DIET AND CARDIOVASCULAR DISEASE PREVENTION: WHAT WORKS?

Linda Van Horn, Ph.D., R.D.
Northwestern University Medical School

Rae-Ellen Kavey, M.D.
SUNY Health Science Center at Syracuse

ABSTRACT

Diet is routinely recommended as the primary strategy for the prevention and treatment of high blood cholesterol. The National Cholesterol Education Program (NCEP), the American Heart Association (AHA), and a host of other health and medical organizations have advocated a diet low in total and saturated fat and cholesterol for reducing risk of cardiovascular disease. What is the evidence supporting these guidelines and the expected efficacy of dietary treatment? There is growing awareness that despite well-documented rationale for the dietary approach, many eligible patients are not routinely prescribed dietary treatment, and among those who are, there is limited response. What are the obstacles in implementing effective dietary intervention for prevention of cardiovascular disease? What are both the theoretical and practical limitations to achieving long-term adherence to diet and what strategies have been shown to be most effective? A review of the data surrounding these diet–lipid relationships is presented along with recently tested and promising behavioral approaches to facilitating patient adherence.

(**Ann Behav Med** 1997, *19*(3):197–212)

INTRODUCTION/OVERVIEW

In considering what works in preventing and treating cardiovascular disease (CVD), diet is a proven contender, but achieving adherence is a challenge. Data from observational studies have repeatedly demonstrated that populations with lower intakes of saturated fat and cholesterol have lower rates of cardiovascular and, in many studies, all-cause mortality as well (1,2). There is now ample evidence that the association between level of serum cholesterol and associated risk of cardiovascular mortality is continuous and graded (3,4). On the basis of epidemiological and observational studies in men, women, African–Americans, and elderly individuals, as well as postmortem angiographic studies in young people, the National Institutes of Health (NIH) and the European Atherosclerosis Society have concluded that the relationship between elevated blood cholesterol and coronary artery disease is causal (2,5,6).

Other trials have tested the effectiveness of lipid-lowering intervention in primary and secondary prevention of coronary disease (5,7). A recent meta-analysis of four major, randomized trials reported 24% and 14% reductions in non-fatal and fatal myocardial infarction due to cholesterol-lowering (8). Many of these studies also involved multiple risk factors and diverse treatments resulting in overall favorable decreases in coronary heart disease (CHD) mortality, but the specific contribution of dietary change is difficult to quantify. A comprehensive review involving 10 prospective studies, 3 international studies, and 28 randomized controlled clinical trials reported consistent reductions in the incidence of ischemic heart disease by approximately 50% at age 40 and 20% at age 70 by decreases of 0.6 mmol/L (or about 10%) in serum cholesterol concentrations (9). Such reductions are readily achievable through careful adherence to dietary changes, especially in controlled settings.

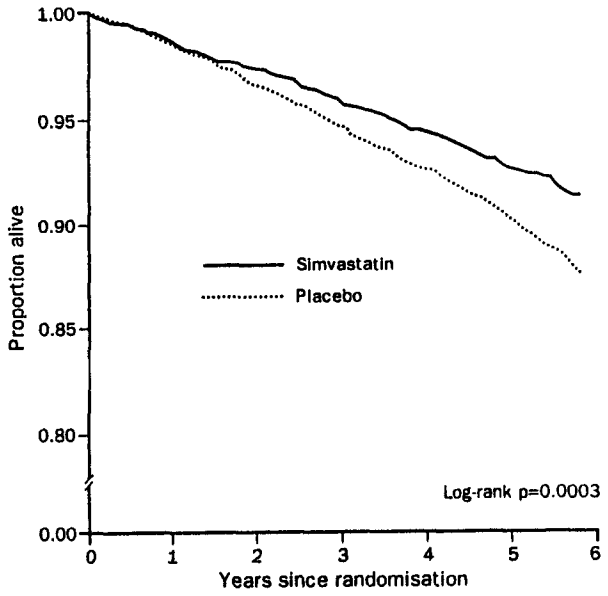
Compared to pharmacologic intervention, dietary intervention has produced less net reduction in total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels in randomized trials (10). Yet, as shown in Figure 1, two recent secondary intervention trials—one with drugs (Scandinavian Simvastatin Survival Study or 4S) and one with a Mediterranean, α-linolenic rich diet (Lyon Diet Heart Study)—reported reductions in cardiac deaths over five years that were remarkably similar (11–14). Likewise, adherence to the Cretan Mediterranean diet among French coronary patients offered protective benefits in reduced mortality that were statistically correlated with increases in omega-three (n-3) fatty acids and linolenic and oleic acids and decreases in linoleic acid, but not specifically with serum concentrations of TC, LDL-C, or high-density lipoprotein cholesterol (HDL-C) (11,12).

Major diet and angiography trials such as the Cholesterol Lowering Atherosclerosis Study (CLAS), the Lifestyle Heart Study, the Heidelberg Trial, the St. Thomas Atherosclerosis Regression Trial (STARS), and the Familial Atherosclerosis Treatment Study (FATS) have also reported favorable effects on blood cholesterol-lowering with varying degrees of dietary fat restriction (15–19). Greater declines in LDL-C were reported when total fat and saturated fat were aggressively restricted, especially in the metabolic ward setting (16). With total fat intake less than 20% of total calories, LDL-C levels declined 25% in three weeks, but after one year this decrease from baseline was 4% in free-living conditions (16). In the Lifestyle Heart Trial, LDL-C was reduced 37.8% when total fat was restricted to 10% of total calories, along with exercise, stress reduction, and other behavioral interventions, but the sample size was relatively small and follow-up limited (17). Extensive reviews of these dietary intervention studies appear elsewhere (2,20,21). The prevailing theme in these reviews is that aggressive dietary treatment with diets low in total and saturated fat induces blood cholesterol-lowering and favorable angiographic changes and potentially helps improve symptoms such as angina and other non-lipid related symptoms, but achieving and maintaining dietary adherence long-term remains a major challenge.

It was on the basis of cumulative scientific evidence from epidemiological and controlled clinical trials that the National Cholesterol Education Program (NCEP) advocated diet as the

Reprint Address: L. Van Horn, Ph.D., R.D., Northwestern University Medical School, Department of Preventive Medicine, 680 North Lake Shore Drive, Suite 1102, Chicago, IL 60611-4402.

Scandinavian Simvastatin Survival Study (4S)

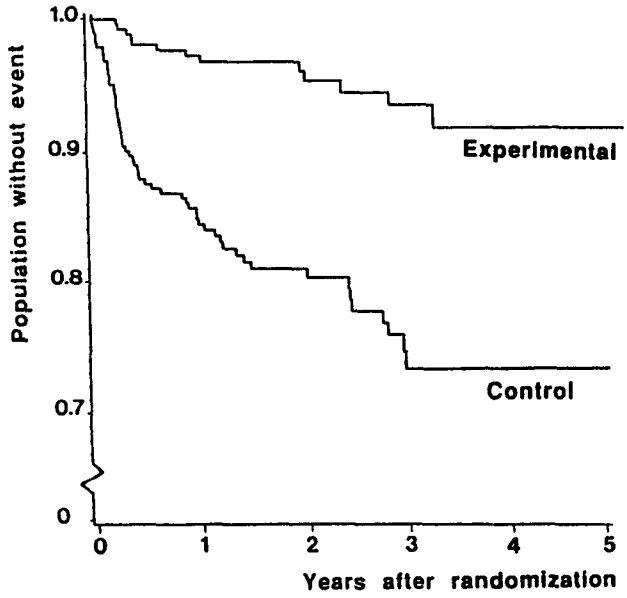


S	2221	2193	2160	2131	2097	2060	113
P	2223	2193	2152	2103	2059	2011	115

Kaplan-Meier curves for all-cause mortality
Number of patients at risk at the beginning of each year is shown below the horizontal axis.

Anonymous.: Randomized trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival 1994;344(8934):1383-1389.

Lyon Diet Heart Study
(Mediterranean α-linolenic rich)



Survival curves for combined cardiac death, nonfatal myocardial infarction, unstable angina, heart failure, stroke, and thromboembolism. Log rank test using only the time of the first event. Note the striking difference between the control and experimental groups within the first year ($P < 0.0001$).

Renaud, S., de Lorgeril, M., Delaye, J., Guidollet, J., Jacquard, F., Mamelle, N., Martin, J-L., Monjaud, I., Salen, Study (4S). *Lancet.* P. and Toubol, P.: Cretan Mediterranean diet for prevention of coronary heart disease. *Am. J. Clin. Nutr.* 1995;61(suppl):1360S-1367S.

FIGURE 1: Comparison in mortality rates between drug and diet studies: 4S and Lyon Diet Heart Study.

cornerstone of treatment in its first and second Reports of the Adult Treatment Panel, as well as the Population Based Panel and Pediatric Panel reports (22,23). It was on the same day in 1989 that both the NCEP and the American Heart Association (AHA) held press conferences announcing population-wide recommendations for the Step I and Step II diets as the primary strategies for prevention and treatment of high blood cholesterol (22,24).

Since then, considerable progress has been made in some aspects of the U.S. diet, and there has simultaneously been a steady decline in the nation's blood cholesterol level. In 1977, the average intake of total fat and saturated fat in the U.S. were 42% and 14%, respectively (25). In 1988–1991, they were 34% and 12%, respectively (25). A three-year United States Department of Agriculture (USDA) Survey reported total fat intake was 33% in 1994 (26). Although the vast majority of U.S. adults still eat more than the recommended 30% of total calories as fat, the country as a whole has had a major downward shift in total fat intake.

Paradoxically, obesity and weight gain are increasing at epidemic rates, especially among young women and children. This seems counterintuitive until levels of inactivity (addressed elsewhere in this journal) and several other aspects of the diet, such as energy sources and diet assessment methods, are further evaluated. Self-reported energy intake as well as activity expenditure are often unreliable, especially in obese individuals (27–29).

Another potential dietary contributor to the population's energy imbalance problem is the low mean intake of total dietary fiber estimated to be 14.82 grams in 1988–1991 (30). AHA recommends 25–30 grams of dietary fiber a day from whole grains, vegetables, and fruits (31). Because on average, the population eats less than half of the recommended minimum of five servings of fruits and vegetables per day, intake of more energy-dense but nutrient-poor foods is the likely consequence. For example, high-fructose corn syrup has increased from less than 25 pounds per capita in 1980–1983 to over 52 pounds per capita in 1994 (32). Simultaneously, despite increased use of non-caloric sweeteners, overall use of caloric sweeteners has increased 13.4% during this same time period (33). Any or all of these factors may be contributing to weight gain.

This article will address the role of diet in preventing and treating cardiovascular disease by reviewing the dietary factors that contribute to effective lipid-lowering and the population strategies that have reportedly been effective in improving dietary adherence. It has been estimated that approximately 52 million Americans are candidates for diet therapy to reduce high blood cholesterol (see Table 1); yet according to recent surveys, less than 10% have been prescribed such diets (34,35). Concerns regarding limited or ineffective prescription of the Step I or Step II diets in favor of pharmacologic intervention will be discussed. The poten-

TABLE 1

Percentage of the Adult Population Based on NHANES III Lipid Data Who Are Candidates for Dietary Intervention by NCEP Criteria[1]

Population Group	Borderline-High-Risk (LDL 130–159 mg/dL); ≥2 Risk Factors	High-Risk (LDL ≥160 mg/dL)	CHD and LDL >100 mg/dL	Totals
All persons	7	16	6	29 (74)[2]
Ethnicity:				
Mexican–American	5	12	4	21 (63)
Non-Hispanic Black†	7	15	5	27 (78)
Non-Hispanic White	7	17	6	31 (74)
Sex/age in years:				
Men				
≥20	9	17	6	32 (74)
20–34	4	9	2	14 (61)
35–44	6	20	2	27 (73)
45–54	13	25	6	45 (74)
55–64	16	26	14	56 (83)
65–74	14	24	15	53 (77)
≥75	14	15	18	47 (81)
Women				
≥20	6	15	6	27 (73)
20–34	2	6	1	9 (51)
35–44	2	5	7	13 (55)
45–54	4	19	7	29 (85)
55–64	12	30	9	52 (80)
65–74	13	35	6	54 (83)
≥75	13	26	18	58 (89)

[1] Adapted from Sempos et al. (34).

[2] In parentheses are percent requiring fasting lipoprotein analysis (22, 23).

† All Hispanic persons were excluded.

tial impact of combined use of clinical and population-based dietary change will further be assessed.

EVIDENCE THAT DIET LOWERS RISK OF CORONARY HEART DISEASE

Observational data and controlled clinical trials suggest that each 1 mg/dl increment in LDL-C increases CHD risk by 1%. For every 1% decrease in TC and LDL-C, CHD risk is expected to decrease by 2–3%, and a 1 mg/dl decrease in HDL-C is associated with 2–3% increased risk (23). Studies in patients with CHD have reported that lowering LDL-C through dietary change combined with smoking cessation or medication resulted in less progression and measurable regression of coronary artery lesions compared to the control group (15–19).

It was on the basis of metabolic ward studies testing saturated fatty acids (SFA), cholesterol, and polyunsaturated fatty acids (PUFA), the major nutrients known to affect blood cholesterol at the time, that Keys and colleagues developed their classic formula in the 1950s that is still used to predict blood lipid response to dietary change (1,36,37). The formula incorporates their findings that saturated fatty acids raise total cholesterol twice the amount that polyunsaturated fat lowers it. Dietary cholesterol raises TC but proportionately less than saturated fatty acids. More specifically,

$$\text{Keys Equation: } C = 1.35\,(2S - P) + 1.5z$$

where C = change in serum TC (mg/dl), S = change in percent energy intake from saturated fatty acids, P = change in percent energy from polyunsaturated fatty acids, and z = difference in square roots of baseline versus treatment cholesterol intakes (mg/1000kcal). Subsequent modifications by Hegsted (38,39), Mensink and Katan (40), and Yu et al. (41) continue to concentrate on these fundamental dietary factors as well as assessing the impact of monounsaturated fatty acids (MUFA) and stearate and including overall impact on LDL and HDL. As summarized by Kris-Etherton and Yu (42), these predictive equations report that SFA raise and PUFA lower TC and LDL-C and monounsaturated fatty acids are half as potent as PUFA in lowering TC.

More recent studies among high-risk CHD patients have reported significant reductions in TC and LDL-C with diets very low in fat and/or strictly vegetarian (43,44). Studies comparing vegetarian versus non-vegetarian populations likewise favor the former (44–47). Also, beyond specific nutrient associations with lipids, the use of fruits and vegetables in a fat-modified diet has had favorable impact (47).

In addition to advances in qualifying and quantifying the impact of other dietary factors beyond those included in the Key's formula, there is also compelling evidence that an increase in HDL-C is strongly inversely related to cardiovascular events (2,7,23). To date, there is some evidence that SFA, MUFA, and PUFA may increase HDL-C levels, but no quantitative estimates have been confirmed (42). Studies have reported a protective effect on HDL when MUFA is substituted for SFA but not PUFA (40–42). No specific foods or nutrients have been linked to increasing HDL. Rather, behavioral changes related to weight loss, exercise, and smoking cessation are reportedly the non-pharmacologic factors that favorably influence HDL-C (23).

A more comprehensive formula that encompasses non-lipid as well as lipid components that predict blood cholesterol response has not yet been tested. The list of potential antiatherogenic factors continues to grow as new research provides data illustrating adjunctive lipid-lowering benefits beyond fat-modified diet alone. Antioxidants, soluble fiber, monounsaturated fatty acids, omega-three fatty acids, and other factors have also been reported to have favorable impact on blood lipids, but the composite effects have not been quantified. A brief review of these studies is summarized below.

Saturated Fatty Acids

Saturated fatty acids differ in chain lengths and in their respective impacts on TC and LDL-C levels. Individually, palmitic (16:0), lauric (12:0), and myristic (14:0) acids raise TC and LDL-C, but stearic acid (18:0) lowers them or has a neutral impact (42,48). Common food sources of these saturated fatty acids are comprised of mixtures of them. Since food labeling laws do not currently differentiate between types of saturated fatty acids, the practical relevance of this finding is limited. For example, isolated studies of chocolate and beef, both relatively rich sources of stearic acid, have reported little or no cholesterol-raising impact in some individuals following the Step I diet (49,50). Because these foods are also sources of palmitic and myristic acids, the overall recommendation is to limit intake along with other saturated fat-containing foods.

In an extensive review of the cholesterolemic effects of individual fatty acids, Kris-Etherton and Yu (42) evaluated data from well-controlled and well-defined experimental diet studies, using strict criteria regarding appropriate experimental design, endpoints, sample sizes, etc. Among individual saturated fatty acids, it was concluded that there is heterogeneity in cholesterol-

emic response, but the 12:0–16:0 saturated fatty acids are distinctly hypercholesterolemic with myristic acid apparently yielding the most potent effect (42). Stearic acid, though saturated, produces LDL-C lowering when substituted for lauric, myristic, or palmitic acid, and oleic and linoleic acids are hypocholesterolemic compared to the saturated fats. In general, total cholesterol and LDL-C responses are typically similar and parallel (51). Lauric, myristic, and palmitic acids all increase TC and LDL-C, and stearic acid lowers TC and LDL-C with apparently no impact on HDL-C or triglycerides (52).

Dietary Cholesterol

Dietary cholesterol is second only to SFA in its atherogenic impact on LDL-C, and recommended levels are less than 300mg/day or 200mg/day as part of the Step I or Step II Diet, respectively (22–24). Epidemiologic studies have reported that dietary cholesterol has an independent risk of CVD mortality that transcends the effect on lipids (53–57). On a practical level, by lowering SFA intake to the recommended levels, dietary cholesterol intake is typically automatically reduced. Since the greatest contributor of dietary cholesterol in the American diet is egg yolks, limiting total egg consumption will help reduce dietary cholesterol intake. Because of the body's endogenous synthesis, there is no biological requirement for dietary cholesterol, thereby eliminating the need for a minimum intake.

Unsaturated Fatty Acids

Since the early days of the development of the Key's formula, much data have accumulated that document LDL-C lowering benefits from not only PUFA but MUFA as well (48,58–61). Substituting either of these unsaturated fatty acids for saturated fat in the diet has resulted in favorable reductions in LDL-C. Because of concerns about overall safety and the fact that very few populations have consistently reported PUFA intakes $\geq$10% of total calories, the Step I–II Diets have advocated no more than this amount (23). In vitro, increased PUFA intake contributes to increased PUFA composition in the LDL-particle and may increase its susceptibility to oxidation, thereby increasing atherogenecity (51,59,62). It remains to be established whether this has clinical relevance.

On a practical level, it is difficult to achieve PUFA intakes much above 5–6% of calories without intentionally increasing use of margarine, salad dressings, and cooking oils. This approach is problematic for at least three reasons. First, it may be more effective for weight-conscious individuals to replace calories lost from saturated fat with less calorically dense options such as complex carbohydrates. Second, this approach may also result in increased intake of trans fatty acids, shown to raise TC (63,64). Third, there are some data suggesting that increased PUFA intake may reduce HDL-C levels (48,51).

Growing evidence supports a measured favorable impact of MUFA on TC and LDL-C. Studies comparing the benefits of substituting MUFA for PUFA sources report an HDL-C sparing effect of the former (48,62,65). Again, from a practical viewpoint, food labels do not currently list monounsaturated fatty acid content of foods, making it important for patients to recognize the most common food sources. Olive oil and canola oil are currently the most concentrated sources of monounsaturated fats available commercially. The Mediterranean diet, high in olive oil, has captured recent scientific attention as a comprehensive lipid-lowering plan (21,66,67). Because total fat content is typically higher than the recommended 30% of total calories, the potential for weight gain in the U.S. population with its significant obesity problem must be considered before population-wide recommendations can be made.

Weight loss among obese individuals following the AHA Step I Diet with elevated blood cholesterol levels intensified the reduction of LDL-C without decreasing HDL-C and possibly even increasing it (48,67,68). Weight loss in overweight, high-risk individuals is one of the most powerful strategies for reducing risk of CHD (68,69). [It is also more difficult to achieve and maintain long-term than qualitative changes in the diet (69)]. Dietary patterns that are lower in total fat also tend to be lower in total calories, but a combined diet plus exercise approach has been consistently advocated for maximum benefit (70–72).

Omega-Three Fatty Acids

Among the PUFA, the three major omega-three fatty acids are alpha linolenic (LNA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). The latter two are found in fish and fish oils. LNA is found in tofu, soy bean and canola oil, nuts, and flaxseed oil. Clinical trials have shown triglyceride-reducing benefits from fish oil therapy (73–75). Cross-cultural and prospective studies in Japan, Greenland, Zutphen, and Chicago reported lower CHD mortality among areas consuming more fish (2,20,73). Conversely, no association was demonstrated in two other large observational studies, the Physician's Health Study and the Health Professionals' Follow-Up Study (75,76). The Diet and Reinfarction Trial (DART) reported a 29% drop in all-cause mortality in men with CHD who were assigned to regular fish consumption (77). Despite these inconsistent findings and the absence of conclusive mechanisms, patients are advised to substitute fish for fatty meats, but fish oil supplements are not routinely recommended.

Trans Fatty Acids

Trans fatty acids are created when polyunsaturated fats are hydrogenated to produce margarine and shortenings. They also occur naturally from anaerobic bacterial fermentation in ruminants and exist in meat and dairy products (78). The normal cis form of polyunsaturated fatty acids lowers LDL-C, but may lower HDL-C as well (79). Very few controlled clinical studies have been conducted with sufficient evidence to draw definitive conclusions about its atherogenic potential. Compared with unsaturated fatty acids, trans fatty acids raised total cholesterol and LDL-C and lowered HDL-C, as well as increasing LP(a) levels in the few studies that have documented effects (40,80,81). Due to these limited findings, results should be interpreted with caution and specific dietary recommendations on trans fatty acids are as yet premature.

Summary of Lipid Response to Changes in Individual Fatty Acids

It is beyond the scope or purpose of this paper to review the full extent of reported reductions in TC and LDL-C in response to changes in individual fatty acids. Excellent, comprehensive reviews appear elsewhere (42,51,82). A summary of the most relevant data are presented in Table 2 (adapted from reference 42), illustrating predicted plasma total, LDL-C, and HDL-C responses to specific dietary changes as measured in tightly controlled feeding studies. Despite some differences in the specific equations, the common theme in comparing these predictive formulas devel-

TABLE 2
Predicted Plasma Total and Lipoprotein Cholesterol Response to Dietary Change, mmol/L (mg/dl)

Diet Change	Predicted TC Response			Predicted LDL-C Response		Predicted HDL-C Response	
	Keys[a]	Hegsted[b]	Mensink[c]	Hegsted[b]	Mensink[c]	Hegsted[b]	Mensink[c]
AAD1 → AAD2	−0.14 (−5.4)	−0.11 (−4.2)	−0.08 (−2.9)	−0.09 (−3.5)	−0.06 (−2.3)	−0.03 (−1.0)	−0.03 (−1.3)
AAD1 → Step 1	−0.70 (−27.0)	−0.41 (−16.0)	−0.23 (−8.9)	−0.33 (−12.6)	−0.19 (−7.4)	−0.12 (−4.8)	−0.07 (−2.8)
AAD2 → Step 1	−0.54 (−20.9)	−0.31 (−11.8)	−0.16 (−6.0)	−0.24 (−9.2)	−0.13 (−5.1)	−0.10 (−3.9)	−0.05 (−1.9)
AAD1 → Step 2	−1.02 (−39.5)	−0.66 (−25.6)	−0.35 (−13.6)	−0.52 (−20.1)	−0.30 (−11.5)	−0.21 (−8.2)	−0.11 (−4.2)
AAD2 → Step 2	−0.88 (−34.1)	−0.55 (−21.4)	−0.28 (−11.0)	−0.43 (−16.6)	−0.24 (−9.2)	−0.19 (−7.2)	−0.08 (−3.0)
Step 1 → Step 2	−0.49 (−18.9)	−0.25 (−9.7)	−0.12 (−4.7)	−0.19 (−7.4)	−0.11 (−4.1)	−0.09 (−3.3)	−0.03 (−1.1)
AAD1 → VLSF	−1.47 (−57.0)	−0.91 (−35.3)	−0.47 (−18.1)	−0.71 (−27.4)	−0.40 (−15.4)	−0.30 (−11.6)	−0.14 (−5.6)
AAD2 → VLSF	−1.18 (−45.6)	−0.80 (−31.1)	−0.40 (−15.3)	−0.62 (−24.0)	−0.34 (−13.0)	−0.28 (−10.7)	−0.11 (−4.4)
Step 2 → VLSF	−0.49 (−18.9)	−0.25 (−9.7)	−0.12 (−4.5)	−0.19 (−7.4)	−0.10 (−3.8)	−0.09 (−3.4)	−0.04 (−1.4)
AAD1 → MUFA1	−0.70 (−27.0)	−0.41 (−16.0)	−0.25 (−9.8)	−0.33 (−12.6)	−0.24 (−9.1)	−0.11 (−4.1)	−0.02 (−0.8)
AAD1 → MUFA2	−0.70 (−27.0)	−0.41 (−16.0)	−0.24 (−9.4)	−0.33 (−12.6)	−0.22 (−8.4)	−0.11 (−4.4)	−0.05 (−1.8)
AAD2 → MUFA1	−0.56 (−21.6)	−0.31 (−11.8)	−0.18 (−6.9)	−0.24 (−9.2)	−0.18 (−6.8)	−0.08 (−3.2)	+0.01 (+0.5)
AAD2 → MUFA2	−0.56 (−21.6)	−0.31 (−11.8)	−0.17 (−6.5)	−0.24 (−9.2)	−0.15 (−5.8)	−0.09 (−3.5)	−0.01 (−0.5)

AAD: average American diet, VLSF: very-low saturated fat diet, MUFA: high MUFA diet, [a] Keys et al. (192), [b] Hegsted et al. (39), [c] Mensink and Katan (40).

Adapted from Kris-Etherton P, Yu S: Individual fatty acid effects on plasma lipids and lipoproteins: Human studies. *American Journal of Clinical Nutrition* (in press, 1996).

oped by Keys, Hegsted, and Mensink is that the greatest reductions in TC and LDL-C are achieved when the average American diet is replaced by a diet very low in saturated fat (i.e. 3% of total calories or less) (36,37,39,40). This approach shifts the majority of energy intake to complex carbohydrate foods.

Fiber and Lipids

In addition to the recognized lipid-lowering benefits of dietary fat reduction, alterations in both the quality and quantity of complex carbohydrates and fiber can yield additional lipid reduction. Observational data have reported inverse associations between total dietary fiber intake and risk for CHD and all-cause mortality (83). Over the last two decades, numerous clinical trials have explored the impact of fiber, most often soluble fiber in the form of oats or psyllium, on blood total cholesterol and LDL cholesterol. Both short-term and longer-term studies have reported additional 2–3% reductions in LDL-C and total cholesterol with two or more servings of soluble fiber from oats within the context of Step I or II Diet (84–88). Together, lowering saturated fat and cholesterol intake and increasing sources of soluble fiber can have additive benefits, especially among those with the highest blood cholesterol levels. Behaviorally, emphasizing increased intake of soluble fiber-rich foods can have a favorable impact on lipid-lowering simply by triggering substitution of carbohydrate for fat (89). Rather than focusing on sacrificing fat-rich foods, people are encouraged to enjoy eating more fruits, vegetables, and whole grains, thus shifting the emphasis away from deprivation to enhanced intake. Current intake of total dietary fiber is approximately 13 grams per day, only half of the recommended amount of 25–30 grams per day (31). If population-wide increases in fiber intake occurred, the concomitant impact on dietary fat reduction could yield mean cholesterol-lowering benefits of at least 2–3%, potentially without further restricting saturated fat intake. This remains a viable public health strategy that has yet to be effectively implemented.

Soy Products and Phytochemicals

Soy protein substituted for animal protein also lowers blood cholesterol (90). It has been estimated that 25 grams of soy protein per day can reduce serum total cholesterol by 9 mg/dl with greatest responses estimated at 7–17% occurring among individuals with TC > 260 mg/dL (91). Soy contains phytochemicals, specifically the isoflavones genestein and daidzen, that may help lower blood cholesterol. Tofu and other soy protein products are rich sources of these isoflavones. Other phytosterols, tocotrienols found in garlic, and other non-nutrients like fiber have been studied with favorable results (90). The limiting amino acid in soy is methionine that may help reduce homocysteine levels, thereby offering further protective benefits against CHD, independent of the lipid-lowering efforts (91). Conclusive data and specific mechanisms have yet to be quantified.

Salt

On the basis of epidemiologic data and clinical trials on salt restriction, the AHA recommends a limit of 6 grams of sodium chloride per day or 2.4 grams of sodium (31). In reporting findings from INTERSALT, a weak but significant correlation was observed between standardized blood pressure measurements and urinary sodium excretion in over 10,000 individuals from 52 centers around the world (92,93). Multiple regression analyses across populations indicated that with 100 mmol/day lower sodium intake, the rise in systolic over diastolic blood pressure is less by 10/6 mm Hg on average from age 25 to 55 years (94). In hypertensive individuals, a meta-analysis involving 23 studies and 1,500 patients reported reductions of 4.9 mmHg and 2.6 mmHg in systolic and diastolic pressure, respectively (95). Similarly, the Trials of Hypertension Prevention (TOHP) reported significantly reduced blood pressure resulting from weight reduction as well as salt restriction in men and women with high normal blood pressure (96).

Despite periodic objections suggesting that sodium intake is unimportant in normal, healthy, non-sodium sensitive individuals (97), there is growing evidence that moderating sodium intake offers public health benefits. Current estimates of sodium chloride intake in the United States range from 7.5 to 10.0 grams per day. The biological requirement for sodium is less than 500 mg/day. Overall, it is feasible to reduce sodium intake, and despite some

variability in blood pressure response, less than 2,400 mg/day is considered beneficial to overall cardiovascular risk reduction (31).

Other Nutrients, Antioxidants, Homocysteine

Other nutrients have also been explored regarding their impact on blood lipids. Antioxidant vitamins E, C, and β-carotene delay and reduce the oxidation of LDL-C in vitro and may be especially important among smokers (20,98–100). Observational studies in female nurses and male health professionals have reported favorable reductions in CHD risk among those who took vitamin E supplements, but no intervention trials have yet been completed (99). Use of vitamin E supplements were associated with a 35% reduction in CHD after adjustment for other risk factors, multiple vitamins, β-carotene, and vitamin C (98,99). Conversely, iron has been suggested to serve as a pro-oxidant in the arterial wall, thereby potentially contributing to LDL-oxidation, endothelial injury, and myocardial injury (101). Studies on iron and risk of CHD are as yet inconclusive.

Elevated levels of homocysteine damage endothelial cells and promote arterial occlusion (102). Homocysteine is produced by the demethylation of methionine, an amino acid found mainly in animal protein foods. This conversion is dependent upon folic acid, with vitamins B_6- and B_{12}-dependent enzymes playing a role as well. If these factors are reduced or inactivated, elevations in homocysteine can result. Diets rich in dark green and yellow fruits and vegetables and whole grains that provide an optimal amount of 400 mg/day of folate are adequate to produce serum folate levels of 15 μmol/L.

SO, IF DIET WORKS, WHY THE RELUCTANCE TO PRESCRIBE IT?

On the basis of these findings, a diet reduced in total and saturated fat, cholesterol, trans fatty acids, homocysteine, sodium, alcohol, and excess calories and increased in dietary fiber (especially soluble fiber), antioxidants, folate, vitamin B_6, and n-3 fatty acids will lower cardiovascular risk for most people. The resulting dietary pattern is rich in fruits and vegetables; whole grains and cereals; low-fat and skim dairy products; and fish, lean meats, and/or soy protein foods. Foods should be cooked without added saturated fat and, when necessary, cooked in a small amount of liquid vegetable oil, preferably olive, canola, or other monounsaturated non-hydrogenated liquid oil. Egg yolks should be limited to less than two per week.

Why is there any resistance to encouraging such dietary adherence? Are there overarching issues that make diet seem impotent as the primary intervention, regardless of how aggressively it is prescribed? Three of many factors that could inhibit such treatment decisions include the impressive lipid-lowering results of pharmacologic intervention that appear to guarantee success in most patients, the perception that genetics overwhelms any potential lipid response to diet, and the dismal results of weight loss efforts that appear to prevent lipid responses to qualitative changes in the diet.

DIET AND/OR DRUGS: NON-ADHERENCE MEANS NON-RESPONSE

The Step I and II Diets encompass the dietary factors shown to be helpful in improving risk status, but the challenge of achieving adherence may seem unreasonably daunting, thereby prompting the decision to skip to one of the more consistently reliable pharmacologic approaches instead. The NCEP has conducted four Cholesterol Awareness Surveys since 1983 that monitor the changing attitudes and behaviors of practicing physicians and the public over time (35).

Progress has been made in numerous parameters, but dietary intervention efforts continue to fall short of the desired outcomes. For example, regarding the main NCEP message "Knowing Your Cholesterol Level," 3%, 7%, 37%, and 49% of the public surveyed knew their cholesterol levels in 1983, 1986, 1990, and 1995, respectively (35). Also, in 1986, 65% of physicians surveyed knew their own cholesterol levels versus 90% in 1995. Physicians reported initiating dietary therapy in their patients at levels of total cholesterol (mg/dl) above 200 in 1990 and 1995 versus levels of 260 and 240 in 1983 and 1986, respectively. Conversely, even in 1995, only 8% of the public surveyed reported being prescribed a cholesterol-lowering diet by their physicians, down from 9% in 1990 and up only slightly from the 3% and 4% reporting such behavior in 1983 and 1986. Part of the reason relatively few people report being prescribed a diet may be due to physicians' lack of confidence in patient compliance. Only 1% of physicians surveyed estimated that "all" patients complied with diet; 17% reported "most," 59% reported "some," and 22% estimated that "few" patients complied with diet. There appear to be major discrepancies between the numbers of patients who are eligible for dietary intervention and those who have either been prescribed a diet or sought dietary counseling on their own.

GENETIC INFLUENCES ON LIPID RESPONSE: NOT A DETERRENT TO DIETARY INTERVENTION

Despite aggressive dietary treatment, there is recent evidence that certain genetic polymorphisms influence lipid response. Individuals characterized with subclass pattern Type B have small, dense LDL and appear to be more responsive to low-fat diets than those with Type A pattern (103,104). For some patients with elevated triglycerides and lower levels of HDL-C and without markedly elevated LDL-C, a very low-fat, high-carbohydrate diet may have an adverse effect by further reducing HDL-C (104,105). In such cases, replacement of SFA with MUFA may be especially beneficial. Obesity plays a major role in exacerbating these genetically-altered lipid responses, and weight loss should be encouraged wherever possible. It is not possible to predict what percent of the population may have inherited genetic traits that limit lipid response to diet, but it appears that qualitative adjustments to the diet may even benefit some of these patients. More data are needed to determine whether more precise dietary recommendations can be targeted to certain phenotypes that will more accurately predict lipid response even in these individuals.

OBESITY: ENEMY NUMBER ONE (?)

Independent of the qualitative objectives of dietary adherence, obesity both directly and indirectly plays a major causal role in coronary heart disease and stroke (106). It contributes to dyslipidemia, diabetes, and hypertension, and its strong correlation with reduced physical activity further exacerbates cardiovascular disease (107–109). The increased national prevalence of obesity, currently estimated at 30% among adults and children, demands more effective strategies for prevention and reversal of this urgent public health problem (110,111).

Excess body weight has been causally linked with deleterious changes in the lipoprotein profile (107). As body mass index increases, serum total cholesterol, LDL cholesterol, and triglyceride levels rise, and the HDL cholesterol level falls. These changes are seen in both adults and children. Several studies document that the lipoprotein abnormalities are induced with weight gain and

reversed with weight loss, again across age groups (70,112). Particularly in obese children, even small amounts of weight loss can result in dramatic normalization in the lipid profile (112). Per kilogram of weight lost, estimated reductions in triglyceride and TC average 1.33 and 1.94 mg/dL, respectively (113). Obesity, therefore, has deleterious but reversible effects on the lipid profile.

Obesity contributes to Type II diabetes, a potent risk factor for cardiovascular disease (108). Abnormalities in insulin secretion and action have been well-described with obesity (114,115). Weight loss alone is an effective treatment for adult onset diabetes. The association of obesity with insulin resistance, hypertension, increased serum triglyceride levels, and reduced HDL cholesterol levels is collectively known as Syndrome X and is associated with increased risk for cardiovascular disease (116). Even in children, the incidence of non-insulin dependent diabetes mellitus (NIDDM) associated with obesity is increasing (117), but weight reduction is effective therapy in this age group as well (118).

There is also a well-established direct relationship between body size and blood pressure, with obesity as an important independent risk factor for the development of systemic hypertension (119–121). In both adults and children, weight loss alone is often the only treatment needed for reducing mild hypertension, but adherence to weight loss regimens long-term is generally poor (122,123).

PREVENTION AND TREATMENT OF OBESITY

The overwhelming difficulty of losing weight among adults points to the need for primary prevention beginning early in life as the most strategic approach (124–126). Obesity is known to have a strong familial basis, with twin studies identifying both genetic and environmental origins (123,127). Recent reports have identified specific genetic loci associated with obesity, but lifestyle, environmental, and cultural factors are equally important (128). Eighty percent of children with two obese parents will be obese as adults (129). Between 60% and 80% of obese adolescents will become obese adults (130). As the low-risk child becomes an obese adult, the acquisition of excess risk in terms of higher blood pressure, increased LDL cholesterol, decreased HDL cholesterol, and non-insulin dependent diabetes mellitus is largely mediated by the acquisition of excess weight gain (131–133).

A recent NIH conference on Voluntary Methods for Weight Loss and Control concluded that most existing adult obesity interventions are ineffective, with one-third to two-thirds of the weight loss being regained in one year and almost all weight being regained in five years (134). Training in calorie restriction and use of packaged diet foods are not effective long-term. The greatest success in treating obese adults has occurred with combined dietary fat restriction, behavioral skill development, and regular exercise (135,136). Other effective strategies include family-oriented intervention and booster sessions (124). Pharmacologic appetite suppressants and gene therapy may offer promising options in the future for some patients, but inevitably diet and energy balance must be addressed.

Successful primary prevention of obesity has been reported among children who were initiated on a prudent diet at age three months and followed for three years compared to a control group (137). Favorable initial responses have also been reported to a family-based health education program incorporating behavioral change, diet, and exercise beginning in 2- to 4-year-olds (138). In older children, ages 8 to 12, combined behavioral, dietary, and exercise change has resulted in reduced cholesterol levels after six months and decreased obesity in a ten-year follow-up study (139,140). The "Shapedown" program has reported successful group intervention in 11- to 13-year-olds during a live-in camp experience that is generalizable to other settings as well (141). Cognizance of the causes of obesity, behavioral skill development, and social support are combined with increased physical activity to induce weight loss and establish weight maintenance over time. Behavioral, family-based treatment for obesity among those who have been followed for several years has yielded modest success in maintaining initial weight loss and/or stabilizing percent of ideal body weight below baseline levels (139–141). Successful long-term weight losers have a high degree of self-efficacy concerning their abilities to lose weight, further implicating parents' confidence in the efficacy of weight loss training as a potentially important factor in successful weight control of their children (142–144). Primary prevention remains the preferred strategy for physiological as well as emotional reasons, including early intervention in families prone to obesity regarding appropriate diet and exercise patterns.

BEHAVIORAL APPROACHES TO ENHANCE ADHERENCE: THEORETICAL BASIS

It has long been recognized that knowledge alone is not sufficient motivation to change behavior (145). Metabolic ward and other controlled feeding studies have demonstrated the efficacy of dietary intervention, illustrating that fat-modified diets can dramatically lower blood total and LDL cholesterol levels, especially when accompanied by weight loss in obese individuals (61,70). Such results are often perceived as being non-generalizable due to the presumed inability to achieve adherence among free-living individuals. Intervention studies have reported disappointing or at best modest lipid-lowering results, even when trained dietitians and/or other knowledgeable health professionals are involved in the intervention process (146–148). At least two limitations must be addressed.

First, dietary assessment methodology is often inadequate, inaccurate, or non-existent in these studies. This makes it impossible to evaluate true intake and determine the extent of the inter- versus intraindividual variability in lipid response to diet (38,39,48). Because, to date, there are no standardized biomarkers for dietary adherence, neither acute nor long-term adherence to the diet can be objectively calculated, thereby leaving no option but the use of self-reported data.

Recent evidence from the behavioral literature suggests that a second major limitation in previous studies could be the method used to deliver the dietary intervention. Earlier intervention studies typically applied a didactic, informative approach with little attention paid to what are now recognized as important differences in learning styles or levels of motivation to change behavior (149). New behavioral models identify psychosocial factors that influence food choices and delineate the process of motivating changes in behavior (150). Several behavioral models have evolved over time and these are potentially additive. For example, the individual's concept of personal vulnerability was first characterized via the Health Belief Model (HBM) and found to be predictive of positive health-related behaviors (151,152). The Social Learning Theory (SLT) incorporates behavior modification methods that include cognitive, interpersonal, and environmental influences on eating (153). Basic components of this approach include self-monitoring and analysis of behavior; self-management including stimulus control of external cues; replacement of less desirable (i.e. high-fat foods) with more healthful behaviors; and reinforcement of desirable behaviors (145).

Although not originally designed to address eating behaviors, these models have been adapted to modern dietary intervention studies. Over the past ten years, research on the transtheoretical model and the stages of change construct have yielded valuable and useful data regarding how, why, and when people change behavior (154). With its origins in the treatment of addictions, great strides have been made in applying these models to changing eating behavior as well (155–157).

To summarize this large and growing body of evidence, behavior change is achieved through a series of stages including precontemplation, contemplation, preparation, action, and maintenance (156). Since most previous dietary intervention studies applied approaches suitable for the action stage, it is not surprising that uniform adherence was not achieved. It is likely that some of these participants were overwhelmed, uncommitted, or simply unwilling to adapt the recommended eating pattern. Because dietary assessment data in these studies are often limited to a single day of recall or very few days of food records, the validity of these data is also problematic. Even if level of adherence was adequately captured, it may reflect an acute rather than a usual state. Ultimately, analyses of these data could easily produce negative findings regarding the impact of diet on lipids, interpreted as lack of efficacy, when in reality the results were due to inconsistent or non-adherent behavior. In other words, the diet will work only if it is followed.

COMBINING BEHAVIORAL AND DIETARY CHANGE STRATEGIES: EMERGING EVIDENCE OF SUCCESS

Glanz et al. (155) reported the use of stages of change in assessing fat and fiber intake in the Working Well Project. At that time (1994), their review of the literature cited only two studies that adapted the stages of change model regarding dietary fat reduction in addressing issues of healthy individuals (157,158). Previous diet-related, theoretically-driven research had concentrated primarily on weight loss. Together, these findings further supported the theory that individuals can be classified into mutually exclusive stages of dietary change and that dietary fat intake decreases and fiber intake increases as stages progress toward action and maintenance (155,157–160).

The dietary intervention program designed for the Women's Health Initiative (WHI), patterned after the Women's Health Trial (WHT), focused on reducing total fat to less than 20% of baseline energy, thereby achieving saturated fat intakes below 7% of total calories (161). The WHT successfully achieved long-term dietary adherence (161–164). The food pattern goals emphasized whole foods and servings of fruits, vegetables, and grains rather than nutrients in order to simplify the process of dietary change. A mixture of psychosocial and behavioral principles have been similarly applied in the Women's Health Initiative currently in progress (165).

These principles include reinforcements and motivators, self-management skills, self-control skills, social support, relapse prevention, and training in other behavioral skills such as stress-management and assertiveness that have been effective in influencing behavior change (165,166). Like the Multiple Risk Factor Intervention Trial (MRFIT), the Women's Health Trial, the Lipid Research Clinics-Coronary Primary Prevention Trial (LRC-CPPT), the St. Thomas Atherosclerosis Regression Study, the Stanford Coronary Risk Intervention Project (SCRIP), and other dietary intervention trials, WHI involves a series of group intervention sessions to achieve the desired behavior changes (16–18,165,167,168). Such studies combine the cognitive knowledge and behavioral skills needed to achieve dietary change. Problem-solving in a group setting exposes individuals to both the questions and answers others have experienced with various degrees of success. In the WHT, women reduced total fat intake from a range of 36% to 38% of total calories to 21% to 24%, with declines of 15 to 22 mg/dL in total cholesterol after six months of intervention (162,164).

In clinical settings, achieving dietary adherence has not always proven successful, but some studies have yielded favorable results, especially when strategies involving the tailored intervention approach have been applied (169). Despite a lack of formal nutrition training, physicians and nurses were effective in increasing patient knowledge and motivation for changing dietary fat intake in less time than registered dietitians, but compliance and long-term lipid control was enhanced by more frequent dietary consultations (170). Similar strategies have been successfully applied to physician-based counseling for increasing physical activity among previously sedentary patients using the stage of change assessment and adding booster phone calls from health educators (171). Because of their comprehensive nutrition knowledge and clinical counseling skills, registered dietitians can be effective in training physicians, nurses, or other health professionals to initiate routine dietary intervention efforts. Referral to dietitians could thus be reserved for patients with competing nutrient needs, adherence problems, and/or those who require more practical dietary guidance.

In a study of over 558 adults recruited from family practices who were assessed for stage of change and dietary intake, those who received tailored computer-generated intervention messages with feedback regarding current diet assessment reported significantly reduced fat intake compared to the control group (172). The authors further reported that the stage of change framework could be applied to developing the most appropriate intervention messages and these were ultimately associated with decreased fat intake.

Conversely, in a randomized study among over 40 physician practices testing the feasibility of the NCEP guidelines, the overall impact of nutrition intervention delivered via the physician practices was less than clinically desired (147). The authors concluded that the delivery system for nutrition therapy was inadequate and more aggressive approaches utilizing behavioral techniques were recommended. In general, there is a relative paucity of diet-related behavioral data reporting successful approaches to changing eating patterns in adults. Didactic, cognitive intervention is the norm in many clinical practices, but this may be ineffective in most patients.

PREVENTION STRATEGIES FOR CHILDREN

In 1990, the National Cholesterol Education Program's Expert Panel on Blood Cholesterol Levels in Children and Adolescents established guidelines for screening, prevention, and treatment of high blood cholesterol in children (173). On the basis of existing evidence from laboratory, clinical, pathological, and epidemiological studies demonstrating that atherosclerosis begins in childhood, dietary intervention strategies were recommended (173). Growing evidence has provided further rationale for encouraging a lipid-lowering diet in children over the age of two (174).

The NCEP recommended a two-pronged strategy that combines a population approach for all American children over the age of two and an individualized approach for those children and adolescents who are at the greatest risk for developing high blood cholesterol and subsequent cardiovascular disease. One example of

an applied population-based dietary intervention focused on making changes in the school lunch program to better meet the criteria for heart-healthy eating. The Child and Adolescent Trial on Cardiovascular Health (CATCH) was tested in 96 schools (175–177). Initial analyses demonstrated reduced intakes of total fat, saturated fat, cholesterol, and sodium in these third-grade students with normal lipid levels (178). Differences in cholesterol reduction between groups were not statistically significant, but dose response of family intervention was favorable toward acquisition of positive knowledge and attitudes toward health habit changes (179). CATCH also demonstrated that by modifying even one meal, overall adherence to the Step I Diet would be achieved in these free-living children. CATCH further demonstrated that cooperation between school food service workers, educators, and parents can lay the foundation for future reduced fat intake (178).

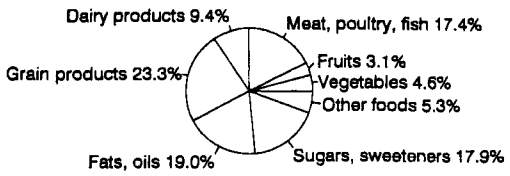
Other school-based studies have involved campaigns to switch from whole milk to low-fat or skim milk in order to reduce intake of a major source of saturated fat and cholesterol in children's diets. Such a change requires relatively little nutrition intervention if the concept is marketed in a fun and entertaining way, but can significantly reduce total and saturated fat intake without compromising overall nutrient intake in this age group. One study involved a live, costumed character named Low-Fat Lucy the cow, who promoted drinking skim milk among the school children (180). Such simple but targeted efforts can achieve overall adherence to the total fat and saturated fat objectives.

When the individualized approach is required for high-risk children, once again, therapy begins with the Step I Diet. If adherence to this diet for at least three months does not achieve adequate lowering of blood cholesterol, the Step II Diet is prescribed. Saturated fat intake is reduced to less than 7% of total calories and cholesterol intake to less than 200 mg/day. Are such diets safe and effective in growing children?

The Dietary Intervention Study in Children (DISC) randomly assigned 663 preadolescent children with elevated LDL-C to either the intervention or usual care group. After three years of intervention with a diet that was 28% of total calories from total fat and less than 8% of calories from saturated fat, a diet similar to the NCEP Step II Diet, LDL cholesterol levels decreased 3.3 mg/dL ($p = 0.02$) more than children in the usual care group, without compromising growth and development (181). Dietitians, behaviorists, and health educators provided intensive dietary intervention to both participants and their parents in the intervention group. Dietary adherence and nutrient adequacy were carefully monitored to prevent adverse affects. Concerns about inappropriate implementation of the low-fat diet and inadequate nutrient intake among children are potentially more warranted in medical settings where dietitian input and follow-up are lacking.

Another example of the individualized approach is the Children's Health Project. This study was clinically-based, involving preadolescent children aged 4–10 years with elevated baseline LDL cholesterol. Children randomly assigned to a Parent–Child Auto-Tutorial (PCAT) method of intervention received nutrition education via a tape-recorded storybook and accompanying workbook. These children also successfully reduced LDL cholesterol levels without compromising growth (182,183).

Such studies demonstrate the efficacy and safety of the Step I or Step II Diets in growing children, but caution must be used in applying these strategies clinically. Key factors include trained, qualified dietitians and health educators who monitored both adherence and nutrient adequacy (184). Well-meaning but untrained parents may unintentionally deny their children the energy, protein, or other essential nutrients they need for growth due to overly-zealous emphasis on fat restriction or other misleading messages from the popular press or elsewhere. Excessive use of abundantly available fat-modified or fat-free products without including all food groups can lead to nutrient deficiencies and/or obesity if calories and serving sizes are not addressed. Parents need help translating dietary information and recommendations into behaviors they can use routinely, safely, and effectively at home.



Food energy

NOTE: The "other foods" category includes legumes, nuts, and soy (2.8%); eggs (1.3%), and miscellaneous foods (1.2%).

Data from U.S. Department of Agriculture. Nutrient Content of the U.S. Food Supply, 1990 (Gerrior SA, Zizza C. 1994. Nutrient Content of the U.S. Food Supply, 1909-1990. Home Economics Research Report No.52. U.S. Department of Agriculture, Washington, DC)[186]

FIGURE 2: Food energy.

POPULATION-WIDE DIETARY ADHERENCE: ROOM FOR IMPROVEMENT

If the population met the Step I dietary guidelines, presumably the estimated impact on total cholesterol lowering would be between 3–10%. Using the extrapolation cited earlier (i.e. for every 1% lipid reduction, there is 2–3% reduction in coronary mortality), this could mean between 1.6 to 5.2 million lives saved from cardiovascular mortality alone. Because the Step I Diet has similarly been recommended for prevention of cancer and other chronic diseases, untold additional benefits could be achieved in reduced overall morbidity and mortality (185).

Figure 2 illustrates the sources of energy intake by food groups (186). In 1990, animal products contributed at least 28% of total calories. Vegetables, fruits and legumes, nuts, and soy together contributed approximately 10.5% of total calories. Sugars and sweeteners contributed almost twice that amount. Fats and oils provided 19% of calories, second only to grains in order of magnitude. Despite encouraging reductions in total and saturated fat intake, the population's dietary pattern is high in fat, sugar, and sodium-rich foods. Table 3 further illustrates that less than a fourth of our population consumes the recommended five or more servings per day of fruits and vegetables with greater intake occurring among women than men. Older people consume more of these foods than younger people. Despite the growing consumer awareness about nutrition and health, only a small proportion of the population appears to be applying the principles to their dietary patterns.

Part of this problem may be due to a perceived lack of credibility of nutrition advice. The print and broadcast media and food labels were identified as the most often cited sources of nutrition information in 1987–1988 (Figure 3) (187). Doctors, nurses, relatives, and friends were cited next often, despite the limited or absent nutrition education required in most American medical schools or any other schools—a fact that goes unrecognized by most consumers (188).

TABLE 3
Percent Distribution of Daily Servings of Fruits and Vegetables Consumed by People 18 Years of Age and Older, by Age and Sex, 1991

Demographic Group	N[1]	0–<2.5 Servings	2.5–<5 Servings	≥5 Servings
		← % Consuming (SE) →		
Men	1,145	37.0 (1.7)	45.1 (1.8)	17.9 (1.3)
18–39 years	715	40.6 (2.2)	41.1 (2.2)	18.2 (1.7)
40–59 years	281	34.4 (3.3)	49.2 (3.5)	16.4 (2.5)
60+ years	144	29.3 (4.4)	51.4 (4.7)	19.3 (3.7)
Women	1,666	24.6 (1.3)	48.3 (1.5)	27.1 (1.3)
18–39 years	878	32.9 (2.0)	44.4 (2.1)	22.7 (1.7)
40–59 years	453	22.4 (2.3)	49.4 (2.8)	28.2 (2.5)
60+ years	303	11.8 (2.2)	54.3 (3.4)	33.8 (3.2)

[1] *N*s by age group do not equal total *N* by gender because of missing data.
NOTE: See Table Notes in Appendix V Section B.
SOURCE: HHS, Baseline Survey of the 5 A Day for Better Health Program, 1991.

Taken collectively, these data illustrate a general lack of science-based nutrition knowledge among most health care providers, resulting in absent or ineffective dietary counseling among patients. Improved delivery of nutrition education and better utilization of existing nutrient information could have highly favorable public health impact. Since physicians and nurses are on the front-line of patient care and preventive efforts, their endorsement of recommended dietary principles and referral for complex dietary intervention are pivotal to achieving improved adherence.

PRACTICAL STEPS TOWARD ACHIEVING DIETARY ADHERENCE

In a review of thirty studies reporting changes in fat intake, Barnard et al. (189) summarized the factors that were commonly associated with increased adherence.

Factors Related to Improved Dietary Compliance

Stricter Limits on Fat Intake: The lower the limit, the closer the mean level of adherence.

Frequent Monitoring: At least monthly monitoring is recommended.

Vegetarian Diets: Use of a vegetarian diet more often achieved recommended fat intake below 30% kcal than non-vegetarian approaches.

Initial Residential Component: Provides intensive training, monitoring, provision of food, and group support on the short-term, resulting in better adherence on self-selected diets long-term.

Family Involvement: Family involvement yields better adherence.

Group Support: Group support is not mandatory, but can be helpful.

Providing Food: Entire meals are not required, but some provision of acceptable products is important.

Symptomatic Patients: These patients are apparently more motivated with healthy, high-risk patients similarly motivated.

Care must be taken in interpreting these findings, since this was not a formal meta-analysis and rigorous statistical principles were not applied. Also, these findings were derived from research studies, and incentives to participate may have been far different than those observed in the clinical or community settings. Despite these limitations, these strategies offer potentially relevant strategies for effectively implementing dietary changes.

Relevant sources of total and saturated fat and cholesterol should be identified before attempting to prescribe a new dietary pattern for any patient. Both the MRFIT and WHT studies reported that meats, fats and oils, dairy, and baked goods contributed more total and saturated fat to the diets of adults than any other food groups (165,168). Fortunately, there are now many acceptable alternatives to these high-fat foods. Substitution of lower-fat or fat-free versions for the high-fat foods (i.e. skim milk for whole milk, fat-free salad dressing for full-fat salad dressing) and adding more servings of fruits, vegetables, and grains to compensate for fewer servings of high-fat meats, dairy, and baked goods are essential components. The food industry has aggressively responded to the request for lower-fat or no-fat products. The greater challenge is achieving the desired shift to greater intake of complex carbohydrate foods.

Changing Dietary Behavior, One Patient at a Time

On the basis of these behavioral principles and accumulated knowledge about diet and risk reduction, the following suggestions can help promote successful dietary adherence.

Start with Dietary Assessment Then Individualize Dietary Intervention: Assessing baseline intake is the only way to identify the foods that are contributing the most saturated fat and cholesterol to a patient's diet. In adults, this is often meats, fats, and sweets. In children, it is often whole milk dairy products. In children who consume the recommended four servings of dairy foods per day, it may be possible to achieve adherence to the Step I Diet (≤10% SFA) simply by switching to skim milk and low-fat dairy products at school and at home.

Provide Clear, Identifiable Goals for Each Individual: For example, the current food labels make it possible to establish a fixed "Fat Gram Goal" rather than the less precise recommendations to eat less than 30% of calories from total fat. This provides each person with an objective target that can be self-monitored. Similarly, establishing the goal number of servings of fruits, vegetables, and grains can further assist the individual to achieve increased fiber, folate, and antioxidant intake.

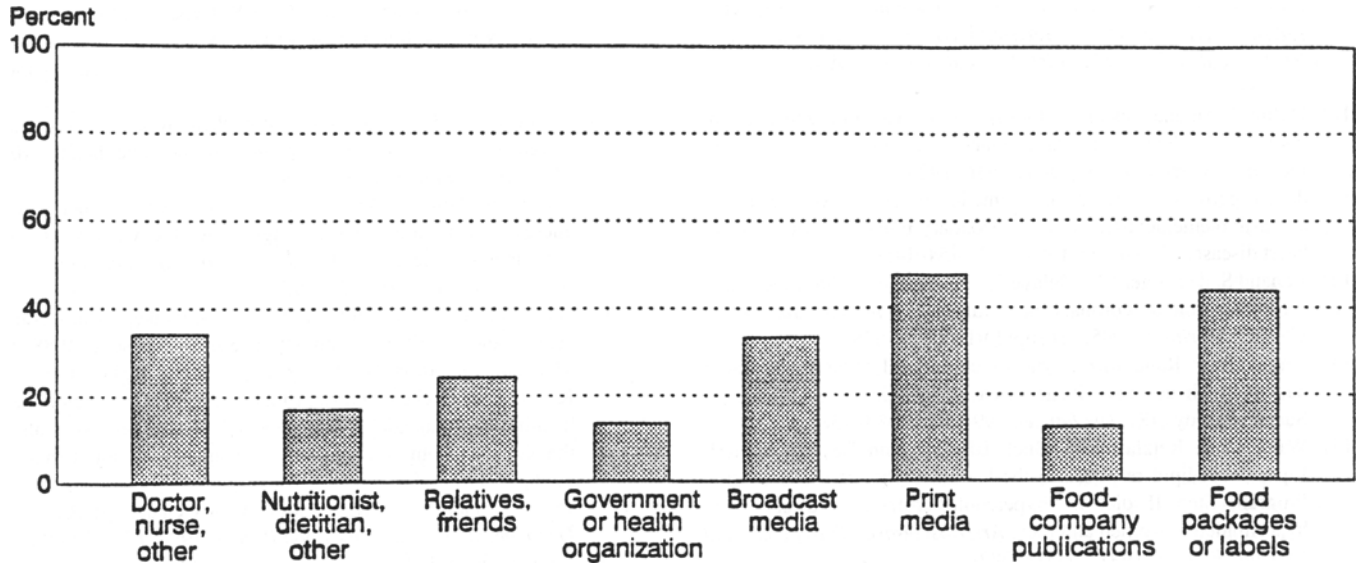
Assess Current Stage of Change: Assess current stage of change and determine the individual's level of confidence in achieving a self-determined adherence goal. Reassess the status at each subsequent visit.

Encourage Self-Monitoring: Encourage self-monitoring through use of food records and/or other simplified fat/fiber goal-counting records. Likewise encourage non-food self-rewards when goals are met.

Promote the Benefit of Adopting Other Health-Oriented Behaviors: Other health-oriented behaviors include exercise, relaxation techniques, stress reduction, etc.

Prevent Relapse: Prevent relapse through ongoing follow-up, reassessment, and establishing new goals as needed.

Patients who are referred to registered dietitians or other trained nutrition counselors may require relatively few follow-up sessions. Comprehensive feedback on dietary adherence can be



Percentage of households that reported using specific sources of nutrition information, 1987-88

NOTE: Data are from table A.F8-21 in appendix VA. "Other" in column 1 means "other health professionals"; in column 2, it means "home economist or extension agent."

SOURCE: USDA, NFCS, 1987-88.

FIGURE 3: Percentage of households that reported using specific sources of nutrition information, 1987–1988.

provided to the referring physician for consideration in determining future treatment plans.

FUTURE DIRECTIONS

Because diet has both biological and behavioral significance, individualized dietary therapy may call for alternate approaches to the population-based Step I–II Diet recommendations in certain cases. For example, while the normal weight patient with elevated LDL cholesterol and normal triglycerides may respond favorably (physiologically) to low saturated fat and high complex carbohydrate intake, the obese patient with elevated triglycerides and low levels of HDL-C may benefit from a more specified substitution of monounsaturated and omega-three fatty acids for saturated fat intake. While generally recommended for everyone, exercise may also be particularly useful in the obese patient and should be aggressively pursued. As research identifies more dietary and non-dietary factors that induce effective lipid modifications, there is improved opportunity to match a specific diet to specific conditions. Genetic influences that render a patient non-diet responsive should be factored into the appropriate treatment, either pharmacologic or dietary. Hormonal differences between men and women may also influence lipid–lipoprotein responses, but these have yet to be delineated. Psychosocial correlates identified as predisposing factors (i.e. beliefs, perceived benefits, and motivation) were highly correlated with healthful dietary intake in cross-sectional baseline data from 16,287 respondents in a worksite trial intervention (190). These data offer further clues to more targeted intervention efforts.

Convincing health professionals and patients that diet works is a prerequisite to achieving the recommended dietary patterns. The evidence reviewed here and elsewhere contributes to that premise, but without motivation to adhere, diet is ineffective. Motivating adult patients to change remains a challenge requiring the combined efforts of nutritional, behavioral, and medical expertise. Finally, primary prevention efforts through establishment of healthy eating patterns beginning in childhood offer the ideal public health strategy to foster lifelong weight control and reduced overall cardiovascular risk.

REFERENCES

(1) Keys A: *Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease.* Cambridge, MA: Harvard University Press, 1980.

(2) Levine G, Keaney J, Vita J: Cholesterol reduction in cardiovascular disease. *New England Journal of Medicine.* 1995, *332*(8):512–521.

(3) Gould AL, Rossouw JE, Santanello NC, Heyse JF, Furberg CD: Cholesterol reduction yields clinical benefit: A new look at old data. *Circulation.* 1995, *91*(8):2274–2282.

(4) Martin MJ, Hulley SB, Browner WS, Kuller LH, Wentworth D: Serum cholesterol, blood pressure, and mortality: Implications from a cohort of 361,662 men. *The Lancet.* 1986, *2*:933–939.

(5) Lipid Research Clinics Program: The Lipid Research Clinics Coronary Primary Prevention Trial results. I. Reduction in incidence of coronary heart disease. *Journal of the American Medical Association.* 1984, *251*:351–364.

(6) Study Group E.A.S.: Strategies for the prevention of coronary artery disease: A policy statement of the European Atherosclerosis Society. *European Heart Journal.* 1987, *8*:77–88.

(7) Manninen V, Elo MO, Frick MH, et al: Lipid alterations and decline in the incidence of coronary heart disease in the Helsinki Heart Study. *Journal of the American Medical Association.* 1988, *260*:641–651.

(8) Roussouw JE: The effects of lowering serum cholesterol on coronary heart disease risk. *Medical Clinics of North America.* 1994, *78*:181–195.

(9) Law MR, Wald NJ, Thompson SG: By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischemic heart disease? *British Medical Journal.* 1994, *308*(6925): 367–372.
(10) Holme I: An analysis of randomized trials evaluating the effect of cholesterol reduction on total mortality and coronary heart disease incidence. *Circulation.* 1990, *82:*1916–1924.
(11) de Lorgeril M, Renaud S, Mamelle N, et al: Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. *The Lancet.* 1994, *343:*1454–1459.
(12) Renaud S, de Lorgeril M, Delaye J, et al: Cretan Mediterranean diet for prevention of coronary heart disease. *American Journal of Clinical Nutrition.* 1995, *61*(Suppl.):1360S–1367S.
(13) Anonymous: Randomized trial of cholesterol lowering in 4,444 patients with coronary heart disease: Scandinavian Simvastatin Survival Study (4S). *The Lancet.* 1994, *344:*1383–1389.
(14) Walden CE, Retzlaff BM, Buck BL, McCann BS, Knopp RH: Lipoprotein lipid response to the National Cholesterol Education Program Step II diet by hypercholesterolemic and combined hyperlipidemic women and men. *Arteriosclerosis, Thrombosis and Vascular Biology.* 1997, *17:*375–382.
(15) Blankenhorn DH, Johnson RL, Mack WJ, El Zein HA, Vailas LI: The influence of diet on the appearance of new lesions in the human coronary arteries. *Journal of the American Medical Association.* 1990, *263:*1646–1652.
(16) Schuler G, Hambrecht R, Schlierf G, et al: Regular physical exercise and low-fat diet. Effects of progression on coronary artery disease. *Circulation.* 1992, *86:*1–11.
(17) Ornish D, Brown SE, Scherwitz LW, et al: Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *The Lancet.* 1990, *336:*129–133.
(18) Watts GF, Lewis B, Brunt JNH, et al: Effects on coronary artery disease of lipid lowering diet, or diet plus cholestyramine, in the St. Thomas' Atherosclerosis Regression Study (STARS). *The Lancet.* 1992, *339:*563–569.
(19) Brown BG, Zhao X-Q, Sacco DE, Albers JJ: Lipid lowering and plaque regression. New insights into prevention of plaque disruption and clinical events in coronary disease. *Circulation.* 1993, *87:*1781–1791.
(20) Stone NJ: Diet, blood cholesterol levels, and coronary heart disease. *Coronary Artery Disease: Reviews in Depth.* 1993, *4:*871–881.
(21) Ascherio A, Willett W: New directions in dietary studies of coronary heart disease. *Journal of Nutrition.* 1995, *125:*647S–655S.
(22) National Cholesterol Education Program: *Report of the Expert Panel on Population Strategies for Blood Cholesterol Reduction,* DHHS Publication No. (NIH) 90-30-46. Bethesda, MD: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Heart, Lung and Blood Institute, November 1990.
(23) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults: Summary of the Second Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *Journal of the American Medical Association.* 1993, *269:*3015–3023.
(24) LaRosa JC, Hunninghake D, Bush D, et al: The cholesterol facts: A summary of the evidence relating dietary fats, serum cholesterol, and coronary heart disease: A joint statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Circulation.* 1990, *81:*1721–1733.
(25) Buefel RR: Assessment of the U.S. diet in national nutrition surveys: National collaborative efforts and NHANES. *American Journal of Clinical Nutrition.* 1994, *59*(Suppl.): 1645–1675.
(26) *Nationwide Food Consumption Survey, Continuing Survey of Food Intakes by Individuals: Women 19–50 Years and Children 1–5 Years, 4 Days.* Washington, DC: U.S. Department of Agriculture, Human Nutrition Information Service,1996.
(27) Schoeller DA: How accurate is self-reported dietary energy intake? *Nutrition Reviews.* 1990, *48:*373–379.
(28) Livingstone ME, McKenna PG, Whitehead RG, et al: Accuracy of weighed dietary records in studies of diet and health. *British Medical Journal.* 1990, *300:*708–712.
(29) Black AE, Prentice AM, Goldberg G, et al: Measurements of total energy expenditure provide insights into the validity of dietary measures of energy intake. *Journal of the American Dietetic Association.* 1993, *93:*572–579.
(30) Daily dietary fat and total food energy intakes: Third National Health and Nutrition Examination Survey Phase I, 1988–1991. *Morbidity and Mortality Weekly Report.* 1994, *43:*116–125.
(31) Krauss RM, Deckelbaum RJ, Ernst N, et al: Dietary guidelines for healthy American adults: A statement for health professionals from the Nutrition Committee, American Heart Association. *Circulation.* 1996, *94:*1795–1800.
(32) Jones-Putman J: American eating habits changing: Part 2. *U.S. Department of Agriculture Food Review.* 1994, *17*(2):36–48.
(33) Jones-Putman J, Duewer L: U.S. per capita food consumption: Record high meat and sugars in 1994. *U.S. Department of Agriculture Food Review.* 1995, *18*(2):2–11.
(34) Sempos C, Cleeman J, Carroll M, et al: Prevalence of high blood cholesterol among U.S. adults. *Journal of the American Medical Association.* 1993, *269:*3009–3014.
(35) Schucker B, Wittes JT, Santanello NC, et al: Change in cholesterol awareness and action: Results from national physician and public surveys. *Archives of Internal Medicine.* 1991, *151*(4):666–673.
(36) Keys A: Serum cholesterol response to dietary cholesterol. *American Journal of Clinical Nutrition.* 1984, *40:*351–359.
(37) Keys A, Parlin RW: Serum cholesterol response to changes in dietary lipids. *American Journal of Clinical Nutrition.* 1966, *19:*175–181.
(38) Hegsted DM, Nicolosi RJ: Individual variation in serum cholesterol levels. *Proceedings of the National Academy of Sciences of the United States of America.* 1987, *84:*6259–6261.
(39) Hegsted DM, Ausman LM, Johnson JA, Dallul GE: Dietary fat and serum lipids: An evaluation of the experimental data. *American Journal of Clinical Nutrition.* 1993, *57:*875–883.
(40) Mensink RP, Katan MB: Effect of dietary fatty acids on serum lipids and lipoproteins: A meta-analysis of 27 trials. *Arteriosclerosis and Thrombosis.* 1992, *12:*911–919.
(41) Yu S, Derr J, Etherton TD, Kris-Etherton P: Plasma cholesterol predictive equations demonstrate that stearic acid is neutral and monounsaturated fatty acids are hypocholesterolemic. *American Journal of Clinical Nutrition.* 1995, *61:*1129–1139.
(42) Kris-Etherton P, Yu S: Individual fatty acid effects on plasma lipids and lipoproteins: Human studies. *American Journal of Clinical Nutrition* (in press, 1996).
(43) McDougall J, Litzau K, Haver E, Saunders V, Spiller G: Rapid reduction of serum cholesterol and blood pressure by a twelve-day, very low-fat, strictly vegetarian diet. *Journal of the American College of Nutrition.* 1995, *14*(5):491–496.
(44) Phillips RL, Lemon RR, Beeson L, Kuzma JW: Coronary heart disease mortality among Seventh-Day Adventists with differing dietary habits: A preliminary report. *American Journal of Clinical Nutrition.* 1978, *31:*S191–S198.
(45) Fraser GE, Sabate J, Beeson WL, Strahan TM: A possible protective effect of nut consumption on risk of coronary heart disease: The Adventist Health Study. *Archives of Internal Medicine.* 1992, *152:*1416–1424.
(46) Fisher M, Levine PH, Weiner B, et al: The effect of vegetarian diets on plasma lipid and platelet levels. *Archives of Internal Medicine.* 1986, *146:*1193–1197.
(47) Singh RB, Rastogi SS, Verma R, et al: Randomized controlled trial of a cardioprotective diet in patients with recent acute myocardial

infarction: Results of one-year follow-up. *British Medical Journal.* 1992, *304:*1015–1019.

(48) Denke M: Review of human studies evaluating individual dietary responsiveness in patients with hypercholesterolemia. *American Journal of Clinical Nutrition.* 1995, *62:*471S–477S.

(49) Kris-Etherton PM, Derr J, Mustad V, Seligson F, Pearson T: Effects of a milk chocolate bar per day substituted for a high carbohydrate snack in young men on an NCEP/AHA Step I Diet. *American Journal of Clinical Nutrition.* 1994, *60*(Suppl.):1037S–1042S.

(50) Scott L, Kimball K, Wittels E, et al: Effects of a lean beef diet and of a chicken and fish diet on lipoprotein profiles. *Nutrition Metabalism and Cardiovascular Disease.* 1991, *1:*25–30.

(51) Katan MB, Zock PL, Mensink RP: Effects of fats and fatty acids on blood lipids in humans: An overview. *American Journal of Clinical Nutrition.* 1994, *60*(Suppl.): 1017S–1022S.

(52) Bonanome A, Grundy S: Effect of dietary stearic acid on plasma cholesterol and lipid levels. *New England Journal of Medicine.* 1988, *318:*1244–1248.

(53) Stamler J, Shekelle R: Dietary cholesterol and human coronary heart disease. The epidemiologic evidence. *Archives of Pathology and Laboratory Medicine.* 1988, *112:*1032–1040.

(54) Shekelle RB, Shyrock AM, Paul O, et al: Diet, serum cholesterol, and death from coronary heart disease. The Western Electric Study. *New England Journal of Medicine.* 1981, *394:*65–70.

(55) Kushi LH, Lew RA, Stare FJ, et al: Diet and 20-year mortality from coronary heart disease: The Ireland–Boston diet–heart study. *New England Journal of Medicine.* 1985, *312:*811–818.

(56) Kromhout D, de Lezenne Coulander C: Diet, prevalence, and 10-year mortality from coronary heart disease in 871 middle-aged men. The Zutphen Study. *American Journal of Epidemiology.* 1984, *119*(5):733–741.

(57) McGee DL, Reed DM, Yano K, et al: Ten-year incidence of coronary heart disease in the Honolulu Heart Program. Relationship to nutrient intake. *American Journal of Epidemiology.* 1984, *119*(5):667–676.

(58) Garg A, Bonanome A, Grundy SM, Zhang ZJ, Unger RH: Comparison of a high-carbohydrate diet with a high-monounsaturated fat diet in patients with non-insulin-dependent diabetes mellitus. *New England Journal of Medicine.* 1988, *319:*829–834.

(59) Mensink RP, Katan MB: Effect of a diet enriched with monounsaturated fat or polyunsaturated fatty acids on levels of low-density and high-density lipoprotein cholesterol in healthy women and men. *New England Journal of Medicine.* 1989, *321:*436–441.

(60) Berey EM, Eisenberg S, Friedlander Y, et al: Effects of diets rich in monounsaturated fatty acids on plasma lipoproteins: The Jerusalem Nutrition Study. II. Monounsaturated fatty acids vs carbohydrates. *American Journal of Clinical Nutrition.* 1992, *56:*394–403.

(61) Ginsberg HN, Barr SL, Gilbert A, et al: Reduction of plasma cholesterol levels in normal men on an American Heart Association Step I Diet or a Step I Diet with added monounsaturated fat. *New England Journal of Medicine.* 1990, *322:*574–579.

(62) Katan MB, Zock PL, Mensink RP: Dietary oils, serum lipoproteins, and coronary heart disease. *American Journal of Clinical Nutrition.* 1995, *61*(Suppl.):1368S–1373S.

(63) Mensink RP, Katan MB: Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. *New England Journal of Medicine.* 1990, *323:*439–445.

(64) Willett WC, Stampfer MJ, Manson JE, et al: Intake of trans fatty acids and risk of coronary heart disease among women. *The Lancet.* 1993, *341:*581–585.

(65) Wahrburg U, Martin H, Sandkamp M, Schulte H, Assmann G: Comparative effects of a recommended lipid-lowering diet vs a diet rich in monounsaturated fatty acids on serum lipid profiles in healthy young adults. *American Journal of Clinical Nutrition.* 1992, *56:*678–683.

(66) Renaud S, Logeril M, Delaye J, et al: Cretan Mediterranean diet for prevention of coronary heart disease. *American Journal of Clinical Nutrition.* 1995, *61*(Suppl.):1360S–1367S.

(67) Grundy SM: Comparison of monounsaturated fatty acids and carbohydrates for lowering plasma cholesterol. *New England Journal of Medicine.* 1986, *314:*745–748.

(68) Dengel J, Katzel L, Goldberg A: Effect of an American Heart Association diet, with or without weight loss, on lipids in obese middle-aged and older men. *American Journal of Clinical Nutrition.* 1995, *62:*715–721.

(69) Dattilo AM, Kris-Etherton PM: Effects of weight reduction on blood lipids and lipoproteins: A meta-analysis. *American Journal of Clinical Nutrition.* 1992, *56:*320–328.

(70) Wood PD, Stefanick ML, Williams PT, Haskell WL: The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *New England Journal of Medicine.* 1991, *325:*461–466.

(71) Wood PD, Stefanick ML, Dreon D, et al: Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *New England Journal of Medicine.* 1988, *319:*1173–1179.

(72) Sonnichsen AC, Richter WO, Schwandt P: Benefit from hypoenergetic diet in obese men depends on the extent of weight-loss regarding cholesterol and on a simultaneous change in body fat distribution regarding insulin sensitivity and glucose tolerance. *Metabolism.* 1992, *41:*1035–1039.

(73) Katan MB: Fish and heart disease. *New England Journal of Medicine.* 1995, *332:*1024–1025.

(74) Harris WS, Rothrock DW, Fanning A, et al: Fish oils in hypertriglyceridemia: A dose-response study. *American Journal of Clinical Nutrition.* 1990, *51:*399–406.

(75) Ascherio A, Rimm EB, Stampfer MJ, Giovannucci EL, Willett WC: Dietary intake of marine n-3 fatty acids, fish intake, and the risk of coronary heart disease among men. *New England Journal of Medicine.* 1995, *332:*977–982.

(76) Guallar E, Hennekens CH, Sacks RM, Willett WC, Stampfer MJ: A prospective study of plasma fish oil levels and incidence of myocardial infarction in U.S. male physicians. *Journal of the American College of Cardiology.* 1995, *25:*387–394.

(77) Burr ML, Fehily AM, Gilbert JF, et al: Effects of changes in fat, fish, and fiber intakes on death and myocardial reinfarction: Diet and reinfarction trial (DART). *The Lancet.* 1989, *2:*757–761.

(78) Lichtenstein A, Ausman L, Carrasco W, et al: Hydrogenation impairs the hypolipidemic effect of corn oil in humans. *Arteriosclerosis and Thrombosis.* 1993, *13:*154–161.

(79) Emken EA: Physiochemical properties, intake, and metabolism. In Trans fatty acids and coronary heart disease risk. *American Journal of Clinical Nutrition.* 1995, *62*(3):659S–669S.

(80) Denke MA: Serum lipid concentrations in humans. In Trans fatty acids and coronary heart disease risk. *American Journal of Clinical Nutrition.* 1995, *62*(3):693S–700S.

(81) Kris-Etherton PM, Emken EA, Allison DB, et al: Trans fatty acids and coronary heart disease risk. *American Journal of Clinical Nutrition.* 1995, *62*(Suppl.):665S–708S.

(82) Jonnalagadda SS, Mustad VA, Yu S, Etherton TD, Kris-Etherton PM: Effects of individual fatty acids on chronic diseases. *Nutrition Today.* 1996, *31*(3):90–106.

(83) Wynder EL, Stellman SD, Zang EA: High fiber intake: Indicator of a healthy lifestyle. *Journal of the American Medical Association.* 1996, *275:*486–487.

(84) Davidson MH, Dugan LD, Burns JH, et al: The hypocholesterolemic effects of B glucan in oatmeal and oat bran. A dose-controlled study. *Journal of the American Medical Association.* 1991, *265:*1833–1839.

(85) Anderson JW, Zettwoch N, Feldman T, et al: Cholesterol-lowering effects of psyllium hydrophilic mucilloid for hypercholesterolemic men. *Archives of Internal Medicine.* 1988, *148:*292–296.

(86) Jenkins DJA, Wolever TMS, Rao AV, et al: Effect of blood lipids of very high intakes of fiber in diets low in saturated fat and cholesterol. *New England Journal of Medicine.* 1993, *329:*21–26.

(87) Jenkins DJA, Hegele RA, Jenkins AL, et al: The apolipoprotein E gene and the serum low-density lipoprotein cholesterol response to dietary fiber. *Metabolism.* 1993, *42:*585–593.

(88) Ripsin CM, Keenan JM, Jacobs DR, et al: Oat products and lipid lowering: A meta-analysis. *Journal of the American Medical Association.* 1992, *267:*3317–3325.

(89) Van Horn L, Moag-Stahlberg A, Liu K, et al: Effects on serum lipids of adding instant oats to usual American diets. *American Journal of Public Health.* 1991, *81:*183–188.

(90) Messina M, Erdman JW (eds): First International Symposium on the role of soy in preventing and treating chronic disease. *Journal of Nutrition.* 1995, *125*(Suppl.):567S–808S.

(91) Anderson JW, Johnstone BM, Cook-Newell ME: Meta-analysis of the effects of soy protein intake on serum lipids. *New England Journal of Medicine.* 1995, *333:*276–282.

(92) Stamler J, Rose G, Elliot P, et al: Findings of the International Cooperative Intersalt Study. *Hypertension.* 1991, *17*(Suppl. I):I-9–I-15.

(93) Elliott P, Stamler J, Dyer AR, et al for the Intersalt Cooperative Research Group: INTERSALT revisited: Further analyses of 24 hour sodium excretion and blood pressure within and across populations. *British Medical Journal.* 1996, *312:*1249–1253.

(94) Elliott P, Dyer A, Stamler R on behalf of the INTERSALT Cooperative Research Group: The INTERSALT Study: Results for 24-hour sodium and potassium by age and sex. *Journal of Human Hypertension.* 1989, *3:*232–330.

(95) Cutler JA, Follmann D, Elliott P, Suk I: An overview of randomized trials of sodium reduction and blood pressure. *Hypertension.* 1991, *17*(Suppl. I):I-27–I-33.

(96) The Trials of Hypertension Prevention Collaborative Research Group: The effects of nonpharmacologic intervention in blood pressure of persons with high normal levels: Results of the trials of hypertension. Prevention Phase I. *Journal of the American Medical Association.* 1992, *267:*1213–1220.

(97) McCarron D: Calcium metabolism in hypertension. *Keio Journal of Medicine.* 1995, *44*(4):105–114.

(98) Princen HM, Van Poppel G, Vogelezang C, Buytenhek R, Kok FJ: Supplementation with vitamin E but not beta-carotene in vivo protects low density lipoprotein from lipid peroxidation in vitro: Effect of cigarette smoking. *Arteriosclerosis and Thrombosis.* 1992, *12:*554–562.

(99) Stamler MJ, Hennekens CH, Manson JE, et al: Vitamin E consumption and the risk of coronary disease in women. *New England Journal of Medicine.* 1993, *328:*1444–1449.

(100) Steinberg D, Parthasarathy S, Carew TE, Khoo JC, Witzum JL: Beyond cholesterol: Modifications of low-density lipoprotein that increase its atherogenicity. *New England Journal of Medicine.* 1989, *320:*915–924.

(101) Hoffman RM, Garewal HS: Antioxidants and the prevention of coronary heart disease. *Archives of Internal Medicine.* 1995, *155:*241–246.

(102) Boushey CJ, Beresford SAA, Omenn GS, Motulsky AG: A quantitative assessment of plasma homocysteine as a risk factor for vascular disease: Probable benefits of increasing folic acid intakes. *Journal of the American Medical Association.* 1995, *274:*1049–1057.

(103) Krauss RM: Genetic influences on lipoprotein response to dietary fat and cholesterol: Overview. *American Journal of Clinical Nutrition.* 1995, *62:*457S.

(104) Dreon DM, Fernstrom HA, Miller B, Krauss RM: Low-density lipoprotein subclass patterns and lipoprotein response to a reduced-fat diet in men. *FASEB Journal.* 1994, *8:*121–126.

(105) Dreon DM: Apolipoprotein E isoform phenotype and LDL subclass response to a reduced-fat diet. *Arteriosclerosis, Thrombosis and Vascular Biology.* 1995, *15:*105–111.

(106) Hubert HB, Feinleib M, McNamara PM, Castelli WP: Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham Heart Study. *Circulation.* 1983, *67*(5):968–977.

(107) Denke MA, Sempos CT, Grundy SM: Excess body weight: An underrecognized contributor to high blood cholesterol levels. *Archives of Internal Medicine.* 1993, *153:*1093–1103.

(108) Medalie JH, Papier CM, Goldbourt U, Herman JB: Major factors in the development of diabetes mellitus in 10,000 men. *Archives of Internal Medicine.* 1975, *135:*811–817.

(109) Tobian L: Hypertension and obesity. *New England Journal of Medicine.* 1978, *298:*46–60.

(110) Health implications of obesity. National Institutes of Health Consensus Development Conference Statement. *Annals of Internal Medicine.* 1985, *103*(Pt. 2):1073–1077.

(111) Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL: Overweight prevalence and trends for children and adolescents: The National Health and Examination Surveys, 1963 to 1991. *Archives of Pediatric and Adolescent Medicine* 1995, *149:*1085–1091.

(112) Becque MD, Katch VL, Rocchini AP, et al: Coronary risk incidence of obese adolescents: Reduction by exercise plus diet intervention. *Pediatrics.* 1988, *81:*605–612.

(113) Franklin F: Nutrition and cardiovascular disease. In Weinser RL, Morgan SL (eds), *Fundamentals of Clinical Nutrition.* St. Louis, MO: Mosby Year Book, 1993.

(114) Faber OK, Christensen K, Kehlet H, Madsbad S, Binder C: Decreased insulin removal contributes to hyperinsulinemia in obesity. *Journal of Clinical Endocrinology and Metabolism.* 1981, *54:*618–621.

(115) Manolio TA, Savaga PJ, Burke GL, et al: Association of fasting insulin with blood pressure and lipids in young adults. The CARDIA Study. *Arteriosclerosis.* 1990, *10:*430–436.

(116) Modan M, Halkin H, Almog S, et al: Hyperinsulinemia: A link between hypertension, obesity, and glucose intolerance. *Journal of Clinical Investigation.* 1985, *75:*809–817.

(117) Pinhas-Hamiel O, Standiford D, Daniels SR, Zeitler P, Dolan LM: Non-insulin dependent diabetes mellitus is increasing among adolescents. *Pediatric Research.* 1995, *37:*71.

(118) Knip M, Nuutinen O: Long-term effects of weight reduction on serum lipids and plasma insulin on obese children. *American Journal of Clinical Nutrition.* 1993, *57:*490–493.

(119) Stokes III J, Kannel WB, Wolf PA, et al: The relative importance of selected risk factors for various manifestations of cardiovascular disease among men and women from 35 to 65 years old: 30 years of follow-up in the Framingham Study. *Circulation.* 1987, *75*(Suppl. V):65–73.

(120) Voors AW, Webber LS, Frerichs RR, et al: Body weight and body mass as determinants of basal blood pressure in children: The Bogalusa Heart Study. *American Journal of Epidemiology.* 1977, *106:*101–115.

(121) Rocchini AP: Adolescent obesity and hypertension. *Pediatric Clinics of North America.* 1993, *60:*81–92.

(122) Rocchini AP, Katch V, Anderson J, et al: Blood pressure in obese adolescents: Effect of weight loss. *Pediatrics.* 1988, *82:*16–23.

(123) Stunkard A, Sorenson T, Hanis C, et al: An adoption of human obesity. *New England Journal of Medicine.* 1986, *314:*193–198.

(124) Garner D, Wooley S: Confronting the failure of behavior and dietary treatments for obesity. *Clinical Psychology Review.* 1991, *11:*729–780.

(125) Wadden TA, Stunkard A, Brownell K: Very low calorie diets: Their efficacy, safety, and future. *Annals of Internal Medicine.* 1983, *99:*675–684.

(126) Wadden TA, Van Italie T, Blackburn G: Responsible and unresponsible use of very low calorie diets in the treatment of obesity. *Journal of the American Medical Association.* 1990, *263:*83–85.

(127) Stunkard A, Foch T, Hrubec Z: A twin study of human obesity. *Journal of the American Medical Association.* 1986, *256:*51–54.

(128) Zhang YR, Procenca R, Maffei M, et al: Positional cloning of the mouse obese gene and its human homologue. *Nature.* 1994, *372:*425–432.

(129) Stunkard AJ: Some perspectives on human obesity: Its causes. *Bulletin of the New York Academy of Medicine.* 1988, *64:*902–923.

(130) Abrahm S, Collins G, Nordsieck M: Relationship of childhood weight status to morbidity in adults. *HSMHA Health Reports.* 1971, *86:*273–284.

(131) Lauer RM, Clarke WR: Childhood risk factors for high adult blood pressure: The Muscatine Study. *Pediatrics.* 1984, *74:*633–641.

(132) Lauer RM, Lee J, Clarke WR: Factors affecting the relationship between childhood and adult cholesterol levels: The Muscatine Study. *Pediatrics.* 1988, *82:*577–582.

(133) Wattigney WA, Harsha DW, Srinivasan SR, et al: Increasing impact of obesity on serum lipids and lipoproteins in young adults. *Archives of Internal Medicine.* 1991, *151:*2017–2022.

(134) National Institutes of Health: Consensus development panel on the health implications of obesity. *Annals of Internal Medicine.* 1985, *103:*1073–1077.

(135) O'Leary KD, Wilson GT: *Behavior Therapy: Application and Outcome.* Englewood Cliffs, NJ: Prentice Hall, 1975.

(136) Brownell KD, Heckerman C, Westlake R: The behavior control: A descriptive analysis of a large scale program. *Journal of Clinical Psychology.* 1979, *35:*864.

(137) Piscano J, Lichter H, Ritter J, Siegal A: An attempt at prevention of obesity in infancy. *Pediatrics.* 1989, *61:*360.

(138) Smicklas-Wright H, D'Augelli AR: Primary prevention for overweight. Preschool Eating Patterns (PEP) program. *Journal of the American Dietetic Association.* 1978, *72:*626.

(139) Epstein L, Wing R, Koeshe R, Valoski A: Long-term effects of family based treatment of childhood obesity. *Journal of Consulting and Clinical Psychology.* 1987, *55:*91–95.

(140) Epstein L, Valoski A, Wing R, McCurley J: Ten-year follow-up of behavioral, family based treatment for obese children. *Journal of the American Medical Association.* 1990, *265:*2514–2523.

(141) Mellin L, Slinkard L, Irwin C: Adolescent obesity intervention, validation of the shapedown program. *Journal of the American Dietetic Association.*1987, *87:*333–338.

(142) Bandura A: Self-efficacy: Toward a unifying theory of behavior change. *Psychological Review.* 1977, *84:*191.

(143) St Jeor ST, Brunner RL, Harrington ME, et al: A classification system to evaluate weight maintainers, gainers, and losers. *Journal of the American Dietetic Association.* 1997, *97:*481–488.

(144) Hertzler A, Vaughan C: The relationship of family structure and interaction to nutrition. *Journal of the American Dietetic Association.* 1979, *74:*23–27.

(145) Glanz K: Nutrition education for risk factor reduction and patient education: A review. *Preventive Medicine.* 1985, *14:*721–752.

(146) Ammerman A, Caggiula A, Elmer P, et al: Putting medical guidelines into practice: The cholesterol model. *American Journal of Preventive Medicine.* 1994, *10*(4):209–216.

(147) Caggiula A, Watson J, Kuller L, et al: Cholesterol-lowering intervention program: Effect of the Step I diet in community office practices. *Archives of Internal Medicine.* 1996, *156:*1205–1213.

(148) Ammerman A, Devells R, Carey T, et al: Physician-based diet counseling for cholesterol reduction: Current practices, determinants, and strategies for improvement. *Preventive Medicine.* 1993, *22:*96–109.

(149) Contento IR, Murphy BM: Psychosocial factors differentiating people who reported making desirable changes in their diets from those who did not. *Journal of Nutrition Education.* 1990, *22:*6–14.

(150) Glanz K, Eriksen MP: Individual and community models for dietary behavior change. *Journal of Nutrition Education.* 1993, *25:80–86.*

(151) Rosenstock IM: The health belief model: Explaining health behavior through expectancies. In Glanz K, Lewis FM, Rimer BK (eds), *Health Behavior and Health Education: Theory, Research, and Practice.* San Francisco, CA: Jossey-Bass, 1990, 39–62.

(152) Glanz K: Nutritional intervention: A behavioral and educational perspective. In Ockene I, Ockene J (eds), *Prevention of Coronary Heart Disease.* Boston, MA: Little Brown and Co., 1992, 231–265.

(153) Bandura A: *Social Foundations of Thought and Action: A Social Cognitive Theory.* Englewood Cliffs, NJ: Prentice-Hall, Inc., 1986.

(154) Prochaska JO, DiClemente CC: Transtheoretical therapy: Toward a more integrative model of change. *Psychotherapy: Theory, Research and Practice.* 1982, *19:*276–288.

(155) Glanz K, Patterson R, Kristal A, et al: Stages of change in adopting healthy diets: Fat, fiber, and correlations of nutrient intake. *Health Education Quarterly.* 1994, *21*(4):499–519.

(156) Prochaska JO, DiClemente CC, Norcross JC: In search of how people change: Applications to addictive behaviors. *American Psychologist.* 1992, *47:*1102–1114.

(157) Curry SJ, Kristal AR, Bowen DJ: An application of the stage model of behavior change to dietary fat reduction. *Health Education Research.* 1992, *7:*97–105.

(158) Shepherd R, Stockley L: Nutrition knowledge, attitudes, and fat consumption. *Journal of the American Dietetic Association.* 1987, *87:*615–619.

(159) Prochaska JO, Velicer WF, Rossi JS, et al: Stages of change and decisional balance for twelve problem behaviors. *Health Psychology.* 1994, *13:*1–8.

(160) Brinberg D, Axelson ML: Increasing the consumption of dietary fiber: A decision theory analysis. *Health Education Research.* 1990, *5:*409–420.

(161) Kristal AK, Bowen DJ, Curry SJ, Shattuck AL, Henry HJ: Nutritional knowledge, attitudes, and perceived norms as correlates of selecting low-fat diets. *Health Education Research.* 1990, *5:*467–477.

(162) Bowen DJ, Henderson M, Iverson D, et al: Reducing dietary fat: Understanding the success of the Women's Health Trial. *Cancer Prevention International.* 1994, *1:*21–30.

(163) Henderson MM, Kushi LH, Thompson DJ, Gorbach SL: Feasibility of a randomized trial of a low-fat diet for the prevention of breast cancer: Dietary compliance in the Women's Health Trial vanguard study. *Preventive Medicine.* 1990, *19:*115–133.

(164) Kristal AR, White E, Shattuck AL, Curry S: Long-term maintenance of a low-fat diet: Durability of fat-related dietary habits in the Women's Health Trial. *Journal of the American Dietetic Association.* 1992, *92:*553–559.

(165) Tinker L, Burrows E, Henry H, et al: The Women's Health Initiative: Overview of the nutrition components. In Krummel D, Kris-Etherton P (eds), *Nutrition in Women's Health.* Garthersberg, MD, Aspen Publications, 1996, 510–542.

(166) Rollnick S: Behavior change in practice: Targeting individuals. *International Journal of Obesity.* 1996, *20*(Suppl. 1):522–526.

(167) Haskell WL, Alderman EL, Fair JM, et al: Effects of intensive multiple risk factor reduction of coronary atherosclerosis and clinical cardiac events in men and women with coronary artery disease: The Stanford Coronary Risk Intervention Project (SCRIP). *Circulation.* 1994, *89:*975–990.

(168) Dolecek TA, Milas NC, Van Horn LV, et al: A long-term nutrition intervention experience: Lipid responses and dietary adherence patterns in the Multiple Risk Factor Intervention Trial (MRFIT). *Journal of the American Dietetic Association.* 1986, *86:*752–758.

(169) Glanz K, Gilboy M: Physicians preventive care and applied nutrition: Selected literature. *Academic Medicine.* 1992, *67:*776–781.

(170) Peiss B, Kurleto B, Rubenfire M: Physicians and nurses can be effective educators in coronary risk reduction. *Journal of General Internal Medicine.* 1995, *10:*77–81.

(171) Calfas K, Long B, Sallis J, et al: A controlled trial of physician counseling to promote the adoption of physical activity. *Preventive Medicine.* 1996, *25:*225–233.

(172) Campbell MK, DeVellis B, Strecher V, et al: Improving dietary behavior: The effectiveness of tailored messages in primary care settings. *American Journal of Public Health.* 1994, *84:*783–787.

(173) National Cholesterol Education Program. Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents. *Pediatrics.* 1992, *89:*525–584.

(174) Gidding S: The rationale for lowering serum cholesterol levels in American children. *American Journal of Diseases of Children.* 1993, *147:*386–392.

(175) Perry CL, Stone EJ, Parcel GS, et al: School-based cardiovascular health promotion: The Child and Adolescent Trial for Cardiovascular Health (CATCH). *Journal of School Health.* 1990, *60*(8):406–413.

(176) Webber LS, Osganian SK, Luepker RV, et al: Cardiovascular risk factors among third grade children in four regions of the United States. *American Journal of Epidemiology.* 1995, *141*(5):428–439.

(177) Lytle LA, Johnson CC, Bachman K, et al: Successful recruitment strategies for school-based health promotion: Experiences from CATCH. *Journal of School Health.* 1994, *64*(10):405–409.

(178) Luepker R, Perry C, McKinlay S, et al: Outcomes of a field trial to improve children's dietary patterns and physical activity: Child and Adolescent Trial for Cardiovascular Health (CATCH). *Journal of the American Medical Association.* 1996, *257:*768–776.

(179) Nader P, Sellers D, Johnson C, et al: The effect of adult participation in a school-based family intervention to improve children's diet and physical activity: CATCH. *Preventive Medicine.* 1996, *25:*455–464.

(180) Weschler H, Wernick SM: A social marketing campaign to promote low-fat milk consumption in an inner-city Latino community. *Public Health Reports.* 1992, *107:*202–207.

(181) The DISC Collaborative Research Group: Cholesterol-lowering diet is effective and safe in children with elevated LDL-cholesterol: Three-year results of the Dietary Intervention Study in Children (DISC). Abstracts from the 67th Scientific Sessions, November 1994, Dallas, TX. *Circulation.* 1994, *90*(4):I-8, 39A.

(182) Shannon BM, Tershakovec AM, Martel JK, et al: Reduction of elevated LDL-cholesterol levels of 4- to 10-year-old children through home-based dietary education. *Pediatrics.* 1994, *94:*923–927.

(183) Tershakovec AM, Mitchell DC, Smickilas-Wright H, Hartzel J, Shannon BM: Lower fat intake in children. Abstracts from the 67th Scientific Sessions, November 1994, Dallas, TX. *Circulation.* 1994, *90*(4):I-8, 37A.

(184) Obarzanek E, Hunsberger S, Van Horn L, et al: Safety of a fat-reduced diet: The Dietary Intervention Study in Children (DISC). (in press, 1996).

(185) National Academy of Sciences: *Diet and Health.* Washington, DC: National Academy Press, 1989.

(186) Federation of American Societies for Experimental Biology: *Third Report of Nutrition Monitoring in the United States (Vol. 1).* Washington, DC: U.S. Government Printing Office, 1995.

(187) U.S. Department of Health and Human Services (DHHS) and U.S. Department of Agriculture (USDA). Ten-year comprehensive plan for the National Nutrition Monitoring and Related Research Program; Notice. Federal Register. 1993, *58:*32752–32806.

(188) Winnick M: Nutrition education in medical schools. *American Journal of Clinical Nutrition.* 1993, *58:*825–827.

(189) Barnard N, Akhtar A, Nicholson A: Factors that facilitate compliance to lower fat intake. *Archives of Family Medicine.* 1995, *4:*153–158.

(190) Kristal A, Patterson R, Glanz K, et al: Psychosocial correlates of healthful diets. Baseline results from the Working Well Study. *Preventive Medicine.* 1995, *24:*221–228.