. F., Holmes, R. H., Beyer, J. 1953. Coronary disease among United States soldiers killed in action in Korea: Preliminary report. *J. Am. Med. Assoc.* 152:1090
18. Epstein, A. M., Oster, G. 1987. Cholesterol reduction and health policy: taking clinical science to patient care. *Ann. Intern. Med.* 106(4):621-23
19. Fihn, S. D. 1987. A prudent approach to control of cholesterol levels. *J. Am. Med. Assoc.* 258(17):2416-18
20. Finberg, L. 1989. Dietary advice: Responsibility for monitoring. *Am. J. Dis. Child.* 143(5):531
21. Deleted in proof
22. Frank, G. C., Berenson, G. S., Webber, L. S. 1978. Dietary studies and the relationship of diet to cardiovascular disease risk factor variables in 10-year old children—The Bogalusa Heart Study. *Am. J. Clin. Nutr.* 31(2):328
23. Frank, G. C., Farris, R. P., Cresanta, J. L., Webber, L. S., Berenson, G. S. 1985. Dietary trends of 10- and 13-year-old children in a biracial community—The Bogalusa Heart Study. *Prev. Med.* 14(1):123-39
24. Freedman, D. S., Srinivasan, S. R., Cresanta, J. L., Webber, L. S., Berenson, G. S. 1987. Serum lipids and lipoproteins. *Pediatrics* 80(5):789-96
25. Frerichs, R. R., Srinivasan, S. R., Webber, L. S., Berenson, G. S. 1976. Serum cholesterol from a biracial community: The Bogalusa Heart Study. *Circulation* 54(2):302-8
26. Garber, A. M. 1989. Where to draw the line against cholesterol. *Ann. Intern. Med.* 111(8):625
27. Garcia, R. E., Moodie, D. S. 1989. Routine cholesterol surveillance in childhood. *Pediatrics* 84(5):751-55
28. Glueck, C. J. 1983. Therapy of familial and acquired hyperlipoproteinemia in children and adolescents. *Prev. Med.* 12(6):835-47
29. Glueck, C. J. 1986. Pediatric primary prevention of atherosclerosis. *New Engl. J. Med.* 314(3):175-77
30. Glueck, C. J., Laskarzewski, P., Mellies, M. J., Perry, T. 1987. Dr. Glueck and co-authors' letter of withdrawal. *Pediatrics* 80(5):766
31. Glueck, C. J., Mellies, M. J., Dine, M., Perry, T., Laskarzewski, P. 1986. Safety and efficacy of long-term diet and diet plus bile acid-binding resin cholesterol-lowering therapy in 73 children heterozygous for familial hypercholesterolemia. *Pediatrics* 78:338-48
32. Glueck, C. J., Waldman, G., McClish, D. K., Morrison, J. A., Khoury, P., et al. 1982. Relationships of nutrient intake to lipids and lipoproteins in 1234 white children. The Lipid Research Clinics Prevalence Study. *Arteriosclerosis* 2(6):523-36
33. Hampton, M. C., Huenemann, R. L., Shapira, L. R., Mitchell, B. W. 1967. Caloric and nutrient intakes of teenagers. *J. Am. Diet. Assoc.* 50:385.
34. Deleted in proof
35. Heber, D., Koziol, B. J., Henson, L. C. 1987. Low density lipoprotein receptor regulation and the cellular basis of arteriosclerosis. *Am. J. Cardiol.* 60(12):4G-8G
36. Hollis, J. F., Sexton, G., Connor, S. L., Calvin, L., Pereira, C., Matarazzo, J. D. 1984. The family heart dietary intervention program: Community response and characteristics of joining and nonjoining families. *Prev. Med.* 13(3):276-85
37. Isles, C. G., Hole, D. J., Gillis, C. R., Hawthorne, V. M., Lever, A. F. 1989. Plasma cholesterol, coronary heart disease, and cancer in the Renfrew and Paisley Survey. *Br. Med. J.* 298 (6678):920-24
38. Kannel, W. B., Doyle, J. T., Ostfeld, A. M., Jenkins, C. D., Kuller, L., et al. 1984. Special Report of the Inter-society Commission for Heart Disease Resources. *Am. Heart Assoc.* 70(1):155A-205A
39. Kaufmann, N. A., Dennis, B. H., Heiss, G., Friedlander, Y., Kark, J. D., Stein, Y. 1986. Comparison of nutrient intakes of selected populations in the United States and Israel. The Lipid Research Clinics Prevalence Study. *Am. J. Clin. Nutr.* 43:604-20
40. Knuiman, J. T., Hermus, R. J. J., Hautvast, J. G. A. J. 1980. Serum total and high density lipoprotein (HDL) cholesterol concentrations in rural and

- urban boys from 16 countries. *Atherosclerosis* 36:529
41. Kolata, G. 1985. Heart panel's conclusions questioned. *Science* 227(4682): 40-41
42. Kondo, S., Takahashi, E., Kato, K., Takahashi, S., Ikeda, M. 1978. Secular trends in height and weight of Japanese pupils. *Tokoku J. Exp. Med.* 126(3): 203-13
43. Kritchevsky, D. 1986. Atherosclerosis and nutrition. *Nutr. Int.* 2:290
44. Kwiterovich, P. O. Jr. 1986. Biochemical, clinical, epidemiologic, genetic, and pathologic data in the pediatric age group relevant to the cholesterol hypothesis. *Pediatrics* 78(2):349-62
45. LaRosa, J. C., Hunnigake, D., Bush, D., Criqui, M. H., Getz, G. S. et al. 1990. *Circulation*. The cholesterol facts. 81(5):1721-33
46. Lauer, R. M., Lee, J., Clarke, W. R. 1988. Factors affecting the relationship between childhood and adult cholesterol levels: the muscatine study. *Pediatrics* 82(3):309-18
47. Leaf, A. 1989. Management of hypercholesterolemia: Are preventive interventions advisable? *New Engl. J. Med.* 321(10):680-84
48. Lee, J., Lauer, R. M., Clarke, W. R. 1986. Lipoproteins in the progeny of young men with coronary artery disease: Children with increased risk. *Pediatrics* 78(2):330-37
49. Lifshitz, F., Moses, N. 1989. Growth failure: A complication of dietary treatment of hypercholesterolemia. *Am. J. Dis. Child.* 143(5):537-42
50. Lipid Research Clinics. 1984. The lipid research clinics coronary primary prevention trial results: reduction in incidence of coronary heart disease. *J. Am. Med. Assoc.* 251(3):351
51. Lipid Research Clinics. 1984. The lipid research clinics coronary primary prevention trial results: the relationship of reduction in incidence of coronary heart disease in cholesterol lowering. *J. Am. Med. Assoc.* 251(3):365
52. Malina, R. M. 1978. Secular changes in size and maturity: Causes and effects. *Monogr. Soc. Res. Child Dev.* 43:59
53. Deleted in proof
54. McGill, H. C. Jr., Geer, J. C., Strong, J. P. 1963. Natural history of human atherosclerotic lesions. In *Atherosclerosis and Its Origin*, ed. M. Sandler, G. H. Bourne, pp. 39-65. New York: Academic
55. McNamara, D. J., Kolb, R., Parker, T. S., Batwin, H., Samuel, P., et al. 1987. Heterogeneity of cholesterol homeostasis in man. *J. Clin. Invest.* 79(6):1729-39
56. McNamara, J. J., Molot, M. A., Stremple, J. F., Cutting, R. T. 1971. Coronary artery disease in combat casualties in Vietnam. *J. Am. Med. Assoc.* 216(7):1185
57. McPherson, S. R., Nichaman, M. Z., Kohl, H. W., Reed, D. B., Labarthe, D. R. 1990. Intake and food sources of dietary fat among schoolchildren in The Woodlands, Texas. *Pediatrics* 86:520
58. Mellies, M., Glueck, C. J. 1983. Lipids and the development of atherosclerosis in children. *J. Pediatr. Gastroenterol. Nutr.* 2:S298-303
59. Mitchell, J. R. A. 1985. Symposium on "Nutrition and Thrombosis" Diet and arterial disease—the myths and the realities. *Proc. Nutr. Soc.* 44(3):363
60. Monsen, E. R., Hallberg, L., Layrisse, M., Hegsted, D. M., Cook, J. D., et al. 1978. Estimation of available dietary iron. *Am. J. Clin. Nutr.* 31(1):134-41
61. Morrison, J. A., Kelly, K. A., Mellies, M. J., deGroot, I., Glueck, C. J. 1978. Parent-child associations at upper and lower ranges of plasma cholesterol and triglyceride levels. *Pediatrics* 62(4): 486-77
62. Morrison, J. A., Larsen, R., Glatfelter, L., Boggs, D., Burton, K., et al. 1980. Interrelationships between nutrient intake and plasma lipids and lipoproteins in school-children aged 6 to 19: The Princeton School District Study. *Pediatrics* 65(4):727-34
63. Multiple Risk Factor Intervention Trial Research Group. 1982. Multiple risk factor intervention trial: risk factor changes and mortality results. *J. Am. Med. Assoc.* 248(2):1465
64. Nader, P. R., Taras, H. L., Sallis, J. F., Patterson, T. L. 1987. Adult heart disease prevention in childhood: a national survey of pediatricians' practices and attitudes. *Pediatrics* 79(6):843-50
65. National Academy of Sciences. 1987. *Recommended Dietary Allowances*, pp. 1-184. Washington, DC: Natl. Acad. Sci. 9th ed.
66. Naughton, M. J., Luepker, R. V., Strickland, D. 1990. The accuracy of portable cholesterol analyzers in public screening programs. *J. Am. Med. Assoc.* 263(9):1213-17
67. Newman, W. P. III, Freedman, D. S., Voors, A. W., Gard, P. D., Srinivasan, S. R., et al. 1986. Relation of serum

- lipoprotein levels and systolic blood pressure to early atherosclerosis. *New Engl. J. Med.* 314(3):138-44
68. Nicklas, T. A., Farris, R. P., Major, C., Frank, G. C., Webber, L. S., et al. 1987. Dietary intakes. *Pediatrics* 80(5):797-806
 69. Nicklas, T. A., Farris, R. P., Smoak, C. G., Frank, G. C., Srinivasan, S. R., et al. 1988. Dietary factors relate to cardiovascular risk factors in early life. *Arteriosclerosis* 8(2):193-99
 70. Oliver, M. F. 1985. Consensus or non-sensus conferences on coronary heart disease. *Lancet* 1(8437):1087
 71. Orchard, T. J., Donahue, R. P., Kuller, L. H., Hodge, P. N., Drash, A. L. 1983. Cholesterol screening in childhood: Does it predict adult hypercholesteremic? The Beaver County experience. *Pediatrics* 103(5):687-91
 72. Palumbo, P. J. 1989. Cholesterol lowering for all: A closer look. *J. Am. Med. Assoc.* 262(1):91-92
 73. Pugliese, M. T., Weyman-Daum, M., Moses, N., Lifshitz, F. 1987. Parental health beliefs as a cause of nonorganic failure to thrive. *Pediatrics* 80(2):175-82
 74. Reiser, R. 1984. A commentary on the rationale of the diet-heart statement of the American Heart Association. *Am. J. Clin. Nutr.* 40(3):654
 75. Resnicow, K., Morley-Kotchen, J., Wynder, E. 1989. Plasma cholesterol levels of 6585 children in the United States: Results of the know your body screening in five states. *Pediatrics* 84(6):969-76
 76. Resnicow, K., Orlandi, M. A., Vaccaro, D., Wynder, E. 1989. Implementation of a pilot school-site cholesterol reduction intervention. *J. School Health* 59(2):74-78
 - 76a. Roberts, C. 1989. Fast-food fare—consumer guidelines. *New Engl. J. Med.* 321:752-56
 77. Roche, A. F. 1978. Secular trends in stature, weight, and maturation. *Monogr. Soc. Res. Child Dev.* 44(3-4):1-120
 78. Ross, R. 1986. The pathogenesis of atherosclerosis—An update. *New Engl. J. Med.* 314(8):488-500
 79. Ross, R., Glomset, J. A. 1976. The pathogenesis of atherosclerosis. *New Engl. J. Med.* 295:369-77, 420-25
 80. Roy, C. C., Galeano, N. 1985. Childhood antecedents of adult degenerative disease. *Pediatr. Clin. North Am.* 32(2):517
 81. Salz, K. M., Tamir, I., Ernst, N., Kwiterovich, P., Glueck, C., et al. 1983. Selected nutrient intakes of free-living white children ages 6-19 years. The Lipid Research Clinics Program Prevalence Study. *Pediatr. Res.* 17(2):124-30
 82. Schucker, B., Bailey, K., Heimbach, J. T., Mattson, M. E., Wittes, J. T., et al. 1987. Change in public perspective on cholesterol and heart disease: results from two national surveys. *J. Am. Med. Assoc.* 258(24):3527-31
 83. Schucker, B., Wittes, J. T., Cutler, J. A., Bailey, K., Mackintosh, D. R., et al. 1987. Change in physician perspective on cholesterol and heart disease: results from two national surveys. *J. Am. Med. Assoc.* 258(24):3521-26
 84. Shull, M. W., Reed, R. B., Valadian, I., Palombo, R., Thorne, H., Dwyer, J. T. 1977. Velocities of growth in vegetarian preschool children. *Pediatrics* 60(4):410-17
 85. Small, D. M. 1977. Cellular mechanisms for lipid deposition in atherosclerosis. *New Engl. J. Med.* 297:873-77, 924-29
 86. Taitz, L. S. 1987. Diet of young children and cardiovascular disease. *Br. Med. J.* 294(6577):920-21
 87. Tarlow, M. J. 1989. Cholesterol and diet. *Arch. Dis. Childh.* 64(5):647-48
 88. Taylor, W. C., Pass, T. M., Shepard, D. S., Komaroff, A. L. 1985. Cholesterol reduction and life expectancy. A model incorporating multiple risk factors. *Ann. Intern. Med.* 106(4):605-14
 - 88a. Tcjada, C., Gorc, I., Strong, J. P., McGill, H. C. Jr. 1958. Comparative severity of atherosclerosis in Costa Rica, Guatemala, and New Orleans. *Circulation* 18:92-97
 89. Texon, M. 1989. Guest Editorial: The cholesterol-heart disease hypothesis (critique)—time to change course? *Bull. NY Acad. Med.* 65(8):836
 90. US Dep. Agric., Human Nutrition Information Service. 1985. Nationwide Food Consumption Survey, continuing survey of food intakes by individuals. *NFCS, CSFII Rep. No. 85-1*, pp. 48-49
 - 90a. US Dep. Health Human Services. 1984. *US Dep. Health Hum. Serv. Publ. No. 851232*. Washington, DC: GPO
 - 90b. US Dep. Health Human Services. 1988. Cholesterol: Current concepts for clinicians. Natl. Cholesterol Educ. Program
 91. Weidman, W. H., Elveback, L. R., Nelson, R. A., Hodgson, P. A., Ellefson,

- R. D. 1978. Nutrient intake and serum cholesterol level in normal children 6 to 16 years of age. *Pediatrics* 61(3):354-59
92. Weidman, W., Kwiterovich, P. Jr., Jesse, M. J., Nugent, E. 1983. Diet in the healthy child. *Circulation* 67(6): A1411-14
93. Werko, L. 1987. The enigma of coronary heart disease and its prevention. *Acta Med. Scand.* 221:(4)323-33
94. Widhalm, K. 1985. Effect of diet on serum lipoproteins in children with various forms of hyperlipidemias. *Prog. Clin. Biol. Res.* 188:145-59
95. Wissler, W. R., McGill, C. H. Jr. 1983. Conference on blood lipids in children: optimal levels for early prevention of coronary artery disease. *Prev. Med.* 12(6):868
96. Wynder, E. L. 1983. Summary and recommendations of the conference on blood lipids in children: Optimal levels for early prevention of coronary artery disease. *Prev. Med.* 12(6):728