DIET AND HEALTH: PERCEPTIONS AND REALITIES

(INVITED REVIEW)

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"The new danger to our well-being, if we continue to listen to all the talk, is in becoming a nation of healthy hypochondriacs, living gingerly, worrying ourselves half to death." Lewis Thomas, 1975.

INTRODUCTION

Observations of associations between the increasing incidence of chronic and degenerative diseases and changes in the composition of the food supply during this century have led many health authorities to conclude that deterioration of the quality of the food supply has become a major threat to health. Public acceptance of this conclusion has resulted in widespread fear of food and fear for health. In most of the highly industrialized nations of the world, the response to these fears has been to propose dietary guidelines for the entire population as a means of "preventing" chronic and degenerative diseases and, thereby, reducing the costs of medical care (Harper 1981). These guidelines have frequently been announced in a way that tends to magnify rather than allay fears for health. In the United States some well-educated, affluent parents have restricted the diets of their young children, in the hope of preventing them from developing cardiovascular and other diseases, to a point where they have been hospitalized for "failure to thrive" (Pugliese et al. 1987).

It seems anomalous, when the health of the populations of the highly industralized nations is better than it has ever been, that such fears about the quality of the food supply should abound. It becomes important to ask whether popular perceptions about relationships among diet, health, and disease are based on validassumptions and objective scientific evidence and whether the responses to these perceptions are rational. It is certainly appropriate that the public should be given reliable advice about healthful diets, but is it justifiable to present dietary advice in a way that undermines the confidence of the consumer in the food supply and promises, by innuendo if not directly, that avoidance of certain foods and selection of others is an effective way of preventing chronic and degenerative diseases? There are many sceptics of the efficacy of mass diet modification as a disease prevention measure (Olson 1986). Information about diet-disease relationships should, therefore, be evaluated critically in order to distinguish clearly between real and perceived problems and to ensure that problems requiring attention will be dealt with in ways that are scientifically, socially, and economically sound.

Diet-Health relationships being proposed as the basis for national food and nutrition policy cannot be viewed in perspective without considering the trends that have occurred in the state of health of the population and in the composition of the food supply since the early part of the century. What is, striking about the exhortations for disease prevention by diet modification, and the reports to the Australian Better Health Commission (1986) follow the pattern, is the failure to view current health problems critically in perspective over time. The report "Healthy People" of the U.S. Surgeon General (U.S.D.H.E.W. 1979), in which each chapter begins with a review of health trends, is an exception. The U.S. report that is cited most frequently, however, is "Dietary Goals for the United States," a report prepared by the staff of a Senate Committee (U.S. Senate 1977a), written by politicians for politicians. It was not adopted

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by the U.S. Government despite statements to that effect in a report to the Australian Better Health Commission (1986, Chapter 7, p.35). The political nature of the U.S. Dietary Goals is also evident from failure to include and discuss in the report views contrary to those accepted by the Committee staff --'a blatantly unscientific approach. The staff that produced the U.S. Dietary Goals included a few contrary opinions in a separate and little known document (U.S. Senate 1977b) but ignored them completely in preparing its recommendations.

TRENDS IN HEALTH STATUS

Indicators of the health status of populations include: infant, childhood, and maternal mortality; mortality from various diseases, including nutritional deficiency diseases; growth rates of children; death rates at various ages; life expectancy; and the proportion of infants who survive to 65 years of age and beyond.

Health status in the highly industralized nations during the early 1900s resembled that in most of the poor nations today. At the turn of the century infant mortality exceeded 100 per 1000 live births; death rates generally were high for those under 55 years of age; infectious diseases were major causes of death and nutritional deficiency diseases were public health problems; less than 40% of infants born could be expected to survive to 65 years of age; only a small proportion of those who survived could be expected to achieve their full potential for growth and development; life expectancy at birth was less than 50 years (Omram 1977; U.S.D.H.E.W. 1979; Fries 1980; Tanner 1986).

During this century the highly industralized nations have undergone striking transitions in health status. As science and technology developed, industralization progressed, knowledge of nutrition expanded, the importance of sanitation was recognized, medical knowledge and practice advanced, and the standard of living rose. All of these impacted on the indices of health giving rise to epidemiologic and demographic transitions.

Infant mortality has fallen in Australia, the United Kingdom and the United States to 10% of what it was; it has fallen even further in Finland, Japan and Sweden. Similar declines have occurred in childhood and maternal mortality. Nutritional deficiency diseases have been virtually eliminated and, in most industralized countries where measurements over time are available, growth rates have increased until it now appears that children are achieving their genetic potential for height at maturity (Tanner 1986).

Death rates have declined at all ages but much-more for the young than the elderly. Close to 80% of infants born can now be expected to live to age 65 or beyond; life expectancy of infant girls is approaching 80 years (79 in Australia) and of infant boys is over 70 years (almost 72 in Australia). Even among people 65 years of age and older in the U.S.A., close to 70% respond that their health is good to excellent (Fries 1980; U.S.D.H.E.W. 1979; U.S.D.H.H.S. 1985).

These improvements in health have led to a demographic transition in the industralized nations. Figures vary from country to country but, in general, the proportion of people 65 years of age or older in the populations of the industralized nations has risen from about 4% around 1900 to about 12% at the present time. It is a little lower in Australia and Canada probably owing to the influx of young people through immigration. Projections indicate that the proportion in this segment of the population will continue to rise for some time (Harper 1984).

It is amazing, in view of these immense improvements in health, that so much attention should be focused on the presumed influence of diet on the high death rates from chronic and degenerative diseases and so little on its contribution to increased survival and growth of children, improved health, and greater life expectancy.

EPIDEMIOLOGIC TRANSITION

Infectious diseases, especially bronchial diseases, gastroenteritis, and tuberculosis were the major causes of death early in this century. They were responsible for high infant, childhood, and maternal mortality and for low life expectancy. As sanitation, food supply, and medical care improved, mortality from these diseases declined, until by the 1940s they had become minor causes of death in thehighlyindustralized nations. The decline was most impressive in Japan where it occurred over a period of only 10 years following World War II (Omram 1977; Harper 1987).

The other side of the epidemiologic transition has been the steady increase in mortality from chronic and degenerative diseases - mainly cardiovascular and cerebrovascular diseases (heart disease and stroke) and cancer. These diseases together, which accounted for only 18% of all deaths in 1900, now account for between 65 and 70% (U.S.D.H.H.S. 1985). This has given rise to claims that major chronic and degenerative diseases are epidemic in the highly industralized nations. Perceptions obtained from looking only at total mortality and crude death rates can be highly misleading (Harper 1983). To place this epidemiologic transition in perspective many other factors must be considered.

To begin with, cardiovascular diseases (CVD) were the major single cause of death in the U.S.A. in 1900, accounting for 14% of all deaths, at a time when only 4% of the population was 65 years of age or older. Death rates from these diseases increase exponentially with increasing age. Now when 12% of the population is in this age category, CVD account for about 45% of all deaths '(Harper 1987). With a threefold increase in the proportion of elderly people and just slightly above a threefold increase in the proportion of deaths from CVD, there would appear to be a close association between aging and death from these diseases. A similar trend is observed for cancermortality. This raises a question as to whether we have had an epidemic of CVD or whether it has been an epidemic of aging. While crude death rates from chronic and degenerative diseases have been rising, total death rates have been declining steadily at all ages, even among people 65 years of age and older. Also, during this period, life expectancy of males has increased by 25 years and females by 30 years. The proportion of infants who survive to age 65 years has doubled. These patterns are essentially the same throughout the highly industralized nations although the relative proportions of deaths from different chronic and degenerative diseases varies considerably from country to country (Harper 1984).

It must be a very strange type of epidemic indeed that accounts for two-thirds of all deaths, yet is accompanied by improved health, declining death rates at all ages, and increased life expectancy. There would seem to be a gap between the perception and the reality.

TRANSITION IN DIET

Changes in diet with increasing personal or national income have been well-documented (Périssé et al. 1969). When income per capita is low, cereal grains, root crops and foods of plant origin comprise a high proportion of the diet: As income rises, the proportion of animal products and saturated fats and sugar in the diet increase until about half of the energy and two-thirds of the protein

are from animal products. This has a striking effect on the nutrient composition of the food supply and the diet. As income rises from very low to very high, the fat content of the diet rises from 10-12% to about 40% of total energy (calories); the saturated fatty acid content of the fat rises; the carbohydrate content of the diet falls from about 75% of total energy to about 45% and, as sugar consumption rises, the decline is almost exclusively in starch; protein content of the diet rises relatively little, from 10-12% to 12-15% of total energy but with the shift from mainly plant to mainly animal sources, the quality of the protein improves. This pattern of change has been evident in Australia, the United States, Canada, New Zealand and Western Europe where, over the past 60 years, the increase in the fat content of the food supply has been from about 30-40% of total energy. More recently it has become evident in Japan where the fat content of the food supply has increased from about 15 to 25% of total energy sources during the past 20 years (Harper 1984, 1987). The amounts of most essential nutrients in the U.S. and Australian food supplies have tended to increase during this century.

These changes in the composition of the food supplies of the highly industrialized nations have occurred during the period of time in which mortality from chronic and degenerative diseases has risen . There is, therefore, an association over time between the changes in diet and the changes in the major causes of death. This association, emphasized by the authors of the Dietary Goals for the United States (U.S. Senate 1977a) and in the reports submitted to the Australian Better Health Commission (1986), is often assumed to be a cause and effect relationship. It merits close examination, for in many countries, including Australia and the United States, death rates from cardiovascular diseases have been declining steadily for at least 20 years, and probably closer to 40 years, in the U.S.A., without evidence of appreciable changes occurring in the composition of the diet (Harper 1983, 1987).

A common finding in health surveys done in the highly industrialized nations is a high incidence of overweight and obesity. In the United States 20-30% of men and 25-40% of women in the various age groups beyond the age of 35 are considered to be overweight (U.S.D.H.H.S. & A., 1986). In Australia 8-21% of men, and 10-15% of women between the ages of 40 and 60 years are considered to be obese (Australian Better Health Commission 1986). Even in Japan, with its much lower fat diet, 13-16% and 15-22% of women are classified as overweight (Harper 1987). Excessive body weight (usually based on body mass index (BMI), weight in kg/height^2 in metres which is a better indicator of overweight - and body weight alone) is a health risk beginning at about 20% above desirable or appropriate BMI, especially if it occurs at an early age (Simopoulos & Van Itallie 1984). There is a strong association between excessive body weight and the incidence of diabetes and hypertension, both of which are risk factors . for ischemic or coronary heart disease (CHD). The incidence of overweight has increased in association with the changing character of the food supply also, and , the high incidence of overweight is often attributed to undesirable changes that have occurred in the nature of the food supply. This association also merits close examination.

There can be little doubt but that a substantial proportion of the populations of countries that have an abundant, appetizing and inexpensive food supply, consume from it in excess of their needs for energy. Quite apart from the increased incidence of diseases associated with excessive body weight, the millions of dollars spent each year on weight reductionprograms is evidence itself that the condition is considered undesirable. The question at hand, however, is can the high incidence of obesity be-attributed to the changes that have occurred in the composition of the food supply? It is certainly easier to consume more energy from'a high fat than from a low fat diet, but the Japanese

experience indicates that it is not difficult to consume more energy than is needed from a diet that is low in fat. Also, with the demand for physical activity declining as much as it has since the advent of industrialization, it is easier now to consume more than is needed than it used to be. Nonetheless, a high proportion of the population is not overweight, so the problem is obviously not attributable simply to the composition of the diet. Overeating, or failing to balance energy intake and energy expenditure, may be a problem for many people either in using the food supply appropriately or in maintaining an adequate level of physical activity but it certainly does not represent a hazard of the food supply itself.

A fact overlooked by critics who indict the food supplies of Australia and the United States as a threat to health, is that Sweden and Japan, countries with health records that are unsurpassed, consume distinctly different proportions of carbohydrate and fat. These critics also ignore observations that the populations of Sweden, the Netherlands, Switzerland, Norway and Denmark, among the highest for fat and sugar consumption in Europe, are all in the top ten in the world for low infant mortality and long life expectancy, with the populations of Australia and the United States in the next five (Harper 1984, 1987). Could it be that the high incidence of chronic and degenerative diseases in these populations is associated more strongly with their excellent state of general health than with changes that have occurred in the composition of their diets during this century? Is the assumption that some unique proportions of fat, carbohydrate and fibre in the diet are critical for good health and prevention of chronic and degenerative diseases valid? Both the diets and the major causes of death in these countries differ greatly, yet overall health status is similar in all of them. It is noteworthy that proposals for mass diet modification have been based almost exclusively on information pertaining to specific diet-disease relationships, not on relationships between diet and general health. The evidence about diet-disease relationships should be scrutinized carefully.

CHRONIC AND DEGENERATIVE DISEASES

Chronic and degenerative diseases are the major causes of death and disability in the highly industrialized nations. Incidence of and mortality from these diseases have been rising steadily during this century. They are major medical problems. Increased death rates from these diseases, as we have noted, have occurred in association with changes in the content of major nutrients in the food supply; they have also occurred in association with improved health generally, with declining overall death rates, increased longevity; and an increased -proportion of elderly people in these populations, These are well-documented realities. If all of these trends, and many we have not discussed, are associated over time, can we establish on sound scientific grounds the validity of perceived cause and effect relationships among then? Attempting to discern cause and effect relationships from information about associations or correlations is fraught with hazard.

A logical place to start in trying to sort out theserelationships is by recognizing that death rates from most diseases'increase essentially exponentially with increasing age; time-trends in crude death rates will not reflect. changes in mortality accurately if the proportion of elderly people in' the population has been increasing. Meaningful comparisons, can be obtained only after adjusting the rates to take into consideration changes over time in the proportion of the population is different 'age. groups. Age-adjusted death rates obtained in this way can then be used to establish accurate time-trends for mortality from chronic and degenerative diseases.

Age-adjusted death rates from various cancers show few significant trends over the past 50 years in countries as diverse as the U.S.A. and Japan. Death rates from lung cancer, associated with heavy cigarette smoking, have risen. In the USA, the age-adjusted death rate from gastric cancer has declined steadily '(American Cancer Society 1986). For most othercancers, rates have remained constant or declined slightly. There has certainly not been an epidemic of cancer associated with whatever changes may have occurred in the environment, including the food supply, over the past 50 years.

The situation with heart disease mortality is more difficult to assess. It is important to note that the time-trends for death rates from heart disease in all of the discussions of relationships betweent diet and heart disease mortality are for coronary (ischemic) heart disease only. The age-adjusted time trends presented for death rates from CHD show a rising trend up to 1968 with a steady decline thereafter. What is not evident from such presentations is that the term "ischemic heart disease" or CHD, was introduced into the classification of diseases in 1965 and, in 1968, 90% of all heart disease deaths in the USA were attributed to CHD. Diseases that can be identified as CHD accounted for 75% of heart disease deaths in 1960, 60% in 1950, and only 22% in 1940. Graphs and tables published by the US National Center for Health Statistics show that death rates from total cardiovascular-renal diseases (CVD), major diseases of the heart, stroke, and hypertension were at their maxima in or prior to 1940 and have declined steadily since then (Harper 1983, 1987). Interpretation of the information on time-trends in age-adjusted death rates from CHD has been the subject of much debate (Slater et al 1985). The trends for death rates from total CVD, major diseases of the heart, and stroke are clear; they have all been steadily downward since the 1940s (Grove & Hetzel 1968). If mortality from CHD rose steadily prior to 1968, this rise must have been accompanied by greater declines in mortality from other diseases of the arteries and heart, otherwise a decline could not have occurred in the death rate from major diseases of the heart (Harper 1983). A similar analysis of this question in the UK by Robb-Smith (1967) led him to conclude that there had been no increase in the incidence of diseases that could be classified as CHD during the years prior to 1960 in England, Wales or the USA.

It is doubtful that the controversyover time-trends in CHD mortality can be resolved. Whether the trends were upward or downward prior to 1968, they occurred during a period when there was no evidence of significant dietary trends. There has been little success in relating changing patterns of heart disease mortality in the USA since 1968 to any of the modifiable-risk factors for this disease, other than smoking and hypertension (Stallones 1983). And more recently, upward trends in CHD mortality have been reported in Norway and Sweden where diets resemble in composition those in the US, Canada and Australia, countries in which trends in CHD mortality have been steadily downward for a number of years . (Oliver 1986).

As serum cholesterol concentration is generally accepted as a major risk factor for CHD, and as effects of diet on serum cholesterol concentration are at the heart of the assumptions that diet composition is a major risk factor for CHD, intervention trials, in which effects of diet or cholesterol-lowering agents' on CHD mortality are examined, should provide a critical test of the diet-health hypothesis. There have been about 20 such trials in which either drugs or diet or both were used as the treatment, mainly on men identified as being at high risk for CHD. The results of all of them have been equivocal (Olson 1986; Oliver 1985). In several such studies, reductions in serum cholesterol concentration . were associated with reduced mortality from CHD but this was balanced by increases in mortality from other causes, so that overall mortality in treated and untreated populations did not differ significantly (Olson 1986; Becker 1987).

Results of this type should not be surprising. Genetic defects of lipid metabolism that result in greatly elevated serum cholesterol concentrations are known to be associated with a high incidence of CHD. The subjects in most of these studies were men at high risk'for CHD from greatly elevated serum cholesterol concentrations. Also, life expectancies of populations in which CHD mortality differs greatly are small. Death rates from CHD per 100,000 men aged 35 to 74 years in the 1979s was only 115 in Japan and 205 in France but 506 in The Netherlands and 793 in the USA. Life expectancy of 73 years was the same for the French and the Americans and was one year higher for the Dutch and the Japanese both of whom have lower infant mortality. Life expectancy at age 65 years, an age reached by close to 80% of these populations, was the same for all (Harper 1987). Despite substantial differences in diet and in the major causes of death, life expectancy varies little among the populations of the highly industrialized nations. It would seem that among these populations the major causes of death may be influenced by the environment without greatly altering longevity. The implication is that changes beneficial for one segment of the population may be detrimental for another (Olson 1986; Becker 1987). Under such conditions would it not be best to identify those who are highly susceptible and treat them specifically?

VARIABILITY AND GENETIC DIVERSITY .

Dietary recommendations proposed for the entire population were, until recently, directed toward ensuring that the genetic potential for reproductive performance, growth and development, and work capacity would be achieved. During the past 15 years the perception has evolved or been created, that other sets of dietary recommendations are needed to ensure longevity and avoidance of chronic and degenerative diseases. These diseases are claimed to be "life-style" diseases and, therefore, avoidable by following a set of specific health practices. Implicit in this viewpoint are the assumptions that heredity and individual variability are minor factors in the development of such diseases and that the population is uniformly at risk of developing them'and will respond uniformly by following the recommendations proposed for preventing them.

Scriver (1982) has questioned these assumptions. He has pointed out that as diseases of environmental origin such as infections and nutritional deficiency diseases are controlled and longevity increases, the probability that diseases of genetic origin will predominate also increases. Age-onset diabetes, hypertension and various hyperlipoproteinaemias, which are known to have a genetic basis, have 'become increasingly important medical problems as life expectancy has lengthened (Scriver 1982; Neel 1984). A major direction in pediatric practice has been to identify patients with genetic defects of metabolism early in life and to treat them rigorously. It has become possible to prevent the severe adverse effects of some of these defects by providing appropriate, and usually stringent, dietary guidance. Even for adults who are genetically susceptible to age-onset diabetes, careful control of dietary intake and body weight greatly reduces-the probability of developing overt disease. Those who are not susceptible remain free of the disease even in an environment in which they accumulate body fat.

How to deal with variability among individuals within a population is one of the first lessons that must be learned by the budding biologist. For most *biological variables, coefficients of variation of 15% are usual. Hence, quite apart from groups that fall into unique populations because of specific genetic defects; even within a randomly selected population, the range of responses to a treatment is predicted from the normal distribution to be ±30-50% of the mean. Associations between risk factors and development of CHD are far from consistent, with a high proportion of CHD occurring among individuals considered to be at low risk,whereas many considered to be at high risk remain free of the disease

(Oliver 1986; Becker 1987). The same type of variability is observed in the distribution of serum cholesterol concentrations and among the responses of individuals to treatments designed to raise orlowerthe concentration. Age-at-death also tends to follow a Gaussian distribution with most deaths in long-lived populations occurring between ages 50 and 90 (Harper 1987). The propensity of advocates of mass dietary treatment for the population to identify deaths prior to 55, 65 and occasionally even 80 years of age as premature deaths is both biologically and statistically unsound. In fact, evidencethat a substantial part of the decline in heart disease mortality during the past decade in the USA can be attributed to increased survival resulting from improved medical care,would support the view that many deaths from CHD are delayed rather than premature deaths (Gomez-Marin et al. 1987).

IMPLICATIONS FOR DIETARY RECOMMENDATIONS

It seems to me incumbent upon public health officials who propose new dietary guidelines for the entire population for "prevention" of chronic and degenerative diseases, to recognize the immense improvements in health that have been achieved during this century. When infant mortality has declined, and death rates at all ages have fallen so that 75-85% of infants born survive to 65 years of age or beyond, and life expectancy at birth of males is 70-73 years and of females is 77-80 years, first consideration should be given to ensuring that these advances in health will not be jeopardized. The impact of risk factors for chronic anddegenerativediseases on the health and survival of individuals should not be exaggerated, especially when the evidence supporting the effectiveness of intervention is not consistent.

Physicians are justified in prescribing for patients whom they know will deteriorate progressively without intervention even though they may have little confidence that their prescription will be effective. For persons who are considered to be at high risk, vigorous intervention to reduce risk factors, including assumed dietary risk factors, is appropriate. It must be recognized, however, that risk factors are not causes of disease; they are variables associated with increased incidence,; they are probabilities. They do not apply to individuals, only on the average to populations. Great caution should, therefore, be exercised in proposing intervention for individuals who are basically well, particularly if promotion of that intervention creates fear of food and fear for health, apprehensions that can themselves be threats to health. It is also inimical to sound nutritional guidance when advocates of disease prevention by diet modification imply that certain foods have unique health-promoting or health-threatening properties rather than focusing on the total diet, which after all is what determines nutritional health.

A major contribution to public health has been achieved through knowledge of what constitutes a nutritionally adequate diet. The entire population is susceptible to nutritional deficiencies; these deficiencies can be prevented in the entire population through consuming a nutritious diet. This can be achieved through selection of appropriate numbers of servings from the majbr groups of foods to obtain as a wide a variety from each group as is possible. The situation is quite different for prevention of chronic and degenerative diseases. Individuals vary greatly in their susceptibility to such diseases; knowledge of relationships between diets and susceptibilityto these diseases is fragmentary; and there is no assurance that efforts to prevent them by mass dietary intervention will be either effective or cost effective (Becker 1987).

The major undesirable effect of development of an inexpensive, abundant, appetizing and nutritious food supply has been that a substantial number of people consume from it in excess of their needs and become overweight. Again, the entire

population is susceptibleto this problem and control of the balance between energy intake and expenditure can solve it. The problem is not as straightforward as many would like to believe, however, for metabolic, endocrine, genetic, and psychologic factors can all influence control of body weight. Lack of success in managing this problem, about which knowledge is much greater than for control of chronic and degenerative diseases, should give us pause to think critically about dietary proposals for disease prevention. Nonetheless, recommendations for energy intake that will just balance energy output to maintain appropriate body weight is based on sound scientific knowledge and applies to all.

Following these two general recommendations should ensure sound nutritional health. Following them should also ensure that the diet will be balanced so that excessive consumption of any one food or nutrient will not occur. It may not be inappropriate to suggest that attention be given to consuming a moderate amount of fiber and not too much fat,but it is highly inappropriate to imply that doing so, or selecting or rejecting certain foods, is the path to disease prevention. To classify nutritious foods as "bad" or "good" is the antithesis of sound nutrition education. We do not have knowledge that permits us to recommend with assurance a diet that will be effective in preventing specific diseases and, if we pretend that we do, we may be in danger of creating fears that will undermine confidence in the recommendation to consume a nutritionally adequate diet.

How then do we deal with the major medical problems of chronic and degenerative diseases? I agree with Stallones (1983) an epidemiologist who, after reviewing the current trends in health and CHD mortality, concluded "we cannot be distressing wrong if we hesitate before taking aggressive action to alter the dietary customs of the nation"; and with Kurtz (1976) that "where we do not have sufficient evidence we ought if possible to suspend judgement'. I would add that we ought to devote our resources, not to more efforts to identify additional risk factors for chronic and degenerative diseases, but to support research directed toward methods for establishing the nature of the metabolic and genetic defects that are truly predictive of development of these diseases at an early'age. Such studies are beginning to provide the information needed for identification, through genetic epidemiology, of those who may benefit from both diet and drug intervention (Scriver 1982).

We need to know the limits of effectiveness of dietary manipulation, we need to know much more about the genetic diversity of the population, and we need to 'be able to distinguish much more clearly among environmental and genetic effects and effects of senescence. It will then be much easier to conform with Thomas James (1981) admonitions during his Presidential address to his dolleagues in the American Heart Association, to be clear about when our advice is based on factual evidence, when it is from logical deductions, and when it is conjectural.

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