Diet-Heart Disease Risk

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Heart Disease Risk Factors

Cardiovascular diseases (CVD) account for over 50% of all deaths in the United States (Table 1). It has been known for a number of years that a variety of risk factors are involved in the development of these diseases. Studies within our population have identified various risk factors for heart disease, some of which are genetic and others related to elements of our life-style. Heart disease occurs more in men than in women and Caucasions are at greater risk than other races. There are some types of coronary anatomy which result in increased risk and genetic patterns of the lipid-carrying lipoproteins can cause a higher incidence of the disease. Life-style factors known to be related to an increased risk of CVD include high blood pressure, cigarette smoking, high blood cholesterol levels, diabetes, a sedentary life-style, obesity and the personality type characterized as Type A. These life-style risk factors can be effectively treated by stopping smoking, losing weight, increasing physical activity and certain changes in the dietary patterns of high-risk patients. The question to be addressed here is whether or not everyone, irrespective of his or her blood cholesterol level, should try, by diet, to reduce their blood cholesterol level and in theory their risk of developing heart disease.

Plasma Cholesterol and Heart Disease

There is no doubt that an elevated plasma cholesterol level leads to an increased risk of developing heart disease, as shown by international studies and by the Framingham Study. As each independent risk factor is added to the calculation of risk, we see that with increasing cholesterol levels, the other risk factors greatly increase disease incidence. A person with two risk factors and a low cholesterol level is at one-third the risk of developing heart disease as another individual with the same risk factors but a cholesterol level of 300 mg/dl. It obviously becomes imperative to treat the patient with a high blood cholesterol level because of its great impact on the overall risk profile.

Table 1. Cardiovascular Disease is the Number One Killer in the U.S.A.

Cause of Mortality	%
Diseases of the heart and vessels	51%
Cancer	20%
Accidents	5%
Pneumonia	2.5%
Diabetes	1.5%
Other	20%

The "lipid hypothesis" predicts that if a patient with a high cholesterol level and a corresponding high CHD risk can reduce his plasma cholesterol level, he will reduce his risk of developing CVD (Ahrens 1976). At the present time we have some evidence for the "lipid hypothesis" but it is far from proven in terms of a linear relationship between reducing cholesterol levels and reducing risk, or in terms of the efficacy of different modalities for lowering the plasma cholesterol level. Irrespective of the validity of this hypothesis, it is important to determine how to effectively lower plasma cholesterol levels and the role that diet may play in determining cholesterol levels in the population.

Plasma Cholesterol and Diet

Epidemiological studies of cultures with various incidences of heart disease have shown that when the intake of saturated animal fats in the diet is high, so are blood cholesterol levels and the incidence of heart disease within that population. These studies (Stamler, 1979) have shown that a number of dietary factors are related to an increased risk of heart disease (Table 2). The only foodstuffs found to be negatively related to heart disease risk are total carbohydrates and grains and starchy vegetables. Such studies have led to a number of dietary recommendations. With the variety of dietary information being given to the American public, it is not surprising that some confusion exists in the public's mind as to what is "good food" and what is "bad food."

The "diet-heart disease" relationship suggests that patients reduce caloric intake to maintain ideal body weight, reduce total caloric intake from fat to 30% of total calories with 10% from saturated fat, 10% from monounsaturated fat and 10% from polyunsaturated fat, and to reduce dietary cholesterol intake to less than 300 mg/day. Before we exhort the American public to change their diets, I believe we need to address three questions: (1) Will the recommended diet reduce the plasma cholesterol levels of a majority of the population or is it even the best diet to recommend? (2) If plasma cholesterols are reduced, will that result in a significant reduction in CHD incidence? (3) Is the recommended diet free of any potential long-term side effects? To date, we do not have definitive answers to any of these questions (Ahrens, 1976; Mitchell, 1984; Oliver, 1981; Olson, 1979).

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Table 2. Correlations Between Dietary Factors and Coronary Heart Disease Mortality

(+)	(−)
Total calories	Total carbohydrates (% cal)
Total protein (g, % cal)	Grains & starchy vegetables (cal, % cal)
Animal protein (g, % cal)	
Total fat (g, % cal)	
Saturated fat (g, % cal)	
Monounsaturated fat (g, % cal)	
Cholesterol (mg)	
Butter (cal, % cal)	
Dairy products (cal, % cal)	
Eggs (cal, % cal)	
Meat & poultry (cal, % cal)	
Sugar & syrup (cal, % cal)	
Tobacco (lb)	
Income ($)	

Efficacy of Cholesterol-Lowering Diets

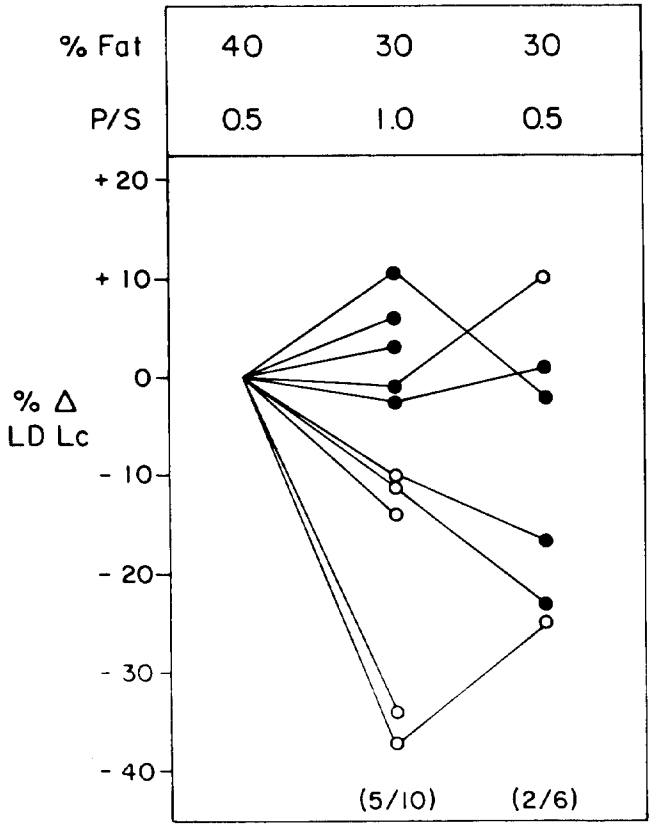
Numerous studies have shown that plasma cholesterol levels can be lowered by shifting from a saturated, animal fat diet to a polyunsaturated, vegetable fat intake. However, the recommended diet shifts the polyunsaturated to saturated fat ratio (P/S) to a modest 1.0 which has been shown to have a variable effect on plasma cholesterol levels (Wolf and Grundy, 1983). Only 50% of subjects exhibited a cholesterol lowering when shifted from a high fat, low P/S diet to the "prudent diet" and only two did so significantly (Figure 1). Reducing fat calories and increasing the P/S ratio had no effect on the plasma cholesterol levels in half the study group. We do not know how the American population will respond to such a dietary change.

These results point out one consistent finding of all clinical studies of the relationship between dietary fat and plasma cholesterol levels; a high degree of patient-to-patient variability in response to dietary constituents. People, unlike inbred strains of laboratory animals, are a metabolically heterogeneous population exhibiting variable responses to dietary fat. This heterogeneity makes it difficult to predict the response of any single patient to any specific diet or to randomly predict the best diet for a specific hypercholesterolemic patient.

The role of dietary cholesterol in determining plasma cholesterol levels is even more complex since cholesterol comes not only from our diets but also from our own production of this sterol. As shown in Table 2, approximately 25% of the total daily turnover of cholesterol comes from the diet. On average, 60% of dietary cholesterol is absorbed while the body's production accounts for the majority of the input. Numerous clinical studies have shown that in most individuals an increase in dietary cholesterol intake is compensated for by a decrease in the endogenous production of cholesterol, thus maintaining a constant plasma cholesterol level. Other compensatory mechanisms known to exist in man include an increased biliary excretion of cholesterol, increased catabolism of cholesterol to bile acids, and/or accumulation of cholesterol in the bulk tissues of the body (McNamara, 1982). At the present time, we have no way of knowing which patients are sensitive to dietary cholesterol and which ones are not; however, studies would suggest that the majority are effective compensators who can handle dietary cholesterol (Samuel et al., 1983).

Studies carried out in free-living out-patients treated with the currently recommended "prudent diet" have not been that encouraging in terms of the cholesterol lowering response (Multiple Risk Factor Intervention Trial, 1982). These clinical studies should be expected to be the most effective in terms of cholesterol lowering since the patients are taught and followed by a trained dietician. Dietary changes made by an untrained public in a somewhat random manner would not be expected to exceed the cholesterol-lowering response obtained under such controlled clinical trial conditions.

Figure 1



Wolf & Grundy (1983) J.Nutr. 113:1521-1528

Response of plasma low density lipoprotein (LDL) cholesterol to dietary fat changes. Patients were fed a 40% fat, P/S 0.5 diet followed by a 30% fat, P/S 1.0 and 30% fat, P/S 0.5 diet and percent change in LDL cholesterol determined (Wolf and Grundy, 1983). Expected changes (0) and unexpected changes (0) according to current diet-heart theory.

Table 3. Cholesterol Mass and Metabolism in Man

Total body cholesterol:	145	g
Metabolically active pool:	95	g
Plasma cholesterol pool:	6.5	g
Dietary cholesterol intake:	0.5	g/day
Absorbed dietary cholesterol:	0.3	g/day
Endogenous cholesterol synthesia:	0.7	g/day
Absorbed plus synthesized:	1.0	g/day
Metabolic requirement:	0.25	g/day
Excess:	0.75	g/day

[Calculated for a 70 kg man with a plasma cholesterol of 210 mg/dl]

Cholesterol Lowering and Heart Disease Incidence

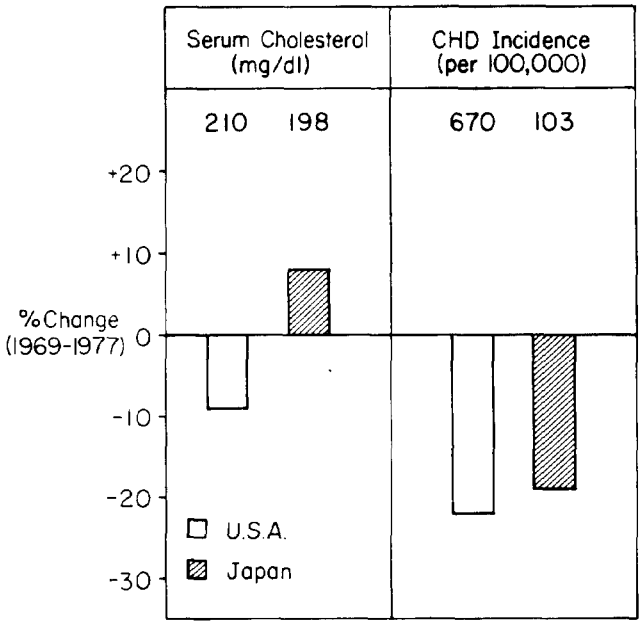
Even if the recommended diet were effective in lowering plasma cholesterol levels in the majority of the population, should we expect to see that result in a reduction in the heart disease incidence in the population? As pointed out by a number of investigators and by the results of the National Pooling Project, the relationship between plasma cholesterol levels and heart disease incidence is not a linear relationship but rather a curvelinear correlation with a sharp increase occurring at cholesterol levels above 240 mg/dl (Table 4). The average plasma cholesterol level in this country for men between the ages of 40 and 59 years is 210 mg/dl; only some 20% of the population have cholesterol levels above 240 mg/dl and are clearly at risk. One must question the advisability of treating the whole population in order to benefit a few (Hulley et al., 1981).

The recently-reported results of the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) (1984) have served as a justification for recommending that everyone lower their plasma cholesterol in order to reduce CVD risk. This trial was specifically a drug trial carried out in hypercholesterolemic men and, as shown in Table 5, extrapolation to the general population in terms of diet is difficult to justify (Consumer Reports, 1985). This study does, however, provide the strongest evidence to date that lowering an elevated plasma cholesterol with the drug cholestyramine can reduce heart disease incidence. Whether similar benefits can be achieved by lowering an abnormally high cholesterol level by diet which functions by a totally different mechanism remains uncertain.

Table 4. Total Serum Cholesterol and Heart Disease Risk

Quintile	*Total Serum Cholesterol* (mg/dl)	*Standardized Incidence Ratio* (per 10,000)
I	<194	72
II	194 - 218	61
III	218 - 240	78
IV	240 - 268	129
V	>268	150

Figure 2



Relationships between changes in plasma cholesterol levels and coronary heart disease (CHD) incidences in USA and Japan between 1969 and 1977. Absolute values as of 1977 presented numerically.

And what about plasma cholesterol levels and dietary patterns of societies with low heart disease mortality rates? One example is shown in Figure 2. The plasma cholesterol levels in Japanese males have gone up between 1969 and 1977 to a level almost equal to our own, in part due to a 220% increase in saturated fat intake (Research Committee on Familial Hypercholesterolemia in Japan, 1983). In contrast to what might be predicted, heart disease incidence over that same time period has actually decreased (Levy, 1981). The most significant difference is in the absolute heart disease death rate in Japan which is one-sixth that found in the USA. It is hard to account for the observed difference in heart disease by the 12 mg/dl difference in plasma cholesterol levels. Perhaps it is time for the epidemiologists to concentrate on this absolute difference in terms of what risk factors other than plasma cholesterol levels and diet may be involved. One thing that can be seen is that there is little

Table 5. Relationship Between the Findings of the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) and Dietary Recommendations to the Public

Comparison	*LRC-CPPT*	*Prudent Diet*
Subjects:	Men	Men, women, children
Cholesterol:	292 mg/dl	210, 190, 160 mg/dl
Treatment:	Drug	Diet
Mechanism:	Excretion	Redistribution
Cholesterol reduction:	7%	4% - 6%
Heart disease reduction:	1.7%	? ? ?

justification in the assumption that the lower the cholesterol the lower the risk and that cholesterol levels of 160 mg/dl are somehow ideal.

Risks of the Prudent Diet

There are no known health risks in following a low-fat low-cholesterol diet, but there are some significant unresolved questions in terms of overall nutrition and in terms of whether or not we could expect an actual extension in life expectancy. For some segments of the population, problems could arise from the "good food - bad food" mentality of eating. Children and older women need a consistent supply of calcium for bone growth and maintenance which is supplied by dairy products, nutrients which have been criticized. Iron needs are met from meats and a severe reduction in intake could have medical consequences. This group is well aware of the nutrient value of beef and pork, yet the advice has been to reduce consumption. Fixed and low-income groups obtain high quality protein and other nutrients from the low-cost egg, which has come under constant attack as a major source of dietary cholesterol. When people start to fear some of the quality components of their diet, we all should be concerned about the overall nutritional pattern of the population (Consumer Reports, 1985).

Quandaries still exist between the relationship of a very low plasma cholesterol level and increased risk of colon cancer in men (Salmond et al., 1985). If we could lower the plasma cholesterol levels of the population by diet, we would lower not only the levels of those at the high end of the curve but also the levels of those at the low end, possibly putting them at risk for a different cause of mortality. Finally, we must ask the question of overall life-expectancy, for no clinical trial, whether diet or drug, has shown a decrease in overall mortality. Indeed, the life expectancies for 40 year-old men in countries with high versus low rates of coronary heart disease mortality differ by only a few months.

Heart Disease Risk Intervention

Scientific proof and public policy are not based on the same criteria and the needs to reduce the epidemic of cardiovascular disease deaths in this country are of great importance. The hypercholesterolemic patient needs to be found and treated, first by diet and then by drugs, if necessary. The population needs to lose weight and get more exercise, stop smoking, normalize blood pressure and become more aware of the benefits of a balanced diet. A major concern is that many people will make dietary changes out of fear without knowing whether they are at increased risk for heart disease due to an elevated plasma cholesterol level. Perhaps the best advice for the public was given by the Food and Nutrition Board in their 1980 report *Toward Healthful Diets* "Good food that provides appropriate proportions of nutrients should not be regarded as a poison, a medicine or a talisman. It should be eaten and enjoyed."

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