Form **1040** U.S. Individual CHD Risk Return **1992** IRS Use Only—Do not write or staple in this space.

OMB No. 1545-0085

Label (See page 14.)

Use the IRS label. Otherwise, please print or type.

LABEL HERE

TAX DAY FOR DIETARY EXCESSES: IS THE PAYMENT CORONARY HEART DISEASE?

Your social security number

Spouse's social security number

For Privacy Act and Paperwork Reduction Act Notice, see page 4.

Presidential Election Campaign Fund (See page 15.)

	Yes	No
Do you want $1 to go to this fund?		
If a joint return, does your spouse want $1 to go to this fund?		

Note: *Checking "Yes" will not change your tax or reduce your refund.*

Filing Status (See Page 10) Check all that apply

1	Age
2	Sex
3	Family History
4	History of Previous Cardiovascular Disease (CVA, MI, Peripheral Vascular Disease)
5	Diabetes

Figure your Total and Adjusted Income Here

Attach Copy B of your Forms W2 or 1099-R Here

7	Total Calories	7			
8a	Taxable Saturated fat	8a			
8b	Tax-exempt monounsaturated and polyunsaturated fat	8b			
9	Dietary Cholesterol	9			
10	Trans fatty acids	10			
11	Coffee	11			
12	Business gain: Ice Cream Parties	12			
13	Capital (loss): fiber	13			
14	Capital (gain): Sunday Sausage Breakfast	14			
15	Other gains: Cheese Enchiladas	15			
16	Total IRA distributions: vitamin supplements	16			
17	Total Pensions and Annuities: Antioxidant vitamins	17			
18	Wine and Cheese Parties	18			
19	Farm (loss): fruits, grains, vegetables	19			
20	Unhealthy compensation: number of cigarettes smoked	20			
21	Add lines 7 through 20. This is your total adjusted income	21			

Figure your tax, credits and Payments

See instructions, Page A

22	Exercise	22			
23	Keeping Weight Lean	23			
24	Earned Income Credit: Aspirin Use	24			
25	Extension Request: Compensation for Overeating	25			
26	Reduction in Saturated Fat and Dietary Cholesterol	26			
27	Other Payments: Reduce Stress, Happiness	27			
28	Add lines 54 through 59. These are your total payments	28			

Figure your Refund or Amount you owe

29	If line 28 is more than line 21, subtract line 21 from line 28. This is the amount you OVERPAID.	29			
30	Amount of line 29 is your cardiovascular health in 1992	30			
31	Amount of line 29 you want applied to your 1993 Estimated Health	31			
32	If line 21 is more than line 28, subtract line 28 from line 21. This is the AMOUNT YOU OWE.	32			

Payment forms that will be accepted: Angina, Myocardial Infarction, Stoke, Claudication, Peripheral Vascular Disease.

Sign your return

Under penalties of perjury, I declare that I have examined this return and accompanying schedules and statements, and to the best of my knowledge and belief, they are true, correct, and complete. Declaration of preparer (other than the taxpayer) is based on all information of which the preparer has any knowledge.

Keep a copy of this return for your records.

Your signature	Date	Your occupation
Spouse's signature. If joint return, BOTH must sign.	Date	Spouse's occupation

Paid preparer's use only

Preparer's signature *MARGO A. DENKE, M.D.*

Firm's name (or yours if self-employed) and address: The Center for Human Nutrition, The University of Texas Southwestern Medical Center, Dallas, TX

Check if self-employed ☐ Preparer's social security no.

E.I. No.

ZIP code

1992

INTERNAL MEDICINE GRAND ROUNDS
APRIL 15, 1993

☆ U.S. GOVERNMENT

DIET AND LIFESTYLE MODIFICATION AND ITS RELATIONSHIP TO ATHEROSCLEROSIS

Margo A. Denke, M.D.

INTRODUCTION

Dietary and lifestyle modification could halve the rates of coronary heart disease (CHD) in the United States.[1,2] Dietary modification has been recommended for the American population as a whole,[3] and as specific therapy for hypercholesterolemia[4] and hypertension,[5] two known risk factors for CHD. This presentation will review the relationship between diet, lifestyle modification, and CHD. To begin, it will be important to review the magnitude of information necessary to implicate a diet-CHD relationship. Typically, this relationship is substantiated by testing the effects of diet on an intermediate CHD endpoint -- modifiable risk factors.

PROVING THE DIET-CHD THEORY

Proving the diet-CHD theory required integration of information collected by epidemiologists, with further refinements made by animal scientists and clinical researchers (Table 1). Specifically, world-wide comparisons found a significant correlation between dietary intake and CHD rates,[6,7] and studies tracking migrants showed that CHD rates changed to those of the new environment.[8] Prospective epidemiologic studies[9,10,11,12] suggested that dietary intake could predict the development of disease. Case control[13] and prospective[14,15] epidemiologic studies identified other "risk factors" that were associated with, preceded, and increased the probability of developing CHD. Additional studies suggested that some risk factors, notably serum cholesterol levels[11] and blood pressure,[16] were associated with differences in population intakes of specific dietary components. Animal models of human atherosclerosis in rat, hamster, primate, and pig demonstrated clear influence of diet on blood cholesterol levels, blood pressure, and anatomical changes in disease progression when these factors were modified.[17] Metabolic diet studies in humans precisely quantitated that changes in saturated fatty acids and cholesterol intake resulted in predictable changes in serum cholesterol levels;[18,19] similarly, changes in sodium, alcohol and caloric content of the diet could produce predictable changes in blood pressure.[20] Lastly, diet[21,22,23,24] and drug[25,26,27] intervention trials of subjects with hypercholesterolemia or hypertension demonstrated that lowering serum cholesterol levels or blood pressure, respectively, lowered CHD rates.

Much of the proof for the Diet-CHD Theory has utilized an intermediate step between diet - CHD: the relationship between diet and CHD risk factors.[28,29] This intermediate step provides a way to test scientifically the basis for a diet-CHD relationship. For this review, the quantitative relationship between dietary components and established CHD risk factors of 1) Total and LDL cholesterol, 2) Triglycerides and HDL cholesterol, 3) Blood Pressure, 4) Obesity, and 5) Diabetes will be presented.

The limitations of relying on this intermediate step to encompass all of the diet-CHD relationship should not be minimized. It is likely that diet alters CHD in other ways besides through established risk factors. New areas of nutrition research in blood coagulation and LDL oxidation, two suspected but not yet proven risk factors, will also be reviewed. These findings on specific dietary components will then be integrated into a general approach to dietary therapy for CHD prevention.

FACTORS THAT ALTER TOTAL AND LDL CHOLESTEROL LEVELS

FAT: SATURATED, MONOUNSATURATED & POLYUNSATURATED FATTY ACIDS

Fat is a mixture of saponifiable and unsaponifiable fractions (Table 2). The saponifiable fraction represents 95% of the fat, and is composed of triglycerides: three fatty acids attached by ester linkage to a glycerol backbone.[30] There are three major classes of fatty acids, characterized by the presence of double bonds in the fatty acid carbon chain: saturated, monounsaturated, and polyunsaturated fatty acids. Whereas fat is typically characterized by its predominant fatty acid (e.g., a "saturated fat"), every fat contains a mixture of the three fatty acids.

From metabolic ward diet studies, a change in saturated fatty acid intake explains 60-80% of the change observed in total cholesterol levels to dietary modifications.[18,19] Other dietary factors, including dietary cholesterol, fiber, monounsaturated fatty acids and polyunsaturated fatty acids, make up the remainder of the cholesterol-lowering dietary response. For this reason, the primary focus of a cholesterol-lowering diet should be a reduction in saturated fatty acids.

Saturated Fatty Acids

Four saturated fatty acids account for 90% of the saturated fatty acids in the American diet: lauric acid C12:0, myristic acid C14:0, palmitic acid C16:0, and stearic acid C18:0. Animal fats are rich in saturated fatty acids. A few vegetable oils, notably palm kernel oil, coconut oil, palm oil, cocoa butter, and cottonseed oil, contain more than 25% saturated fatty acids (Table 3).[31]

The estimated saturated fatty acid intake in America is 20-60 gm/d.[32,33,34] Only three of the four common saturated fatty acids raise serum cholesterol levels.[35] These saturated fatty acids are carbon length 12-16 (S^1). Medium chain triglycerides (C8:0, C10:0)[36] and stearic acid[37] do not raise total or LDL cholesterol levels. Saturated fatty acids raise LDL cholesterol levels by reducing LDL receptor activity.[38]

Whereas one can select specific fats for their saturated fat content (Table 3), the majority of saturated fat is consumed as part of food: fatty meats, whole fat dairy products, and fat hidden in products such as baked goods or casseroles.[39] In general, ingestion of 10 gm of saturated fatty acids daily for several weeks will raise total and LDL cholesterol levels 8-10 mg/dl.[18,19] The effects of intermittent ingestion depends on the average intake of saturated fat for a several week period.[40]

Monounsaturated Fatty Acids

Monounsaturated fatty acids can be subcategorized by the positional isomer of the double bond: cis-monounsaturated and trans-monounsaturated.

1. **Cis-monounsaturated Fatty Acids**

The most common cis-monounsaturated fatty acid is oleic acid, C18:1. Cis-monounsaturated fats are ubiquitous in both animal and vegetable fats. While animal fats are high in monounsaturated fatty acids, they are also rich in saturated fatty acids. Low saturated fat vegetable oils with more than 50% of their fatty acids as monounsaturated are olive oil, high oleic safflower oil, high-oleic sunflower oil, and canola oil (Table 3).

The average cis-monounsaturated fatty acid intake is 20-50 gm/d,[32,33,34] with half coming from animal and half from vegetable sources. Cis-monounsaturated fatty acids are neutral in that they neither raise nor lower total and LDL cholesterol levels.[41] Diets high in cis-monounsaturated fatty acids and low in saturated fatty acids, as consumed during the 1950's in the Mediterranean region, have been associated with low rates of CHD.[11]

2. **Trans-monounsaturated fatty acids**

While most monounsaturated fatty acids are of the cis configuration, trans-configured monounsaturates can be found in certain meat fats as well as hydrogenated vegetable oils. Hydrogenation is a fat processing technique where vegetable oils with undesirable characteristics (hardness, crystalline structure, potential to become rancid, etc.) are altered to form hydrogenated fats with more desirable characteristics. Hydrogenation converts polyunsaturated fatty acids into cis-monounsaturated fatty acids and trans-monounsaturated fatty acids with lesser quantities of saturated fatty acids and trans-polyunsaturated fatty acids being formed.[30] Hydrogenation occurs naturally by action of bacterial degradation of fatty acids in ruminant animals; hence, trans fatty acids are also present in milk fat, beef fat, and mutton fat.

The estimated American intake of trans fatty acids is 6-8 gm/d. One-third of the dietary trans comes from animal fats, while two-thirds is from hydrogenated vegetable oils. The trans fatty acid content of a particular product using hydrogenated oil depends on its manufacturing characteristics.[42] To remove easily oxidized polyunsaturated fatty acids, only minimal hydrogenation is required. For example, cookies and crackers contain typically 3-9% trans; snack foods, 8-10% trans. To change the melting point of a fat, more extensive hydrogenation is required. For example, tub margarines contain 13-20% trans, whereas stick margarines and shortenings contain 25-35% trans.

Several studies suggest that a specific trans-monounsaturated fatty acid, elaidic acid (C18:1), raises total and LDL cholesterol levels;[43] recent epidemiologic investigations suggest that trans fatty acid intakes are related to lipid levels in

free-living populations.[44] If these findings can be confirmed, a causal relationship between trans fatty acid intake and CHD could be suggested. Trans fatty acids may be one-third less potent than saturated fatty acids in raising total and LDL levels.[45] If a linear relationship is confirmed, ingestion of 8 gm elaidic acid could raise total and LDL cholesterol levels 5-7 mg/dl.

Polyunsaturated Fatty Acids

There are two types of polyunsaturated fatty acids: Omega 6 and Omega 3. These fatty acids are named by the location of the first double bond. Omega 6 fatty acids are primarily found in vegetable oils. Omega 3 fatty acids are primarily found in fish oils.

1. **Omega 6 fatty acids**

The most common Omega 6 polyunsaturated fatty acid in the diet is linoleic C18:2. Polyunsaturated fatty acids are found in high concentrations exclusively in vegetable oils. Common low saturated fat vegetable oils with more than 50% of their fatty acids as polyunsaturated are safflower oil, corn oil, and soybean oil (Table 3).

The average polyunsaturated fatty acid intake is 10-30 gm/d.[32,33,34] Polyunsaturated fatty acids were once felt to have a unique cholesterol lowering property that could counterbalance the cholesterol raising effects of saturated fatty acids.[18,19] This notion was popularized by rating fats based on their polyunsaturated to saturated fat or P/S ratio; this ratio does not convey the cholesterol raising potential of a fat, and should not be used (e.g., olive oil [Table 3]). More recent metabolic diet studies have shown that polyunsaturated fatty acids are roughly equivalent to monounsaturated fatty acids in their effects on total and LDL cholesterol levels.[35] That is, neither monounsaturated nor polyunsaturated fatty acids raise or lower total and LDL cholesterol levels. No population has consumed a high polyunsaturated fat diet for a prolonged enough period of time to establish the relationship between dietary intake and CHD rates.

2. **Omega 3 fatty acids**

The most common long chain omega 3 fatty acids are eicosapentaenoic (EPA; C20:4) and docosahexaenoic (DHA;C22:6). Omega 3 fatty acids are found predominately in fish oils; some vegetable oils (notably canola oil and soybean oil) have a shorter chain omega 3 fatty acid, linolenic (C18:3). The average omega 3 fatty acid intake in America is 0-5 gm/d. While initial reports suggested a unique cholesterol lowering property for Omega 3 fatty acids,[46] follow-up studies confirmed that omega 3 fatty acids are neutral in their effects on total and LDL cholesterol levels.[47] Nevertheless, countries with high dietary intakes of long-chain omega 3 fatty acids have low rates of CHD.[48]

DIETARY CHOLESTEROL

Dietary cholesterol is found in the unsaponifiable fraction of animal fats (Table 2), and in animal cell membranes. Dietary cholesterol is present in all animal products, including, meats, poultry, and fish at 15-30 mg/oz and also in dairy fat. Egg yolks (213 mg/yolk) and organ meats (liver, 110 mg/oz; brain, 580 mg/oz) are rich in dietary cholesterol.

The estimated dietary intake of cholesterol in America is 300-500 mg/d. Unlike fatty acids, which are >95% absorbed,[49] only 40-60% of dietary cholesterol is absorbed. Even after correction for variable absorption,[50] the effects of dietary cholesterol on total and LDL cholesterol levels remains variable. Some patients exhibit an exuberant increase in LDL levels with chronic ingestion while other show little response.[51] The variability in response remains unexplained. Theoretical explanations include Apo E characteristics[52] (an apoprotein which aides in LDL metabolism), or inherent differences in the ability to down-regulate endogenous cholesterol synthesis.[53] For populations, the expected serum total and LDL cholesterol raising effect of 25 mg of dietary cholesterol is 1 mg/dl. The connection between dietary cholesterol and CHD is impressive: multivariate analysis of several prospective studies [10,12] has shown significant positive relationships between dietary cholesterol intake and subsequent CHD.

DIETARY PHYTOSTEROLS

Dietary phytosterols are found in the unsaponifiable fraction of vegetable fats. The major phytosterol, beta-sitosterol, is poorly absorbed. Beta sitosterol can block absorption of dietary cholesterol on a 1:1 basis.[54]

The average phytosterol content of the diet is 300-500 mg/d. Phytosterols are found in all vegetable oils, and in high concentrations in corn oil and rice bran oil. The high phytosterol content of these oils may partially explain their greater than predicted cholesterol lowering properties.[55,56] In populations consuming higher quantities of dietary cholesterol phytosterol compounds have produced effective total and LDL cholesterol lowering of 20-30 mg/dl. For populations consuming less than 200 mg dietary cholesterol/d, 3 gm of beta-sitostanol (a hydrogenated derivative) produced LDL cholesterol lowering of 6 mg/dl.[57]

FIBER

Dietary fiber can be characterized in several ways: digestible/nondigestible, soluble/insoluble, crude/dietary, etc. Water solubility appears to confer the lipid lowering effect of fiber.[58]

The average total dietary fiber intake in America is 5-20 gm/d. Soluble fibers lower total and LDL cholesterol levels, while insoluble fibers do not. Daily intake of 2 ounces oat bran (11 gm total fiber; 6 gm soluble fiber) or oatmeal (5 gm total fiber; 3 gm soluble fiber) can lower total and LDL cholesterol levels 5 mg/dl.[59,60] The mechanism by which dietary fiber lowers cholesterol levels has been elusive:

attractive possibilities such as a reduction in fat or bile acid absorption have not been supported by studies analyzing fecal fat or fecal bile acid excretion.

CALCIUM

Dietary calcium has been shown to reduce total and LDL cholesterol levels. Supplementation of a diet rich in saturated fatty acids with 2 gm of elemental calcium has produced an 18 mg/dl fall in LDL cholesterol levels,[61] and at more moderate intake of saturated acids, an 8 mg/dl fall in LDL.[62] The exact mechanism of action is unclear; there is evidence that excess dietary calcium can minimally interfere with fat absorption, particularly saturated fatty acids, which require longer time for absorption.

ALCOHOL

Alcohol does not affect total and LDL cholesterol levels in populations. Anecdotally, hypercholesterolemia in an alcoholic can remit with alcohol cessation.

COFFEE

Isolated reports have claimed caffeinated and decaffeinated coffee is associated with higher total and LDL cholesterol levels,[63,64] but just as many reports[65,66] have shown no association. Carefully performed metabolic studies suggest that the fat extracted by boiling coffee beans has an unsaponifiable fraction that can raise total and LDL cholesterol levels.[67] This fat is captured by paper filters; since American coffee is typically filtered coffee, it is unlikely that coffee consumption will play a major role in altering lipid levels.[68]

BODY WEIGHT/ENERGY BALANCE

Multiple cross-sectional population studies have shown that total and LDL cholesterol levels correlate positively with body weight.[69,70] Three large, prospective studies[12,71,72] have observed that when participants gained weight, their total cholesterol levels rose; conversely when they lost weight, serum cholesterol levels fell. These associations have been observed in small, metabolic-ward studies[73,74] and in a meta analysis of exercise induced weight loss.[75] When weight loss occurred during cholesterol-lowering dietary modification, as in The Diet-Heart Feasibility Study,[76] the Multiple Risk Factor Intervention Trial (MRFIT),[77] and the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT)[78], greater cholesterol lowering was achieved.

The Framingham Study has estimated that for every 10 lb. weight gain, total cholesterol level increases 7 mg/dl in men, and 5 mg/dl in women.[71] In the MRFIT study, while Step-One dietary modification produced a 9 mg/dl fall in LDL, dietary modification plus a 10 lb weight loss produced a 14 mg/dl fall in LDL cholesterol levels.[77] This enhanced reduction persists over years,[20] and reinforces the need to encourage leaner body weights.

CONSIDERATIONS FOR PATIENTS WITH SEVERE LDL DYSLIPIDEMIA

Patients with extremely high total (>300 mg/dl) and LDL cholesterol levels (>220 mg/dl) are likely to have a predominantly genetic cause for their dyslipidemia. This should not imply that dietary modification will be unsuccessful. In fact, on a percentage basis, patients with hypercholesterolemia experience greater reductions in total and LDL cholesterol with diet than patients with normal cholesterol levels.[79] The lipid lowering achieved by diet is additive to the lipid lowering achieved by drug therapy.[80] Table 4 lists practical advice summarizing the major dietary factors that alter LDL cholesterol levels.

DIETARY FACTORS THAT ALTER TG/HDL LEVELS

It has yet to be proven that raising HDL cholesterol levels with either diet or drugs will lower CHD rates. Since the link between diet, total and LDL cholesterol levels and CHD is well established, any dietary therapy proposed for raising HDL cholesterol levels must not raise LDL cholesterol levels. In this way, the recommended diet clearly would lower CHD risk through LDL lowering and theoretically could lower CHD risk through HDL raising. Since the metabolism of HDL cholesterol is inextricably linked to the metabolism of triglycerides, dietary factors that affect either TG/HDL will be presented.

CALORIES / ENERGY BALANCE / BODY WEIGHT:

HDL cholesterol levels are inversely associated with body weight, and TG levels are positively associated.[69,70] Similar to the LDL-body weight relationship, weight reduction through either caloric restriction or caloric expenditure have been shown to lower TG levels and raise HDL cholesterol levels.[75,81] The time course of this change in HDL cholesterol levels should be noted. Whereas TG levels change acutely with caloric intake, HDL cholesterol levels measured during a hypocaloric diet may be even lower than before the diet was initiated.[82,83] After weight loss has been achieved, HDL cholesterol levels typically rise over a 3-6 month period to a higher concentration.

In general, obese individuals - either men or women - have 5-10 mg/dL consistently lower HDL cholesterol levels as compared to lean counterparts, and 35-110 mg/dl higher triglyceride levels.[69,70] The addition of exercise to the weight loss program will enhance the increase in HDL cholesterol levels above weight reduction alone.[81]

ALCOHOL:

Alcohol is the most striking predictor of TG levels in population studies[84,85], but the effect of alcohol at raising TG levels is only a transient effect in metabolic ward studies of young, lean, healthy subjects[86]. This might suggest that alcohol ingestion by the population is intermittent; alternatively, it might suggest that the alcohol effect observed in populations reflects the effects of alcohol in older, fatter subjects. This latter hypothesis is supported by studies documenting the graded effects of alcohol on TG and HDL cholesterol levels in older subjects.[87] The effects of alcohol on raising TG levels appear to be dose dependent and dependent upon the baseline TG level; while subjects with triglycerides under 150 mg/dl typically have little increase

with alcohol, subjects with higher baseline triglycerides have more dramatic increases of 50-100 mg/dl[88]. Since overweight individuals already have an overproduction of triglyceride-rich lipoprotein,[89] alcohol may be more likely to produce increases in triglycerides in overweight individuals than in lean individuals.

In subjects with normal triglycerides, the predominant lipid effect of alcohol is to raise HDL cholesterol levels.[90] This effect is dose dependent, and at a daily dose of 63 gm of ethanol (4-5 drinks),[a] HDL cholesterol levels may increase 5-12 mg/dl. Whether or not this increase in HDL cholesterol levels explains the reduced total mortality observed among light drinkers[91] is unknown.[92] Current dietary recommendations do not advocate alcohol ingestion for the purpose of reducing CHD risk.[3]

FAT / CARBOHYDRATES:

The balance of percent calories from carbohydrate vs percent calories from fat influences triglyceride and HDL cholesterol levels. Carbohydrates tend to raise triglycerides and lower HDL, but this effect is only statistically significant with very-low-fat diets (i.e., less than 25% of calories from fat or more than 60% of calories from carbohydrate)[93]. In normolipidemic subjects, the magnitude of triglyceride raising from very-low-fat diets is typically 30-100 mg/dl, with a concomitant reduction of HDL of 3-8 mg/dl.[94] While some have argued that these adverse changes with high-carbohydrate, very-low-fat diets are transient,[95] an international sampling of diet and lipid levels in young boys suggests they are chronic and graded.[96]

Despite the "adverse" effects of high carbohydrate/very-low-fat diets on triglycerides and HDL cholesterol levels, population studies do not support an increase in CHD risk on these diets.[97] Reasons for this discrepancy are multiple: 1) Such populations may have low prevalence rates of other CHD risk factors such as hypertension, obesity, physical inactivity,[98] 2) such populations have low LDL cholesterol levels from their low saturated fat, low cholesterol diet,[99] and 3) studies of lipoprotein composition suggest that high carbohydrate/very-low-fat diets produce large VLDL particles which are different than the dense, cholesterol rich VLDL observed in states of abnormal triglyceride metabolism.[100] Clearly a better understanding of the role of HDL and VLDL in atherogenesis will be needed before the long term consequences of a high carbohydrate/very-low-fat diet can be predicted.[101]

Saturated, cis-monounsaturated and omega 6 polyunsaturated fatty acids

Besides percent calories from fat, the fatty acid composition of the diet can influence TG and HDL cholesterol levels. Medium chain saturated fatty acids behave similar to carbohydrate in raising TG levels.[102] Long chain fatty acids -- saturated,[103] cis-

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a. COMPARISON OF ALCOHOL DOSE BY TYPE

TYPE	% ETHANOL	"ONE DRINK"	AMOUNT ETHANOL
Beer	3-5%	12 Oz.	13.6 gm.
Wine	12%	5 Oz.	13.6 gm.
Spirits	40%	1.5 Oz.	13.6 gm.

monounsaturated,[104] and polyunsaturated,[105] tend to lower TG and raise HDL cholesterol levels. This is not to imply that fat should be added indiscriminately to the diets of patients with low HDL levels. Fat is calorie dense, and simply increasing dietary fat would lead to weight gain, further depressing HDL levels.[69,70] Very high intakes of polyunsaturated fatty acids (i.e., > 13% of calories) lowers HDL cholesterol levels.[106] S^1 saturated fatty acids also raise LDL cholesterol levels.[35] Therefore, recommendations for increasing dietary fat to raise HDL cholesterol levels should be given to lean patients with instructions to add monounsaturated fatty acids to the diet using foods low in saturated fatty acids (e.g., oils, nuts, avocados).

Trans-Monounsaturated Fatty Acids

High doses (34 g/d) of trans-monounsaturated fatty acids have been shown to lower HDL cholesterol levels 6 mg/dl.[107] Whether typical intakes of trans fatty acids also produce HDL cholesterol lowering has yet to be determined. If a linear relationship was confirmed, current intake of 6-8 gm/d of trans-fatty acid could produce a 1 mg/dl lowering of HDL.

Omega 3 Fatty Acids

Of all dietary constituents, long chain omega-3 polyunsaturated fatty acids have the most potent effect on triglyceride levels. In normal subjects, omega 3 fatty acids lower triglycerides 30-50 mg/dl,[108] with little effect on HDL levels.[109] The omega 3 lowering effect has been best demonstrated with fish oil administration with 3-7 gm given in capsules. Since large quantities of fatty cold water fish (salmon, mackerel) must be consumed to achieve this intake, from a practical standpoint, small changes in dietary omega 3's will have little effect on triglyceride levels in normal people.[110]

CONSIDERATIONS FOR INDIVIDUALS WITH SEVERE TG/HDL DYSLIPIDEMIA

While secondary causes of TG/HDL dyslipidemia such as diabetes and hypothyroidism respond best to control of the primary metabolic disorder,[111,112] dietary therapy can play an important role in primary dislipidemia. Disturbances of chylomicron and remnant metabolism such as Type III hyperlipoproteinemia and acute chylomicronemia syndrome may be exacerbated by high fat diets which increase the exogenous contributions of dietary lipids to total blood lipids.[113] On the other hand, chronic Type IV and V hyperlipidemias generally improve on high fat diets because a high fat diet avoids carbohydrate induced hypertriglyceridemia.[114] Some general dietary principles apply (Table 5). High-carbohydrate/very-low-fat diets may increase triglycerides 50-300 mg/dl,[115] but if these diets produce weight loss, a reduction in fasting triglycerides can be achieved. Omega 3 fatty acids may lower triglyceride levels 100-1200 mg/dl in hypertriglyceridemic patients.[116,117] Alcohol abstinence in hypertriglyceridemic subjects whose ethanol intake is greater than 20-30 gm/d[118] may achieve further triglyceride lowering. Unfortunately, HDL cholesterol levels may not return to normal levels despite good control of fasting triglycerides. That is, although the triglyceride abnormality can be corrected with diet, HDL cholesterol levels, while increasing somewhat, may remain below 35 mg/dl.[119]

DIETARY FACTORS THAT ALTER NON-LIPID RISK FACTORS FOR CHD

HYPERTENSION

Body weight/caloric balance

The Framingham Study estimates that for every 10 lb. excess weight, systolic blood pressure increases 4 mmHg in men and women.[71] Weight change can be a good predictor of blood pressure.[120] Weight loss of at least 6-10 kg in obese hypertensives reduced systolic blood pressure 20-48 mmHg, and diastolic blood pressure 18-24 mmHg;[121] smaller changes have been observed with lesser degrees of weight loss.[122]

Sodium

Changes in dietary sodium intake of 2.4 gm/d has been associated with a 2.2 mmHg difference in systolic blood pressure for populations. These INTERSALT findings[123] have lead some to conclude that dietary sodium plays a relatively minor role in determining blood pressure. However, several studies have suggested that only some individuals are "sodium sensitive". For example, INTERSALT found that lower sodium diets attenuate by 9 mmHg the higher blood pressure observed in older ages. Hypertensives switching to a 2 gm sodium diet experienced a 10 mmHg drop in systolic and a 6 mmHg drop in diastolic pressure;[124] obese adolescents appear more sensitive to dietary sodium than non-obese.[125] Therefore, it seems prudent to restrict dietary sodium in individuals who are older, overweight, or hypertensive.[126]

Alcohol

Alcohol consumption has been associated with higher blood pressures in cross-sectional studies.[127,128] Cessation of alcohol intake has been shown to produce a 4-8 mmHg reduction in systolic blood pressure within three days.[129]

Other Factors

Some investigators suggest that blood pressure can be reduced by polyunsaturated fatty acids,[130] but this has yet to be proven. Similarly, work proceeds on the effects of other cations, notably potassium and calcium, on lowering blood pressure.[131]

DIABETES

Diabetic control can be enhanced by several dietary maneuvers: weight loss, meal planning to standardize daily intake and to space out calories during the day, and reducing ingestion of concentrated sweets.[132,133,134] A major controversy continues as to whether diabetics should follow a low fat/high carbohydrate diet or a high fat/low carbohydrate diet. However, while there is little evidence that diabetic control per se can be altered by source of calories, diabetic dyslipidemia can be improved by a high fat/low carbohydrate diet.[135] Nevertheless, a hypocaloric diet will improve the diabetic dyslipidemia irrespective of its macro-nutrient balance.

OBESITY

Caloric imbalance -- excessive intake and/or inadequate expenditure -- promotes weight gain.[136] The magnitude of weight gain appears dependent on the number of calories , but not the source of calories (fat, carbohydrate, protein, alcohol). Overweight individuals tend to have eating patterns of few, infrequent meals.[137] Many people suffer from a "yo-yo" dietary syndrome with cycles of weight loss followed by weight gain. Unfortunately, no breakthrough has been made in the treatment of this prevalent CHD risk factor.[138] Treatments for obesity with very-low-calorie diets (600-1,000 calories/d), typically administered by liquid diets rich in protein, vitamins and minerals, are effective at producing weight loss and are a popular means of achieving a "quick fix".[139] Unfortunately, treatment for obesity has a high recidivism rate.[138] Successful weight reduction programs include several means to improve caloric balance: behavior modification targeted at binge eating, caloric restriction while training new dietary habits, and maintaining good caloric expenditure through exercise.

Obesity is an independent risk factor for CHD.[140] However, excess body weight also confers CHD risk through raising total and LDL cholesterol and triglycerides, lowering HDL cholesterol, and raising systolic and diastolic blood pressure.[141]

DIETARY FACTORS THAT ALTER FACTORS SUSPECTED TO INCREASE CHD RISK

LDL OXIDATION

New evidence suggests that degradation (so called oxidation) of the LDL particle is a necessary step for LDL to be taken up by macrophages, which in turn become foam cells - the progenitor of the fatty streak. In vitro tests of LDL oxidation have been established to evaluate if the oxidation potential can be changed.[142] LDL particles containing polyunsaturated fatty acids are more likely to undergo oxidation than LDL particles containing either monounsaturated or saturated fatty acids.[143] In addition to LDL fatty acid content, a high micronutrient content of the plasma (Vitamin C)[144] and of the LDL particle itself (Vitamin E,[145] Vitamin A[146]), appears to retard the oxidative process.[147,148] Since polyunsaturated oils are rich in antioxidants (Table 1), the impact of ingesting polyunsaturated oils on LDL oxidation may be neutral. Further research is needed as to the benefit and need of antioxidant vitamin supplements. In the meantime, a CHD-prevention diet enriched with a variety of fruits, vegetables, and grains will provide these antioxidants, albeit in lower doses than can be achieved using supplementation.

BLOOD COAGULATION

The final event in a myocardial infarction is a thrombus that completely occludes the arterial lumen. Multiple investigations have evaluated possible links between coagulation and diet. In general, it appears that diets high in omega 3 fatty acids can reduce thrombotic tendency of platelets.[149] While some have suggested that saturated fatty acids can increase thrombotic tendency,[150] the inherent variability in

measurements of clotting parameters make this hypothesis difficult to prove. A recent report suggests a relationship between high fat meal ingestion and thrombosis; clearly further research is needed to substantiate these speculations.

PRESCRIBING A CHD PREVENTION DIET: KEYS TO SUCCESS

DIETARY PRESCRIPTIONS

The recommendation for a cholesterol-lowering and blood-pressure lowering diet is specified in Table 7.[151] Two diets are proposed: A Step-One diet for the population at large, and a Step-Two diet for intensive dietary treatment for patients in whom the Step-One diet has failed to achieve target LDL cholesterol goals.[4] An alternative way to describe this diet uses servings of food groups (United States Department of Agriculture, Figure 1). These diet prescriptions are nutritionally balanced, and can be adopted by the entire family.[152] The soon-to-be released second report of the Adult Treatment Panel of the National Cholesterol Education Program will include menu plans to illustrate how a Step-One or Step-Two can be achieved.

Some physicians question whether these diets are restrictive enough to prevent CHD or retard its progression. Alternative diets, typically very-low-fat-vegetarian diets,[153] greatly exceed the guidelines in Table 7 through intense restriction of dietary intake. While these diets provide alternatives, it remains to be seen whether they confer more favorable lipid lowering or CHD prevention.[154] For example, in the Lifestyle Heart Trial,[23] the treatment group not only followed a very-low-fat, vegetarian diet, but also participated in regular exercise, lost 10 kg of weight, and meditated. How much these additional changes augmented the benefit attributed to the very-low-fat vegetarian diet cannot be determined. Since alternative diets require greater patient commitment than traditional diets, the physician should begin dietary counselling using the current guidelines. Traditional diets provide for the use of a wide variety of foods, and this variety can make the diet enjoyable. Alternative diets, if nutritionally adequate, can be followed by patients who desire more restricted dietary intakes.

EVOLVING AREAS IN PRACTICAL ASPECTS OF DIET

Food Labeling

In January 1993, the Food and Drug Administration published new regulations for food labeling.[155] These regulations will simplify and standardize labeling of food products. Every label will list calories, calories from fat, total fat, saturated fat, cholesterol, sodium, total carbohydrate, dietary fiber, sugars, protein, Vitamin A, Vitamin C, calcium, and iron content. The percent of daily value for a 2,000 calorie diet will guide consumers as to how a specific food contributes to the overall diet. Suppliers of fruits, vegetables and processed meats will be required to display similar information at point-of-purchase. Descriptive terms, such as "reduced", "light", "low", and "free" will be restricted to products that meet specific requirements (Table 8). The FDA has defined saturated fat as all dietary saturates including medium chain triglycerides and stearic acid. Therefore, the labelled saturated fat content will overestimate the cholesterol raising potential of cocoa butter by 130%, beef fat by 66%, dairy fat by 59%, lard by 56%, coconut oil by 24%, and palm kernel oil by

12% (Table 3). Unfortunately, these overestimations may reduce the perceived role that lean red meats, low fat dairy products, and occasional chocolate consumption can provide to the variety of foods used in a cholesterol-lowering diet.

Food Manufacture

Food manufacturers must balance consumer demand on one hand with taste, convenience, cost, and nutritional value on the other. New products have been successfully developed, marketed, and sold that are lower in fat, cholesterol, and sodium. This product development has relied on multiple technologies. Low fat and non-fat salad dressings have been developed using water soluble gums to replace fat. Other water-based products, such as frozen desserts, have substituted lower fat ingredients (low fat milk, yogurt plus gums) for full fat ingredients with good success. Similarly, cheese made with part skim milk have been well received.

The food manufacturer's dilemma has been how to reduce the fat content in low moisture foods such as crackers, chips, and fried foods while maintaining the sensory properties of these foods. Cooking methods have been modified to create different tasting products. (e.g., baking corn chips and spraying later with fat). However, to create more acceptable products to consumers, a fat substitute is needed. Fat substitutes can mimic the sensory properties of fat. One improved fat substitute is Simplese® (Monsanto Company), a product made from rearranged egg whites recently approved by the FDA as generally recognized as safe. Simplese®, as a digestible protein, is 4 cal/gm. Since protein bonds degrade with cooking, Simplese® cannot substitute for fats that are subject to heating. Simplese® has been incorporated into water-based foods such as frozen desserts, and in non-heated foods such as frostings. For a fat substitute that has both the sensory and cooking properties of fat, artificial fats such as Olestra® (Procter and Gamble Company) have been developed and are awaiting approval by the FDA. Olestra® is composed of fatty acids attached by ester linkage to a ring of carbons instead of the straight glycerol molecule (Table 2). Because of stearic limitations, the fatty acids on Olestra® cannot be cleaved by intestinal lipases, and therefore Olestra® passes down the intestine without being absorbed. Olestra® can be used in lieu of fat for any purpose (salad dressings, frozen desserts, baked goods, frying oils, etc), and has 0 calories/gm. Additional fat replacements are under development, and may reach the marketplace if approved by the FDA.

Plant Products

Selective plant breeding has improved the fatty acid content and per-acre yield of seeds harvested for oil. The most striking example is Canola oil, harvested from rapeseed. Canola oil is one of the lowest in saturated fatty acids (Table 3); work is underway to selectively breed other seeds for saturated fatty acid content, vitamin content, taste, and growth characteristics.

Animal Products

Similar to the food and plant industries, meat producers have responded to consumer demands for healthier products. Leaner cows are sold at market than were offered

20 years ago; name brand beef products guaranteeing leaner content are now widely distributed. Chickens and turkeys have been inbred to produce strains that have more white meat. New technologies are continuing; for example, the saturated fat content of pork can be reduced if pigs are fed a diet enriched in monounsaturated fat.[156]

SUMMARY & CONCLUSIONS

Much of the CHD-protective effects of diet are mediated through the effects of diet on risk factor levels. The best studied are the effects of diet on lipoprotein levels and blood pressure. With these effects in mind, specific dietary recommendations have been made to reduce CHD rates among healthy Americans.[2,3,4,5] Dietary modification will reduce risk factor levels in the majority of people, and will have measurable impact on CHD rates in the nation.[1,2]

In the treatment of patients, it is important to distinguish which lipoprotein and blood pressure abnormalities are amenable to diet, and which ones will require drug therapy. This distinction can be made by understanding the quantitative relationship between dietary intake and risk factor levels that have been presented above, and by assessing the current dietary intake of the individual. There must be a significant change in dietary composition in order to produce a significant change in risk factor level. This point, although it appears obvious when stated, is a common source of confusion concerning the efficacy of diet. For individuals, most non-response to dietary change reflects the lack of a dietary change.[76] Poor adherence, in turn, may be the direct result of inadequate diet counselling.[157,158] Counselling with a registered dietician may be useful not only in educating the high-risk patient concerning the nutrient content of food, but also in establishing the current dietary intake of the patient.

Compliance is not the only cause of non-response. For some patients who are already following a CHD-prevention diet, additional risk factor reductions may not be evident when minor dietary constituents are altered. Since the majority of the diet effect has already been achieved, this "non-response" should not be surprising. The benefit of a change in a minor dietary constituent may not be detectable above the random day-to-day variation in risk factor levels. Lastly, while true non-responders to diet have been observed, at least for blood cholesterol levels, the prevalence of non-response or minimal response is less than 10% of the population.[76]

Although the small changes in risk factor levels achieved by diet cannot compare with the greater changes achieved by drug therapy, dietary therapy will always have an important place in altering the health of the nation and its people. For some individuals, dietary and lifestyle modification may be all that is needed for CHD prevention; for others, dietary and lifestyle modification will provide a foundation upon which drug therapy can be added for maximum CHD risk reduction.

TABLE 1

LINES OF SCIENTIFIC EVIDENCE SUPPORTING THE DIET-CHD THEORY

- Population data demonstrating a trend between both food balance and food disappearance data and national rates of disease.
- Epidemiologic studies showing that CHD rates of migrants were more similar to the rates of the adopted environment than the native environment.
- Epidemiologic prospective studies suggesting an association between dietary intake and the subsequent development disease.
- Epidemiologic case-control studies suggesting that specific factors (so called risk-factors) are more prevalent among cases than controls.
- Epidemiologic, prospective studies identifying risk factors that precede disease and increase the probability of developing subsequent disease.
- Case-control and prospective epidemiologic studies suggesting an association with dietary intake and levels of risk factor.
- Animal models confirming that the development of a disease can be altered by changes in dietary intake, and further investigating mechanisms for a how specific dietary constituent could alter the development of disease.
- Clinical intervention trials in animals documenting that altering dietary intake indeed alters endpoints. These endpoints can be risk factors, tests of disease, or disease rates.
- Human metabolic studies quantitating the relationship between changes in dietary intake of a specific dietary constituent and changes in risk factor level.
- Clinical intervention trials in humans documenting that altering dietary intake alters endpoint measurements of either risk factors, prevalence of disease as estimated by tests for this disease or by disease endpoints themselves.

Table 2

HOW TO CHARACTERIZE A FAT

FAT

95%
SAPONIFIABLES

Triglycerides

Glycerol
+
Fatty Acids of various types

Saturated (S)
Medium Chain (MCT: derived from coconut oil)
Cholesterol-raising saturates (S^1)

Monounsaturated (M)
cis-monounsaturated (animal and vegetable oils)
trans-monounsaturated (hydrogenated fats; some animal fats)

Polyunsaturated (P)
omega-6 polyunsaturated (vegetable oil)
omega-3 polyunsaturated (fish oil)

5%
UNSAPONIFIABLES

Sterols
Phytosterols (vegetable)
Cholesterol (animal)

Vitamins
Minerals

Other substances
(as in coffee)

TABLE 3

FATTY ACID COMPOSITION OF COMMON FATS AND OILS RANKED BY CHOLESTEROL RAISING SATURATED FATTY ACID CONTENT

	S	S^1	M	P	P/S Ratio
Palm Kernal	81	72	11	2	.0
Coconut	87	70	6	2	.0
Palm	49	45	37	9	.2
Butter Oil	62	39	29	4	.1
Beef Tallow	50	30	42	4	.1
Cocoa Butter	60	26	33	3	.1
Lard	39	25	45	11	.3
Cottonseed	26	24	18	52	2.0
Chicken Fat	30	23	50	21	.7
Olive	14	11	74	8	.6
Corn	13	11	24	59	4.6
Soybean	14	10	23	58	4.0
Peanut	17	10	46	32	1.9
Safflower	9	6	12	75	8.2
Canola	7	5	56	33	4.9
Safflower-Hi Oleic	6	5	75	14	2.3

S = Total saturated fatty acids; the definition currently used by FDA.

S^1 = Cholesterol raising saturated fatty acid (C12:0, C14:0, C16:0)

M = Monounsaturated fatty acids

P = Polyunsaturated fatty acids

TABLE 4

PRACTICAL ASPECTS OF FACTORS WHICH ALTER
TOTAL AND LDL CHOLESTEROL LEVELS

FACTOR	DIETARY GOAL	NUANCES
Body Weight/ Caloric Balance	Reduce/Improve	Weight reduction enhances lipid lowering achieved by dietary composition changes.
Saturated Fatty Acids	Reduce	Decrease consumption of full fat dairy products, fatty meats, meats ground with fat added, and full fat baked goods. Since a common saturated fatty acid, stearic acid, is not cholesterol raising, lean cuts of beef have equivalent cholesterol raising potential to skinless dark chicken meat; cocoa butter does not raise cholesterol levels as much as predicted from its total saturated fat content.
Monounsaturated Fatty Acids	Reduce Selectively	Reduce high saturated fatty acid sources of monounsaturated fatty acids: animal fats.
Polyunsaturated Fatty Acids	Do Not Increase Consumption	Polyunsaturated fatty acids have no distinct advantages over monounsaturated fatty acids.
Trans Fatty Acids	Reduce	Use tub margarine instead of stick margarine; reduce portion size so that margarine is a condiment and not a major calorie source. Make desserts at home using modified recipes that call for oil instead of shortening or stick margarine. Choose low fat diary and lean red meat products.
Dietary Cholesterol	Limit to <300 mg Step-One <200 mg Step-Two	Egg yolks and some shellfish are rich in dietary cholesterol, but low in saturated fat; their use in a cholesterol-lowering diet should be moderated.
Fiber	No Specific Recommendations	Water soluble fiber found in some grains (notably oat bran, gur gum, psyllium) has a small cholesterol lowering effect. These sources may serve as alternatives to a traditional breakfast of bacon and eggs.
Alcohol	Moderate Intake	In a few patients, alcohol can increase total and LDL cholesterol levels. In all patients, alcohol is a source of calories without micro- or macro-nutrients.
Coffee	No Specific Recommendations	Boiling coffee beans release a fat that raises cholesterol levels; this fat is removed by paper coffee filters. Caffeine can be an appetite suppressant. The use of caffeinated coffee may aid weight loss.

TABLE 5

Practical Aspects of Factors which Alter
Triglyceride and HDL Cholesterol Levels

Factor	Dietary Goal	Nuances
Fat	Reduce if weight loss is needed Increase if patient is lean	Very-low-fat-diets (less than 25% of calories from fat) will lower HDL cholesterol levels. Reducing calories by decreasing fat intake and not replacing these calories should result in weight loss, and hence, in long term improvements in HDL cholesterol levels. Acute management of hypertriglyceridemia includes a very-low-fat diet to reduce exogenous contribution of triglycerides to serum triglycerides.
Omega 3 Fatty Acids	Consider as treatment option for hypertriglyceridemia	Can be an effective therapy for hypertriglyceridemia. Since high dose is required to see an effect, fish oil capsules can be used. The capsules should be carefully selected: some are derived from fish skin and have high concentrations of dietary cholesterol. In diabetes, fish oil treatment may worsen diabetic control.
Body Weight/ Caloric Balance	Reduce Weight Avoid Caloric excess Add more physical activity into daily activities	Excess body weight is the most common cause of low HDL levels. Even if weight loss cannot be achieved, regular exercise without weight loss will increase HDL levels.
Alcohol	If consumed, hold intake to moderate levels.	Dose dependent relationship to alcohol and HDL cholesterol levels suggests that excess quantities are needed to improve HDL; since excess quantities are associated with cognitive impairment, alcohol is not recommended as a means to reduce CHD risk. Alcohol intake is notoriously under-reported. Patients with hypertriglyceridemia should have lipids evaluated after a 3 week period of abstinence.

Table 6

Practical Aspects of Factors Which Alter Blood Pressure

Factor	Dietary Goal	Nuances
Sodium	Reduce	Patients with hypertension are more likely to be sodium sensitive; obese patients are also more likely to be sodium sensitive. Sodium is ubiquitous in the diet: canned foods, prepared meats are typically high in sodium. Consultation with a registered dietician will optimize patient education and compliance.
Alcohol	Reduce	The alcohol/blood pressure association appears linear.
Body Weight/ Caloric Balance	Reduce	For optimum success, weight reduction should include mild caloric restriction plus increased caloric expenditure. While exercise raises blood pressure acutely, patients who exercise regularly have lower resting blood pressure and pulse rates.

TABLE 7

DIETARY THERAPY FOR CHD PREVENTION

Nutrient	Recommended Daily Intake	
	Step-One Diet	Step-Two Diet
Total Calories	To achieve and maintain desirable weight	
Total Fatty Acids	Less than 30% of total calories	
Saturated	Less than 10% of total calories	Less than 7% of total calories
Polyunsaturated	Up to 10% of total calories	
Monounsaturated	10% to 15% of total calories	
Carbohydrates	50% to 60% of total calories	
Protein	10% to 20% of total calories	
Cholesterol	Less than 300 mg	Less than 200 mg
Sodium	1650-2400 mg	
Alcohol[a]	Less than 30 g	

a. COMPARISON OF ALCOHOL DOSE BY TYPE

TYPE	% ETHANOL	"ONE DRINK"	AMOUNT ETHANOL
Beer	3-5%	12 Oz.	13.6 gm.
Wine	12%	5 Oz.	13.6 gm.
Spirits	40%	1.5 Oz.	13.6 gm.

Table 8
Descriptive Labeling Terms Approved by the FDA: A Translation to Components Important in a CHD Prevention Diet.

Nutrient	Free	Low	Reduced/Less/Fewer	Other
Synonyms for terms	Synonyms for "Free": "Free of", "No", "Zero", "Without", "Trivial Source of", "Negligible Source of", "Dietary Insignificant Source of"	Synonyms for "Low": "Contains a Small Amount of", "Low Source of", "Low in"	Synonyms for "Reduced / Less / Fewer": Reduced in", "Lower", "Low".	
Total Calories	Less than 5 calories/serving	Less than 40 calories/serving	Reduced by at least 25%	
Total Fat	Less than 0.5 g/serving	3 g or less/serving and 30% or less calories from fat. Meal-Type Products: 3 g or less per 100 g. product.	Reduced by at least 25%	"___% Fat Free", "___% Lean", must meet requirements for "Low Fat"
Saturated Fat	Less than 0.5 g/serving, levels of trans fatty acids must be 1% or less of total fat.	1 g or less/serving and 15% or less of calories from saturated fatty acids. Meal-Type Products - 1 g or less per 100 g, and less than 10% of calories from saturated fat.	Reduced by at least 25%	
Cholesterol	Less than 2 mg/serving; saturated fat content must be 2 gm or less.	20 mg or less/serving; saturated fat content must be 2 gm or less per serving. Meal -Type Products 20 mg. or less per 100 g, with saturated fat content less than 2 gms/100 gms.	Reduced by at least 25% Contains 2 gm or less saturated fat per serving.	
Sodium	Less than 5 mg/serving	140 mg. or less/serving Meal-type Products: 140 mg. or less/ 100 g of food.	Reduced by at least 25%	"Very Low Sodium", "Very Low in Sodium": 35 mg or less/serving

TABLE 9

Practical Aspects of a CHD Healthy Diet

Clinical Quotes	Translation into Risk Factor Reduction	Translation into Specific Recommendations
Stay Lean	Reduces total, VLDL and LDL cholesterol levels. Reduces triglyceride levels. Increases HDL levels. Reduces blood pressure. Increases exercise tolerance.	Control portion sizes Avoid "empty" calories from excess alcohol. Avoid overconsumption of food. Even "healthy" foods, when consumed in excess, will result in weight gain.
Adopt a Healthy Diet	Reduces total, VLDL and LDL cholesterol levels. Reduces triglyceride levels. Increases HDL levels. Reduces blood pressure.	No more than 6 oz. lean meat/day 2-3 servings low fat dairy products 4-6 servings fruit, vegetables 8-10 servings grain products This serving distribution will not only control the macronutrient content of the diet (protein, carbohydrates, fat), but will also improve the micronutrient content of the diet (vitamins, minerals).
Exercise	Reduces resting blood pressure Increases HDL cholesterol levels Increases exercise tolerance	Regular program of exercise, 30 minutes 2-3 times/week. Take advantage of opportunities: Stairs Walk the dog Park further from the store

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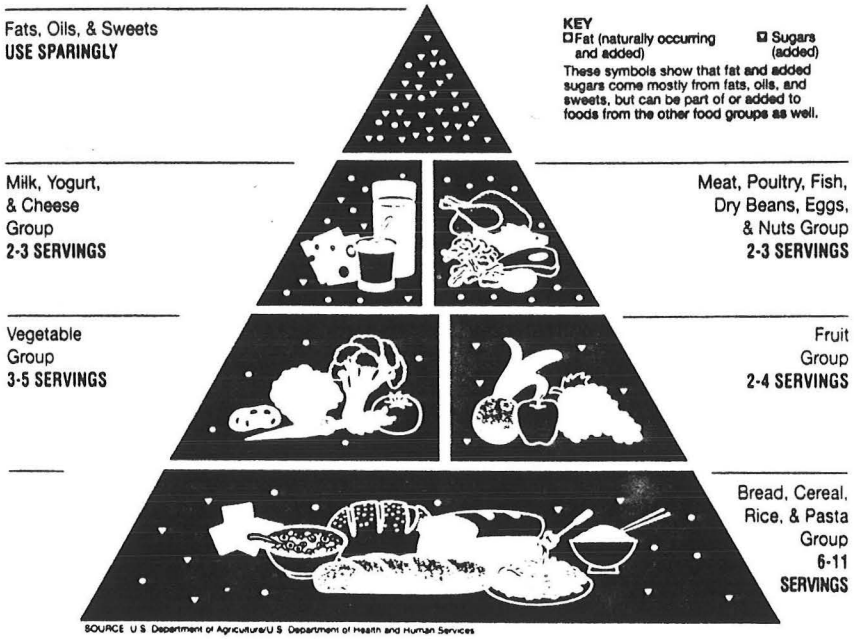
FIGURE 1

Food Guide Pyramid

A Guide to Daily Food Choices



How to Use The Daily Food Guide

What counts as one serving?

Breads, Cereals, Rice, and Pasta
1 slice of bread
1/2 cup of cooked rice or pasta
1/2 cup of cooked cereal
1 ounce of ready-to-eat cereal

Vegetables
1/2 cup of chopped raw or cooked vegetables
1 cup of leafy raw vegetables

Fruits
1 piece of fruit or melon wedge
3/4 cup of juice
1/2 cup of canned fruit
1/4 cup of dried fruit

Milk, Yogurt, and Cheese
1 cup of milk or yogurt
1-1/2 to 2 ounces of cheese

Meat, Poultry, Fish, Dry Beans, Eggs, and Nuts
2-1/2 to 3 ounces of cooked lean meat, poultry, or fish
Count 1/2 cup of cooked beans, or 1 egg, or 2 tablespoons of peanut butter as 1 ounce of lean meat (about 1/3 serving)

Fats, Oils, and Sweets
LIMIT CALORIES FROM THESE especially if you need to lose weight

The amount you eat may be more than one serving. For example, a dinner portion of spaghetti would count as two or three servings of pasta.