THE EFFECT OF THREE LEVELS OF A SOY POLYSACCHARIDE FIBER ON PLASMA LIPIDS, FECAL FIBER, AND APPARENT FIBER DIGESTIBILITY

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INTRODUCTION

It has been found that populations which have elevated levels of plasma total cholesterol (TC) and low density lipoprotein-cholesterol (LDL-C) have an increased risk for developing atherosclerosis and a higher incidence of coronary heart disease (CHD) (9, 12, 13, 14). The risk for developing CHD with elevated levels of plasma total triglycerides (TG); however, has been less clear cut (9, 12, 13, 14). High density lipoprotein-cholesterol (HDL-C) levels were observed to be inversely related to CHD and appeared to protect against atherosclerosis (20, 23, 24). A number of dietary factors, such as saturated fat, animal protein, and cholesterol, were positively associated with plasma cholesterol levels while other dietary components such as plant protein, dietary fiber, and polyunsaturated fatty acids were negatively associated with plasma cholesterol levels (10).

Epidemiological evidence has indicated that those areas that have higher levels of dietary fiber intake tended to have lower levels of plasma TC and a lower incidence of CHD (15, 16). Experimentally, those foods that have contained high amounts of dietaty fiber in the form of water soluble fiber such as pectins, gums, some of the hemicelluloses, mucilages, algal polysaccharides, and the storage polysaccharides were found to decrease plasma TC (38, 40, 59, 60, 61). The results of feeding these dietary fiber sources; however, have been less consistent on plasma total TG, HDL-C, and LDL-C (38, 40, 59, 60, 61).

In addition to decreasing plasma TC, dietary fiber was found to

cause laxation of the bowel. Many sources of dietary fiber have been found to increase total fecal output including wheat bran, corn bran, soy hulls, oat bran, fruits and vegetables, cellulose, and bagasse (36, 37, 43, 51, 53, 70). It has also been observed that by increasing dietary fiber there was increased fecal fiber (34, 72, 73, 74). Even though dietary fiber was said to be indigestible in the small intestine (30), there was some digestion of all dietary fiber in the small and large intestine depending on the type and amount of dietary fiber in the diet (70, 71, 72, 73, 74).

Due to the limited and conflicting data on the metabolic effects of soy polysaccharide fiber, it was proposed to observe the effects of a soy polysaccharide fiber, added to a zero-cholesterol, liquid formula diet on the plasma lipids and lipoprotein cholesterol, fecal fiber, and fiber digestibility of 22 healthy young men.

REVIEW OF LITERATURE

I. Plasma Lipids, Lipoproteins, and Coronary Heart Disease

A. General Information on Coronary Heart Disease

In 1979, heart diseases, including Coronary Heart Disease (CHD), were the number one cause of death in the United States which accounted for 331.3 deaths per 100,000 persons (1). The exact cause of heart disease is unknown at this point; however, the toll of heart disease is known. Heart disease causes pain, activity restriction, economic burden, and disruption of families by early death (2). In addition, the patient with heart disease has feelings of anxiety, depression, fear, hostility, denial, and lacks confidence and motivation (3).

There is one positive note when looking at heart disease. The number of deaths per 100,000 persons has been declining since 1960. In 1960, 369.0 persons per 100,000 died from heart disease. The number declined to 362.0 per 100,000 in 1970 and to 331.3 per 100,000 in 1979 (1).

B. Pathogenesis of CHD

One factor that appears to lead to heart attacks and strokes is hardening of the arteries or atherosclerosis. Atherosclerosis comes from the Greek word, athere, which means "soft, fatty, gruel-like". It is given this name due to the accumulation of lipid in the artery(4). This is one of the greatest health hazards for middle-aged males in the United States since approximately 25% of this population develops atherosclerosis (5).

There are two basic theories on the origin of atherosclerosis.

The first theory, associated with Rudolf Verchow, states that fatty substances in the blood give rise to the deposition of lipid in the artery wall which acts as an irritant to cause cell proliferation. The second theory, called the encrustation theory, is based on the belief that a plaque begins as a small blood clot attached to the arterial wall which is converted into a mass of tissue in the intima layer. Cells migrate to the clot and secrete substances thought to be related to the repair process. This allows substances, such as lipids, to pass from the plasma into the artery wall (6).

The artery wall has been found to contain both fatty streaks and atherosclerotic placques (6). Fatty streaks have been found in children of all populations and are characterized as fatty-filled smooth muscle cells that appear as small white and yellow dots in the aortas (6, 7). It has been postulated that these fatty streaks are the beginning of atherosclerotic plaques (4, 8); however, recent evidence has shown that fatty streaks tend to develop at the same time as atherosclerotic plaques which does not indicate that fatty streaks are precursors to atherosclerotic plaques (7). The relationship is not clear at this time.

It is not exactly clear how an atherosclerotic placque originates but it appears that initially there is an injury of some sort to the artery wall which causes proliferation of smooth muscle cells (4, 6, 7, 8, 9). The stimulation of cell proliferation may be caused by hyperlipidemia (specifically low density lipoprotein cholesterol or LDL-C), platelets, diabetic serum, hypertensive serum, lysosomal products or possibly a mutagenic substance (for example, a factor in

cigarettes) (7, 9). Once the smooth muscle cells begin to proliferate, several other things begin to occur, probably at the same time. The plaque begins active collagen synthesis (7, 8) and also in the presence of high levels of LDL-C, the plaque tends to take up more esterified cholesterol, which is the major lipid component of plaques (9). Following the uptake of lipid, in more advanced plaques, there appears to be an increase in the amount of fibrin in the plaque (7). The advanced atherosclerotic plaque may then become calcified, degenerate, and possibly ulcerate (6, 7).

It has been theorized that formation of an atherosclerotic plaque causes the arterial wall to thicken. If the arterial wall has thickened due to atherosclerosis, this puts arterial tissue in a hypoxic state which increases its uptake of extracellular lipids, therefore, increasing the thickness of the arterial wall even more. This leads to a continuing cycle. Also, this decrease in available oxygen to the cells may further increase the chance of injury to the cell (8).

Occlusion of an artery can result from thrombosis at the site of a plaque. A thrombus is a complex aggregation of blood platelets, white cells, and red cells in a network of fibrin. Special conditions are required for a thrombus to form. Examples of these special conditions are the slowing of the flow of blood, a region of turbulence, or an injury which would cause platelets to stick in that region (6). When the artery becomes occluded, blood cannot pass through the artery leading to a myocardial infarction in the case of a coronary artery (5).

C. Plasma Lipids and CHD

The incidence of CHD is related to the prevalence of atherosclerosis

(4). Atherosclerosis appears in the presence of elevated plasma total cholesterol (TC) and plasma total triglycerides (TG) (10, 11). With increased levels of TC, there seems to be a clear increased risk for the development of CHD (9, 12, 13, 14). The role of TG as a risk factor in developing CHD is less certain (9, 12, 13, 14).

In looking at populations with a low incidence of CHD, they have been found to have very low levels of plasma lipids. Trowell (15) mentioned that the incidence of CHD among African tribesmen was very low as were their serum TC levels. Connor et al. (16) reported on the Tarahumara Indians, a primitive Indian population, with relatively few deaths from cardiac problems and circulatory problems. The average plasma TC for the total population was 125 mg/dl; the average plasma TC for children was 116 mg/dl and was 136 mg/dl for adults. The average plasma total TG for the total was 120 mg/dl. Children had an average plasma TG of 115 mg/dl and adults had an average plasma TG of 126 mg/dl.

Other studies have observed TC and TG levels of populations with a history of CHD and compared these with the TC and TG levels of a population without CHD (14, 17). Seftel et al. (17) looked at the plasma TC and TG levels of 9 obese and 11 non-obese Bantus that had had myocardial infarctions (MI) and compared these with 36 obese and 19 non-obese controls. The obese MI patients had plasma TC that were a mean of 18 mg/dl (not significant) higher than the obese controls and the non-obese MI patients had plasma TC that were a mean of 42 mg/dl ($P<.0025$) higher than the non-obese controls. The TG levels for the obese MI patients were higher than those for the obese controls by a

mean value of 13.0 mg/dl (not significant) and the non-obese MI patients had TG levels which were 83.0 mg/dl ($P<.025$) higher than the non-obese controls.

Lewis et al. (14) compared the TC and TG levels of 143 persons with CHD with the TC and TG values of a control London population. The TC and TG levels of the population with CHD were significantly ($P<.001$) greater than those of the control population. When compared with their respective control groups, TC values for those with CHD were a mean of 50 mg/dl higher for men aged 26 to 39 years, 33 mg/dl higher in men aged 40 to 73 years, and 35 mg/dl for women aged 44 to 73 years. The TG levels for those with CHD, when compared with the control groups, were 0.89 mmol/l higher in men 26 to 39 years, 0.53 mmol/l higher in men 40 to 73 years, and 0.59 mmol/l higher for women 44 to 73 years. The men in the younger aged group had a higher incidence of hypercholesterolemia and hypertriglyceridemia than the older men.

Several prospective studies have taken a fairly large population group and observed the correlation between plasma TC and TG and CHD. Kannel et al. (18) in 1961 reported on the results at the fourth biennial examination of the relation between TC and CHD in a sample population, 5209 persons initially 39 to 59 years, in Framingham, Massachusetts. It was found that the subjects with CHD had higher levels of TC than those without CHD. There was not a particular threshold in TC levels above which there was an increased risk for CHD, but it was found that generally with increasing TC levels there was an increased risk for the development of CHD. The researchers noted that

hypercholesterolemia was a greater risk factor in younger persons than older persons.

Gordon et al. (19) in 1977 reported on data from biennial examinations of a sample population of 5209 persons 49 to 82 years from the Framingham study and found at the ages of this population, TC was not indicated as a risk factor associated with CHD. TG levels were found to be a risk factor only in females when the level of other lipids was not accounted for.

Kannel et al. (13) in a review of the Framingham data noted that the risk of TC in developing CHD was dependent on the presence of other risk factors one of which was how the cholesterol was divided among the lipoprotein fractions. This will be addressed further in Section ID.

Castelli et al. (20) reported on the data of 6859 persons from several study populations: male Civil Service employees in Albany, New York; a general population of black and white men and women in Evans County, Georgia; a general population in Framingham, Massachusetts; and general populations of Japanese men living in Honolulu and San Francisco. Those individuals with CHD in these populations had higher mean levels of TC (by 6.82 mg/dl; $P<.01$) and TG (by 21.0 mg/dl; $P<.01$) when compared with populations without CHD. There was a direct relationship between the fasting plasma TG level and the incidence of CHD but it may only be a risk factor in the presence of elevated cholesterol levels.

Shekelee et al. (21) reported in 1981 on a 20 year follow-up to the Western Electric Study in which dietary data and TC were compared with the vital statistics of 2104 men. The researchers found that TC was significantly ($P<.001$) correlated to the risk of death from CHD.

A study from Stockholm reported by Carlson et al. (11) followed a health center population in which 3486 men and 2738 women had plasma TC and TG evaluated as possible risk factors for developing a MI. Researchers found that those individuals that developed a MI had higher mean TC and TG levels. The rate of new MI increased with increasing concentrations of both plasma TG and TC as revealed by quintile statistics. Multiple logistic regression analysis revealed that TG level was an independent risk factor for developing MI whereas TC was not. Researchers found that those individuals with high levels of TG and TC had the highest incidence of MI, followed by those with elevated TG levels but normal TC levels. Those with elevated TC levels but normal TG levels had the third highest incidence of MI while individuals with normal levels of TC and TG had the lowest incidence of MI.

Rosenman et al. (12) reported on the results of a 8.5 year follow-up of the Western Collaborative Group Study in which the TC and TG values of 3154 men 39 to 59 years were compared with the incidence of CHD. TC levels were a significant ($P<.01$) predictor for developing CHD but TG levels showed a negligible association with the incidence of CHD.

In summary, the levels of plasma TC appeared to be a strong risk factor in the development of CHD (12, 17), but not always (19). This risk appeared to be higher when elevated levels of TC were found in younger persons (18). The risk for developing CHD with elevated TC levels appeared to also be related to the presence of other risk factors,one of which was the way TC was divided among the lipoprotein fractions (13). Some studies found that elevated levels of TG were an

independent risk factor for the development of CHD (11) while other studies found that the risk of elevated TG values were not an independent risk factor (20) or had negligible value in predicting CHD (12). When different studies reported conflicting results about the use of TC and TG as a risk factor for development of CHD, this may be attributed to ethnic differences, geographic differences, environmental differences and the use of different criteria for the endpoint definition of CHD (22).

D. Plasma Lipoproteins and CHD

Early experiments in determining the risk for development of CHD centered on the role of TC; however, recent evidence has shown that the strength of this risk factor depends on a number of other factors such as how TC is distributed among the various lipoprotein fractions (13). Two lipoprotein fractions which are of the most interest are the high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C). (The description of these two lipoprotein fractions and their properties will be discussed in Section II). Evidence has shown that high levels of HDL-C (20, 23, 24) are inversely related to CHD whereas high levels of LDL-C are directly related to the risk for CHD (20, 23).

Several research studies have shown that populations that have a low incidence of CHD usually have higher levels of HDL-C (14, 19, 20). Lewis et al. (14) in 1974 reported on the lipoprotein fractions of a population of 143 persons that suffered from CHD and compared these values with those of an apparently healthy London population matched for sex and age. HDL-C levels of the younger population men, 26 to

39 years suffering from CHD were 79% ($P<.01$) of the HDL-C values of the healthy population. The HDL-C levels in the older male subjects (40 to 73 years) and female subjects (44 to 73 years) with CHD were 95% and 97%, respectively of the HDL-C levels of the healthy population, but this difference was not significant.

The study presented by Gordon et al. (19) looked at the data of 5209 men and women from Framingham, Massachusetts that were enlisted in 1949 in a longitudinal study using biennial examinations and correlating these results to the vital statistics for CHD. The researchers found that persons with low HDL-C levels were at high risk for developing CHD. They also noted that persons with HDL-C less than 35 mg/dl were more than eight times as likely to develop CHD as those with HDL-C greater than 65 mg/dl. They found that women had much higher HDL-C than men with a lower incidence of CHD.

Castelli et al. (20) looked at the lipid values of several study populations with a total of 6859 persons. Lipid values of those individuals suffering from CHD were compared with those that were apparently healthy. The researchers found that HDL-C was about 10 mg/dl higher in women than in men and that the average levels of HDL-C in blacks were about 10 mg/dl higher than those of white subjects. In looking at the data across all populations and age groups, researchers consistently found that average HDL-C levels were significantly ($P<.01$) lower by a mean of 3.81 mg/dl in persons with CHD compared to the healthy persons.

HDL-C is thought to protect against atherosclerosis by helping to remove cholesterol from tissues (25). Whenever excess cholesterol

results from the catabolism of chylomicrons and VLDL-C or turnover of cholesterol from extrahepatic cells or scavenger cells, it can be esterified by lecithin cholesterol acyl transferase or remain bound to HDL-C and return to the liver for conversion to bile acids before elimination from the body (26).

It has been shown in the literature, that along with elevated TC levels, elevated levels of LDL-C are related to CHD (14, 19, 20). This observation has come by studying populations that have CHD and comparing their LDL-C levels with those of populations that appeared healthy.

Lewis et al. (14) compared the serum lipoproteins of 143 patients with CHD with those of an apparently healthy London population. The patients with CHD had TC levels significantly ($P<.001$) greater than the healthy population. The researchers reported that the level of LDL-C was significantly correlated ($P<.001$) with the TC levels and that LDL-C levels were significantly ($P<.01$) increased (120%) in the younger patients, 26 to 39 years, in those with CHD over the healthy population. The LDL-C levels were elevated 110% ($P<.05$) in those with CHD over the healthy population in men 40 to 73 years and women 44 to 73 years. The authors said that the elevated LDL-C levels reflect elevated TC levels.

In 1977, Gordon et al. (19) reported on a follow-up to the Framingham study in which it was found that LDL-C was a marginal risk factor for developing CHD in this population (50 years or more).

Castelli et al. (20) reported on the results of serum lipoproteins and the incidence of CHD in several study populations. Re-

searchers found a significant ($P<.01$) increase in the average LDL-C of 6 mg/dl in those subjects with CHD as compared to the healthy population.

The reason why LDL-C appears to be so harmful can be shown in tissue cultures when tissues are incubated with LDL-C (9). In looking at plaque formation, it is thought that when the artery wall is exposed to LDL-C, it tends to take up more esterified cholesterol which is a large component of both LDL-C and atherosclerotic plaques. The amount of cholesteryl ester which accumulates in the atherosclerotic plaque is proportional to the amount of LDL-C present (9). This would cause a proliferation of smooth muscle cells and thickening of the artery wall increasing chances of CHD (9).

Studies of populations containing individuals both with and without CHD have shown that HDL-C levels are higher in those individuals that do not have CHD (20, 23, 24). LDL-C levels are higher in individuals with CHD with a stronger relation to CHD in younger populations (20, 23).

II. Plasma Lipoproteins

A. General Information on Plasma Lipoproteins.

Plasma lipids, triglycerides, and cholesteryl esters, are insoluble in the blood and are transported in the blood attached to apoproteins. Together with phospholipids, triglycerides, cholesteryl esters, and apoproteins are called lipoproteins. This form of lipid is soluble in the blood and can easily be transported throughout the body. There are four classes of lipoproteins in human plasma called chylomicrons, very low density lipoprotein cholesterol (VLDL-C),

LDL-C, and HDL-C (26, 27).

B. Chylomicrons

Chylomicrons, largest of the lipoproteins at 800 to 1200 A in diameter, have a molecular weight of 1 X 10^9 (27). Chylomicrons are the lowest density (d 1.006 g/ml) of all lipoproteins (27, 28). Chylomicrons are found in an electrophoretic gel at the origin (27).

The primary constituent of chylomicrons is lipid, with the primary lipid being triglyceride (85%) and containing smaller amounts of phospholipid (8%) and cholesterol (4%). Chylomicrons have very little protein (2.5%) which contains the apoproteins: apo AI, apo B, and apo C (27).

Chylomicrons originate from dietary fat and cholesterol in the intestine where they are incorporated into lipoproteins. Chylomicrons are then absorbed into the lymph system and go to the bloodstream. Alone they are too large to cross the endothelium and so must be metabolized in the blood stream by an enzyme in the endothelium called lipoprotein lipase. The lipase is activated by the presence of apo C in chylomicrons causing release of fatty acids into muscles or adipose tissue and the release of monoglycerides. The release of TG causes the chylomicron to shrink transferring excess surface material in the form of phospholipid and free cholesterol to HDL-C. The remaining particle is called a chylomicron remnant which goes to the liver where it is internalized and degraded (26, 27).

C. VLDL-Cholesterol

VLDL-C is also a large particle with a diameter of 250 to 800 A and a molecular weight of 1 X 10^7 (28). The density of VLDL-C is

d = 1.006 to 1.019 g/ml (29). VLDL-C is found at the pre B position of an electrophoretic gel (28).

VLDL-C is also composed primarily of lipid containing 50 to 55% TG, 18% phospholipid, and 15% cholesterol. VLDL-C contains 10% protein in the form of the following apoproteins: apo B, apo C-I, apo C-II, apo C-III, and apo E (27, 28).

VLDL-C results from lipid transport from the liver and small intestine with a catabolism similar to that of chylomicrons. Upon contact with lipoprotein lipase in the capillary beds, VLDL-C releases TG causing the size to decrease and the density to increase forming an intermediate density lipoprotein (IDL) particle. Excess surface material, cholesterol and phospholipid, are passed to HDL-C which causes HDL-C to react with lecithin cholesterol acyl transferase (LCAT). LCAT esterifies excess cholesterol and transfers the cholesteryl ester to the IDL to form LDL-C. In the process, most of the remaining TG is lost and all apoproteins except apo B are lost (26, 27).

D. LDL-Cholesterol

LDL-C is the next smaller lipoprotein with a diameter of 175 to 250 A, a molecular weight of 2.3×10^6, and a density of d= 1.019 to 1.063 g/ml (28, 29). LDL-C is found in the B position on an electrophoretic gel (28).

LDL-C is composed primarily of lipid but the types of lipid are different from those found in chylomicrons and VLDL-C. The primary lipid found in LDL-C is cholesterol (30%), followed by phospholipid (22%), and then TG (12%). LDL-C contains more protein (20-25%) than do

chylomicrons and VLDL-C which increases its density. The only major apoprotein in LDL-C is apoprotein B (27, 28).

LDL-C is formed by catabolism of VLDL-C as indicated in the previous section. LDL-C carries cholesterol to tissues by binding to high affinity receptors located in a region of the plasma membranes called the coated pits which invaginate into the cell and pinch off to form endocytotic vesicles and carry LDL-C to the lysosomes. LDL-C is fused with the lysosomal membrane which exposes LDL-C to enzymes that degrade apoprotein B to amino acids. The cholesteryl ester is hydrolyzed by an acid lipase which releases cholesterol for use in cellular reactions. This decreases the cellular rate of cholesterol synthesis. A second way for LDL-C removal is by use of scavenger cells or macrophages. When plasma LDL-C is high, scavengers degrade increasing amounts of LDL-C. When scavenger cells become overloaded with cholesteryl esters, they are converted to foam cells which are components of atherosclerotic plaque (26, 27).

A disease state exists in which the specific binding sites for LDL-C are absent or defective in individuals with homozygous familial hypercholesterolemia (27).

E. HDL-Cholesterol

HDL-C is the smallest of the lipoproteins and has the highest density with a diameter of 70 to 150 A, a molecular weight of 1.7×10^5 to 3.6×10^5, and a density of 1.063to 1.21 g/ml (28, 29). HDL-C is found at the α position in an electrophoretic gel (29).

HDL-C contains more protein and considerably less lipid than other

lipoproteins. The lipids in HDL-C are phospholipid (25%), cholesterol (15%), and TG (6%) HDL-C is 50% protein and contains the apoproteins apo A-I and apo A-II (27, 28).

HDL-C originates in the liver and the intestine. HDL-C takes up and exchanges excess cholesterol and phospholipid from catabolism of chylomicrons and VLDL-C. In addition, when a cell takes up cholesterol from LDL-C, an amount equal to the amount of LDL-C taken in is excreted and thought to be absorbed onto HDL-C where it is esterified by plasma LCAT. Resulting cholesteryl esters are transferred to VLDL-C and IDL to form LDL-C (26).

The ability of HDL-C to absorb or bind cholesterol that is excreted from cells may be an important mechanism by which the body rids itself of excess cholesterol. This cholesterol that is bound to HDL-C may be taken to the liver for conversion to bile acids and then excreted from the body (24, 25).

III. Effect of Dietary Fibers on Plasma Lipids and Lipoproteins

A. General Information on Dietary Fiber

Fiber is defined as that part of the plant which is resistant to digestive enzymes of the small intestine (30). Fiber has been generally measured by two methods. Originally crude fiber analysis, by use of strong acids and bases, was used to determine the amount of lignin and cellulose. This method destroyed a portion of the cellulose and lignin and all of the hemicellulose, thus measuring only a portion of the fiber. A method that has been widely used recently to measure the amount of total dietary fiber was developed by Goering and Van Soest (31). This method measures all cellulose, hemicellulose, and lignin

with the use of detergents giving a more realistic measure of the total fiber present in the plant (31).

Certain dietary fibers have been found to be hypocholesterolemic in man. Purified dietary fibers can be divided into two classifications: those fibers which are water insoluble and those which are water soluble (30). Water soluble fibers include a portion of the hemicelluloses, storage polysaccharides, mucilages, algal polysaccharides, pectins, and gums while water insoluble fibers include lignin, cellulose, and a portion of hemicellulose (30). Studies have found that water soluble fibers of foods tend to be more hypocholesterolemic than water insoluble fibers (30). Since certain dietary fibers tend to be hypocholesterolemic, this may be one way of decreasing plasma cholesterol levels in certain populations and hopefully then the incidence of CHD.

B. Proposed Mechanism for Action of Dietary Fiber

The hypocholesterolemic action of dietary fiber is theorized to occur when dietary fiber binds to bile acids. Dietary fibers would increase bile acid excretion, thus increasing cholesterol degradation and excretion. If there was not as much of a concombinant increase in cholesterol synthesis, a decrease in TC would result. The bile salts would also be unavailable for micellular formation and would therefore, decrease cholesterol absorption (32).

An indirect effect of consuming a diet high in dietary fiber on plasma lipids was noted by Trowell (15). A diet high in dietary fiber generally results in consumption of foods that are low in fat

and cholesterol and a decreased intake of fat and cholesterol also decreases plasma cholesterol levels and, therefore, the risk of developing CHD. (The effect of dietary cholesterol on plasma cholesterol levels will be further discussed in Section IV).

C. Epidemiological Evidence for Hypocholesterolemic Action of Dietary Fiber

Populations consuming diets with differing amounts of dietary fiber have been observed to see the effect that dietary fiber has on plasma lipid levels. Those populations in Western nations have been found to consume diets containing less starchy carbohydrate, more refined carbohydrate (sugar), and little crude fiber. Diets in populations consuming non-Western diets are less refined and have been found to provide as much as four times the crude fiber as refined Western diets (15).

Trowell (15) reported that diets of rural Africans contain starchy foods and complex carbohydrates or lightly processed cereals. Rural South African Bantu tribesmen have been estimated to consume approximately 24.8 grams of crude fiber per day and have very low levels of serum TC as well as a low incidence of CHD. When these tribesmen move to urban areas and adopt Western diets and patterns of living, the incidence of CHD increases.

Groen et al. (33) looked at the diets and serum TC levels of monks from two different orders, Trappists and Benedictine, both in Holland and Belgium. These monastaries have two different dietary philosophies. Benedictine monks consumed an average Western diet while Trappist monks were strict vegetarians consuming a diet

high in fiber rich foods (wholemeal bread, potatoes, oatmeal, vegetables, and fruits). Serum TC levels measured in both orders for monks of all ages showed that monks of the Trappist order had significantly lower TC levels than those in the Benedictine order.

Connor et al. (16) reported on the diets and plasma TC levels of Tarahumara Indians in Mexico. He found that their diet consisted mostly of corn and beans which contributed to 90% of their total kilocalories. The rest of their diet consisted almost completely of other vegetables and they rarely consumed animal products. Their diet was very low in saturated fat (2% of total kilocalories) with a majority of the fat being unsaturated fat (4 to 5% of total kilocalories). Their estimated crude fiber intake was 15 to 21 grams per day. This population had a low incidence of CHD with an average TC for children 116 mg/dl and 136 mg/dl for adults. Average TG values were 115 mg/dl for children and 126 mg/dl for adults. The average LDL-C value in the population was 87 mg/dl and the average HDL-C value was 25 mg/dl. In addition to a low fat diet, high P/S ratio, and low dietary cholesterol, the high amount of dietary fiber may be one reason for low plasma lipid levels.

In summary, populations consuming non-Western diets are generally found to have large amounts of dietary fiber, contain little fat, consist primarily of vegetable protein, and have a high P/S ratio (16, 20, 33). Populations consuming this type of diet usually have lower levels of TC and a lower incidence of CHD (16).

D. Wheat Bran

Results from research on addition of wheat bran to diets has

been inconsistent overall, but with some exceptions, wheat bran has generally been shown to be ineffective on TC levels.

Farrell et al. (34), van Berge-Henegouwen et. (35) and Munoz et al (36) all found the addition of wheat bran achieved a decrease in TC. Farrell et al. (34) observed a significant ($P<.01$) decline from approximately 1.7 g/l to approximately 1.32 g/l when 12 g of unprocessed bran were added to the diets of 14 male subjects. Plasma TG remained unchanged in going from the low to high fiber diet. Van Berge-Henegouwen et al. (35) observed a decline of approximately 10.1% in TC of his 7 healthy male volunteers by addition of 33.5 to 37.8 g per day of raw wheat bran to their diets. HDL-C was significantly decreased by 0.15 mmol/l at week 2 ($P<.005$) and by 0.20 mmol/l at week 4. All other lipoproteins decreased but this was not significant. Total TG had decreased significantly ($P<.01$) by 0.27 mmol/l at week 4. Munoz et al. (36) found that addition of 26 g per day of Hard Red Spring wheat bran to the diet in the form of bread significantly ($P<.05$) reduced plasma TC from 168 mg/dl to 149 mg/dl. LDL-C also significantly ($P<.05$) decreased from 106 mg/dl to 83 mg/dl. However, when feeding subjects 26 g a day of soft white wheat bran, researchers found there was no effect on plasma TC, TG, or lipoprotein fractions.

Eastwood et al. (37) and Jenkins et al. (38) found no change when feeding wheat bran to subjects. Eastwood et al. (37) supplemented a control diet for 3 weeks with 8 g of wheat bran daily and found no change in serum TC levels by addition of wheat bran to the diet (186 mg/dl versus 189 mg/dl). Jenkins et al. (38) fed 36 grams of

wheat bran per day to five healthy men and found that addition of wheat bran to the diets caused a slight increase in serum TC (not significant) from 224 mg/dl by 7 mg/dl. Watts et al. (39) also found that by adding 30 g of wheat bran to the normal diets of 11 male and female subjects, there was no effect on the average serum TC (4.8 mmol/l without bran versus 5.2 mmmol/l with bran) and serum TC levels (0.7 mmol/l without bran versus 0.9 mmol/l with bran).

In contrast to the above studies, Stasse-Wolthius et al. (40) found that addition of 37 g of wheat bran to diets in breads and desserts caused plasma TC to significantly ($P<.01$) increase by 0.34 mmol/l at 5 weeks.

Inconsistent findings of the effect of wheat bran on serum lipids may result from several possibilities. First, the use of raw or unprocessed bran in the studies by Farrell et al. (34) and Van Berge-Henegouwen et al. (35) may be the reason for the decrease in lipids in these studies. Secondly, in the study by Munoz et al. (36), the type of fiber used (hard red spring wheat bran versus soft white wheat bran) seemed to be a factor leading to the decrease in lipids. Two other possible explanations for inconsistent results are the small number of subjects (35, 36) in studies and the varying level of wheat bran (36, 40) added to the diets.

E. Oat Bran

Several studies have shown the effects of the addition of oat bran and rolled oats to the diets of normocholesterolemic and hypercholesterolemic men (41, 42, 43). DeGroot et al. (41) fed 21 healthy male volunteers, aged 30 to 50 years, 300 g of

an experimental bread containing 140 g of rolled oats for three weeks. Subjects were fed the experimental bread instead of their usual bread in their diet. Blood samples were taken weekly starting three weeks before the experimental diet and up to three weeks after the experimental diet. Substitution of the experimental bread caused a significant (P value not given) decrease in serum TC from 251 mg/dl to 239 mg/dl after seven days. Serum TC continued to decrease over the three week period until it reached 223 mg/dl after three weeks. When subjects returned to normal diets, serum TC levels increased to an average value of 246 mg/dl. The researchers speculated that this may partially be due to the polyunsaturated fat content of the rolled oats.

A study by Judd and Trusswell (42) observed the effects of an average intake of 125 g per day of rolled oats on the plasma lipids and lipoproteins of 10 healthy persons. On the average, the plasma TC for the group fell by 8% (not significant) over seven weeks. Plasma HDL-C levels and plasma TG were unchanged during the seven week experiment.

Kirby et al. (43) in 1981 reported on the effects of the addition of 100 g of oat bran per day on the lipids of eight hypercholesterolemic men. Men were placed on either the control or experimental diet in an alternating sequence with diets differing only by inclusion of 100 g of oat bran in the experimental diet. The men were fed the oat bran for at least 10 days. Addition of 100 g of oat bran significantly ($P<.01$) decreased serum TC to 87% of the initial value from 269 mg/dl to 234 mg/dl. LDL-C decreased

by 14% ($P<.05$) from 184 mg/dl to 159 mg/dl. HDL-C was not altered by the addition of oat bran to the diet. Serum TG decreased by 5% from 161 mg/dl to 146 mg/dl (not significant) when oat bran was added to the diet.

The use of oat bran or rolled oats in diets of normal and hypercholesterolemic men appears to have decreased serum lipids in men (41, 42, 43). This hypocholesterolemic action of rolled oats or oat bran may be due to the polyunsaturated fat content (41) or the high concentration of water-soluble fiber in oats (43). Oat bran contains approximately 14.8% water soluble fiber and 11% water-insoluble fiber (43).

F. Soy Fiber

Several studies have observed the results of added soy hulls to the diets of healthy and hypercholesterolemic on their plasma lipids and lipoproteins. Raymond et al. (44) reported in 1977 on the results of addition of 60 g of dietary fiber to eucaloric formula diets of eight healthy adults. The fiber, a mixture of fiber from corn, soy hulls, beans, bran, pectin, and cellulose, was added to the diets in the form of muffins. Subjects were placed on diets for four weeks that were cholesterol free or had 1000 mg of added cholesterol and with or without added fiber. When subjects were placed on the cholesterol free formula, there was a 10 to 31% decrease in plasma TC when compared with baseline values. The addition of fiber to the cholesterol free diet did not further decrease plasma TC (171 mg/dl versus 167 mg/dl). No changes were seen in TG or LDL-C and HDL-C fractions by addition of fiber

to the cholesterol free diet. Addition of 1000 mg of cholesterol to the diet increased plasma TC by 24% ($P<.025$) and LDL-C by 26% ($P<.05$). Addition of fiber to this diet did not change the plasma lipids or lipoproteins consistently.

Palumbo et al. (45) reported on the effects of addition of an unspecified amount of soy hulls to the diets of 14 hyperlipidemic outpatients for 6 months. Eight of these subjects participated in a crossover design experiment for one year in which half received 16 g of cholestyramine and half received the high fiber diet first. The other six subjects were placed on the high fiber diet only for six months. The subjects placed initially on the high fiber diet had a decrease in serum TC levels by 5% (not significant) after 12 weeks. After 14 weeks more, serum TC levels showed a slight increase toward baseline. Mean serum TG increased by 9% (not significant) for subjects on the high fiber diet for the first six months. Four patients on the high fiber diet following the cholestyramine treatment had an increase in serum TC above that level achieved by cholestyramine, but TC levels did not reach the original baseline values. Very little change was seen in serum TG levels. During each cholestyramine treatment serum TC significantly ($P<.05$) decreased by 21% but serum TG increased by 12%.

Munoz et al. (36) used soy hulls and textured vegetable protein as fiber sources and observed their effect on plasma TC, LDL-C, HDL-C, TG, and HDL-C/plasma TC ratios. Five subjects were placed on soy hulls whereas three subjects were placed on textured vegetable protein. All diets had 26 g of fiber added daily for 30 days.

There was a significant ($P<.05$) decrease in plasma TC with addition of soy hulls, dropping from a baseline value of 172 mg/dl to 148 mg/dl at the end of the study. No significant changes were observed in other plasma lipids or lipoproteins. Textured vegetable protein had no effect on any of the plasma lipids or lipoproteins.

Addition of soy fiber, in the form of soy hulls and textured vegetable protein, to human diets has shown inconsistent results. Differences in results may possibly be due to different amounts of soy hulls that were added in these experiments. The results seen in the experiments by Raymond et al. (44) and Palumbo et al. (45) may have masked the effects of the soy hulls because the soy hulls were mixed with other dietary fibers.

G. Legumes

Addition of legumes such as beans, peas, and Bengal gram to diets and their effect on cholesterol levels in men have been observed. Grande et al. (46) placed 25 men on a standard diet for three weeks. Subjects were assigned to experimental diets in a switch back pattern. The subjects received either a diet containing 123 g of sucrose and 45 g of soybean protein or 115 g of carbohydrate and 48 g of protein from a mixture of equal parts of beans, lima beans, and split peas. The men that had been placed on the mixed bean diet had an average serum TC (202 mg/dl) significantly ($P<.0015$) lower than those receiving sucrose and soybean protein diet (221 mg/dl) regardless of when they were placed on these diets.

Mathur et al. (47) observed the effects of added Bengal gram on lipid levels of 30 healthy male subjects aged 15 to 50 years. Subjects were placed on a low-fat diet for two weeks and then fed a high-fat diet for ten weeks. After ten weeks on the high-fat diet, Bengal gram was substituted for wheat flour and other cereals so that they were on a high-fat, Bengal gram diet for 55 weeks. Blood samples were taken weekly and showed a significant ($P<.001$) decrease at week 55 in serum TC when Bengal gram was added to the high-fat diet from 206 mg/dl to 160 mg/dl. Analysis revealed that serum TC decreased at the end of the fourth week of treatment and progressively decreased to the 20th week after addition of the Bengal gram to the diet.

Addition of legumes, peas, beans, and Bengal gram, significantly decreased TC levels in healthy men (46, 47).

H. Fruits and Vegetables

The effect of adding fruits and vegetables in increasing amounts to the diets on serum TC levels of subjects have been studied. Robertson et al. (48) added 200 g of raw carrot to the diets of five subjects aged 25 to 41 years to observe the effect on serum cholesterol levels. The men were on a control diet for one week and then supplemented with 200 g of raw carrot per day for three weeks. Addition of the raw carrot caused a significant ($P<.05$) decrease in serum TC from 6.6 mmol/l to 5.9 mmol/l. Carrot fiber did not have any effect on serum TG levels. After removing the raw carrot from the control diet, serum TC and serum

TG levels remained unchanged from the experimental diet which contained raw carrot.

Gormley et al. (49) observed the effects of addition of two apples a day to the diets of 38 pairs of men. Seventy-six men volunteered for the study and based on a prelimary serum TC value and dietary fiber intake (determined by their weekly intake of fruits, vegetables, cereals, and cereal products) were paired with one another. One group was instructed to eat no more than three apples per week or their fruit equivalent while the other group was to add two apples to their normal daily food consumption. Subjects were asked to record their daily fruit consumption. These diets were consumed from November 17, 1976 to March 10, 1977. Blood samples were taken on December 9, January 13, February 17, March 10 and at a three week follow-up on March 31. Both groups had an increase in serum TC levels from the preliminary value to the February value which was attributed to consumption of a seasonal diet. The group consuming two extra apples daily always had lower serum TC levels during the experimental diet, but these were not significantly (P value not given) lower until February 17 and March 10. Values (mg/dl) at each sampling date were (apple versus non-apple): preliminary - 205 versus 204, December 9 - 223 versus 234, January 13 - 234 versus 235, February 17 - 231 versus 242, March 10 - 210 versus 227, and March 31 - 230 versus 234. HDL-C was measured on March 10 and March 31. The apple group had significantly (P value not given) higher HDL-C (24 mg/dl) than the non-apple group (HDL-C = 21 mg/dl) on March 10. On March 31, both groups had the

same HDL-C (18 mg/dl). These researchers attributed the significantly lower serum TC in the apple-eating group to the pectin in the apples.

Stasse-Wolthuis et al. (40) fed a group of 62 students aged 18 to 28 years a diet that was high in dietary fiber (43 g per day) from fruits and vegetables for five weeks. Use of a diet high in fruits and vegetables decreased serum TC concentration by 0.17 mmol/l (not significant). This decrease was more marked after two weeks than after five weeks. The diet did not have any significant effects on HDL-C.

Lewis et al. (50) divided 12 Trappist monks aged 24 to 60 years into four groups and fed them four diets, five weeks each, arranged in a Latin Square design. Diets were a typical Western diet with 19 g of dietary fiber per 2500 kilocalories (A), a fat restricted diet with 20 g of dietary fiber per 2500 kilocalories (B), a fat restricted diet containing a lot of fruits and vegetables with 55 g of dietary fiber per 2500 kilocalories (C), and a diet not restricted in fat but containing a lot of fruits and vegetables with 43 g of dietary fiber per 2500 kilocalories (D). When compared with diet A, the other three diets decreased TC ($P<.01$) with diet B decreasing TC by 22%, diet C by 29%, and diet D by 25%. Diets C and D had TC that were significantly ($P<.01$ and $P<.05$, respectively) lower than diet B. The TG levels on diets C and D were decreased by 21% ($P<.05$) and 26% ($P<.01$), respectively when compared with TG levels of diets A and B. LDL-C levels were decreased significantly ($P<.01$) by 26%, 34%, and 32% in subjects on diets B, C, and D, respectively when compared with diet A. Nonsignificant decreases were seen on diets

B (12%), C (11%), and D (6%) when compared with diet A.

Robertson et al. (48), Gormley et al. (49), Stasse-Wolthuis et al. (40), and Lewis et al. (50) all found that a variety of fruits and vegetables added in various amounts all decreased plasma lipid levels. This action may be occurring because fruits and vegetables contain a lot of pectin which is a water-soluble fiber (49).

I. Other Fiber Sources

There is a small amount of literature available on the effects of other fiber sources, corn bran and bagasse (sugar cane residue), on lipid levels. Walters et al. (51) in a crossover design added 10.5 g of bagasse to the control diets of 19 nuns for 12 weeks. Addition of bagasse to the diets of nuns did not cause a decrease in plasma TC (5.71 mmol/l versus 5.70 mmol/l) or plasma TG levels (0.84 mmol/l versus 0.83 mmol/l).

Munoz et al. (36) fed six healthy male subjects 26 g of corn bran (92%) dietary fiber daily in a metabolic ward for 30 days. Corn bran did not significantly effect plasma TC (176 mg/dl versus 157 mg/dl), LDL-C (115 mg/dl versus 97 mg/dl), and HDL-C (no values). The researchers stated TG levels decreased significantly ($P<.01$) with addition of corn bran; however, no value was given for this.

Limited data on use of corn bran and bagasse on plasma lipids has shown them to be relatively ineffective in decreasing plasma lipids though reasons for this ineffectiveness are unknown (36, 51).

J. Mixed Fiber Sources

Several studies have observed the effects of the use of mixed

fiber sources on plasma lipids. Albrink et al. (52) reported in 1979 on the results of a cross-over design experiment in which the effects of low and high fiber diets on fasting serum TC and TG levels in seven healthy adults whose average age was 25 years. Both diets were high in carbohydrate. The low fiber diet, a milk based liquid formula with 1 g of crude fiber, elevated fasting TG levels by 45% at day six ($P<.001$) and caused TC to decrease 16% below baseline levels ($P<.02$) by the end of the dietary period. The high fiber diet contained 18.0 g of crude fiber and was composed of high fiber foods from a variety of sources: bran cereal, whole wheat bread, cooked dried beans, brown rice, oranges, celery, and lettuce. The high fiber diet decreased TG to a level slightly below baseline and TC levels decreased 23% below baseline levels ($P<.001$).

Stasse-Wolthius et al. (53) looked at the effect of high and low fiber diets and diets high and low in cholesterol on plasma lipid levels in 46 university students. Half of the group was placed on the high cholesterol diet and half on the low cholesterol diet with these two groups being further subdivided into those on high and low fiber diets. After three weeks on one fiber level, groups were switched to the other fiber level for three weeks. At least half of the dietary fiber came from fruits and vegetables with the remainder coming from cereals. The high fiber diet contained at least 16 g of fiber per 1000 kilocalories and the low fiber diet contained no more than 7 g of fiber per 1000 kilocalories. Researchers measured serum TC, HDL-C, and TG. Subjects that were on low fiber diets first and then switched to high fiber diets

at both cholesterol levels had a decrease in serum TC of 0.44 mmol/l ($P<.01$) at the high cholesterol intake and 0.31 mmol/l ($P<.002$) at the low cholesterol level. When going from the high to the low fiber diet, the increase in serum TC was 0.26 mmol/l ($P<.1$) and 0.18 mmol/l (not significant) with the high and low cholesterol diets, respectively. When subjects were switched from the low fiber diet to the high fiber diet, there was a decrease of 0.1 mmol/l in HDL-C on the high cholesterol diet ($P<.025$) and the low cholesterol diet ($P<.05$). There was an increase of 0.1 mmol/l in HDL-C when subjects were switched from the high fiber diet to the low fiber diet at the high level of cholesterol ($P<.002$) and the low level of cholesterol (not significant). No change was noted in serum TG. The researchers suggest that the high amount of fiber in the diet is not the only possible explanation for changes seen but differences in the fat in the diet may account for part of the changes.

High fiber diets from a variety of sources tend to decrease lipid levels but this may possibly be due to additional differences in the diets such as decreases in dietary fat (52, 53).

K. Purified Fibers

1. General Information

Some studies, instead of adding foods that are high in fiber to the diets of subjects, have added purified fibers extracted from foods. The purified fibers that have been used are lignin, cellulose, hemicellulose, pectin and guar gum. Research concerned with addition of purified fibers, cellulose and lignin, to the diets of subjects have provided inconclusive data (37, 54, 58).

However, hemicellulose, guar gum and pectin when added to diets, appeared to have had a hypocholesterolemic effect (38, 40, 59, 60). Purifying fiber from foods may alter the properties of these fibers and change their effect in the body (32).

2. Lignin

Linder and Moller (54) gave lignin at a dose of 1 g two times daily to seven subjects with type II hyperlipoproteinemia. The subjects were on a control diet for four weeks, a treatment period for four weeks, and another control period for four weeks. Plasma TG values did not vary and were 1.9 mmol/l for the first control, 1.9 mmol/l for the treatment period, and 1.8 mmol/l for the second control period. Plasma TC values increased significantly ($P<.05$) when subjects were placed on the lignin containing diet. Plasma cholesterol went from 304 mg/dl in the first control period to 328 mg/dl in the treatment period to 298 mg/dl in the second control period. In this study, lignin appeared to aggravate hyperlipoproteinemia.

Thiffault et al. (55) observed the effects that a diet with added lignin had on the TC levels of six patients 21 to 50 years of age with type II essential hyperlipoproteinemia. Initially patients were given 12 g of cholestyroamine daily for 2 to 5 months for a comparable study. Following this initial study, a lignin preparation called celluline was given at a dose of 1.2 g a day for 2 to 3 months followed by a dose of 4 g a day. While on a control diet, the cholesterol was 402 mg/dl. Addition of cholestyramine decreased cholesterol to 327 mg/dl, while the use

of celluline decreased the cholesterol level to 329 mg/dl. Lignin appeared to be as effective as cholestyramine in treating type II hyperlipoproteinemia.

Inconclusive results about the effectiveness of lignin in decreasing lipids may be due to possible use of different lignin products and different levels of added lignin. Data is needed on the effectiveness of lignin in normocholesterolemic subjects.

3. Cellulose

Prather et al. (56) observed the effects of addition of 13 g of cellulose to the basal diets of five healthy young women aged 18 to 23 years. Subjects were assigned randomly to either the basal diet or the diet that contained 13 g of cellulose added to the basal diet. After four weeks, subjects were switched to the other diet. Weekly blood samples revealed inconsistent results. Three of the subjects had lower serum TC levels (not significant) after four weeks on the cellulose supplemented diet than after four weeks on the basal diet. Two subjects had lower TC levels (not significant) after four weeks on the basal diet than on the cellulose supplemented diet.

Eastwood et al. (37) reported in 1973 on the results of feeding cellulose for three weeks on serum TC values. During the experiment, subjects were asked to keep a daily record of all weighed food and fluid intake. Four subjects were kept on their control diet for three weeks followed by a three weeks in which 8 g of cellulose were added to the control diet two times daily. Researchers found

that addition of cellulose did not change serum TC values throughout the study (186 mg/dl).

Kaur et al. (57) reported the effects of 21 g of cellulose had on serum TC and TG levels of nine healthy girls aged 16 to 18 years. Subjects were placed on a low fiber diet (2 to 3 g of crude fiber) for three weeks followed by three weeks in which 21 g of cellulose were added to the low fiber diet. Serum TC was 151 mg/dl on the low fiber diet and 148 mg/dl on the cellulose supplemented diet. Total TG went from 94 mg/dl on the low fiber diet to 91 mg/dl on the cellulose supplemented diet.

Shurpalekar et al. (58) observed the effects of cellulose on serum lipids in 10 girls aged 10 to 12 years. The girls were placed on three diets for 10 days each. Diets were a control diet, control diet plus four grams of cholesterol, and control diet plus four grams of cholesterol plus 100 g of cellulose daily. At the end of each period, blood samples were taken for cholesterol determination. Cholesterol levels increased significantly when four grams of cholesterol were added to the control diet; from 138 mg/dl to 226 mg/dl. When cellulose was added to the diets, serum cholesterol was significantly decreased to 170 mg/dl.

Addition of cellulose to the diets of healthy subjects generally appears to be ineffective in decreasing lipids (37, 56, 57), however, it may be effective in decreasing serum TC when dietary cholesterol is added in large amounts to the diet (58).

4. Hemicellulose

Kies et al. (59) in 1977 reported on the effect that graded

levels of hemicellulose had on serum cholesterol levels of 12 male subjects in a controlled feeding study. This 50-day study consisted of a two-day depletion period, a three day pre-adjustment period, three randomly ordered 14 day experimental periods and a three day post-adjustment period. Subjects were divided in half so that six received corn oil/peanut oil as their source of fat while the other six received butter oil/peanut oil. The diet contained 6.8 g of fiber before addition of hemicellulose. The hemicellulose was added at a level of 4.2 g during one experimental period, 14.2 g in another experimental period and 24.2 g in a third experimental period. Researchers found that as dietary hemicellulose was increased the average serum TC decreased for both diets. The group receiving the corn oil/peanut oil diet had TC levels of 178 mg/dl, 163 mg/dl, 158 mg/dl, and 147 mg/dl on the control diet, and the diets with 4.2 g of hemicellulose, 14.2 g of hemicellulose, and 24.2 g of hemicellulose, respectively. Addition of hemicellulose to the control diets of men receiving the corn oil/peanut oil diet resulted in an initial decrease in TG levels, however, the additive effect of hemicellulose did not further decrease serum TG. Serum TG were 125 mg/dl, 83 mg/dl, 90 mg/dl, and 82 mg/dl on the control diet, and the diets with 4.2 g, 14.2 g, and 24.2 g of hemicellulose, respectively. The group receiving the butter oil/peanut oil diet had average serum TC of 194 mg/dl, 193 mg/dl, 182 mg/dl, and 165 mg/dl on the control diet, and the diets with 4.2 g, 14.2 g, and 24.2 g of added hemicellulose, respectively. The group receiving the butter oil/peanut oil diet

had an increase in serum TG levels with added hemicellulose from 152 mg/dl, to 154 mg/dl, to 163 mg/dl, and to 208 mg/dl on the control diet, and the diets with 4.2 g, 14.2 g, and 24.2 g of added hemicellulose, respectively.

5. Pectin

Jenkins et al. (38) reported in 1975 on the effects of 36 g of pectin added to diets of seven men. Pectin was added to their normal, self-selected diets for two weeks. Pectin was added either the first or last two weeks of a four week period or the first or last two weeks of an eight week period. Fasting blood samples revealed that addition of pectin caused a significant ($P<.05$) decrease in serum TC of 29 mg/dl for both the four and eight periods.

Stasse-Wolthuis et al. (40) supplemented a controlled low fiber diet of 62 young healthy volunteers with 28 g of citrus pectin for five weeks. Addition of pectin caused serum TC to significantly ($P<.01$) decrease by 0.34 mmol/l (7%) at week 5. There was no significant change seen in HDL-C by the addition of pectin.

Kay and Truswell (60) observed under controlled conditions the effect of adding 15 g of citrus pectin to the diets of nine subjects aged 21 to 28 years. The experiment consisted of a 14-day control period, 21-day experimental period, and another 14-day control period. Fasting blood samples were taken the last three days of each dietary period. Addition of pectin to the diet resulted in an average decrease in cholesterol levels of 13% ($P<.001$). Plasma triglyceride levels were not changed.

Jenkins et al. (38), Stasse-Wolthius et al. (40), and Kay and Truswell (60) have all found that pectin, when added to the diet in varying amounts, decreased TC.

6. Guar Gum

Jenkins et al. (38) observed the effects of 36 g of guar gum added to the diets of seven men. These men were fed guar gum for either four or eight week periods in which guar gum was fed either the first or last two weeks of the period. Analysis of fasting blood samples revealed that guar gum significantly ($P<.002$) decreased TC levels 36 mg/dl in both four and eight week periods.

Jenkins et al. (61) observed the effects of added guar gum and cholestyramine on the plasma lipids of 11 hyperlipidemic patients. Subjects were given cholestyramine (about 10 g per day) and guar in a semihydrated (11 g per day) or hydrated (11 g per day) form for two weeks. In addition, seven patients were placed on guar crispbread (average intake of 13 g of guar per day) for an additional six weeks to determine the long term effects of this treatment. Use of guar crispbread resulted in significant decreases in TC and LDL-C at week eight when compared with baseline values. TC decreased by 13% ($P<.002$), LDL-C by 16% ($P<.02$), and TG by 13% (not significant) at the end of eight weeks. HDL-C decreased by 8% ($P<.05$) at three weeks but returned to the pretreatment level by the end of eight weeks. Addition of hydrated guar to the diet showed a decrease ($P<.05$) of 8% in TC and a decrease in LDL-C by 11% ($P<.05$); however, TG levels showed an insignificant decrease and HDL-C

remained unchanged. The added semihydrated guar gum and cholestyramine resulted in insignificant changes in all the lipid fractions.

Two studies that have added guar gum to the diets of both normocholesterolemic and hypercholesterolemic subjects found that it decreases TC levels.

IV. The Effect of Dietary Cholesterol on Total Cholesterol and Lipoprotein Levels

A. General Information

Epidemiological studies and clinical trial have both contributed evidence showing that dietary cholesterol has an effect on serum TC levels. Those populations which consume diets that are low in dietary cholesterol have lower plasma cholesterol levels than populations that consume 400 or more mg of cholesterol per day (16, 64). Controlled clinical studies have also shown that subjects on diets with little or no dietary cholesterol have decreased plasma cholesterol levels. It has been noted that there is an upper and lower threshold above and below which dietary cholesterol does not have a linear effect on plasma TC. When dietary cholesterol was below 100 to 200 mg per day, there appeared to be no further decrease in TC. When feeding cholesterol in excess of 500-600 mg per day, there appeared to be no further increase in TC. It is possible that there are changes in storage or transport of cholesterol within the body but no changes were noted in the blood (62, 63).

B. Epidemiological Evidence

It was observed that the level of cholesterol in populations was related to the amount of dietary cholesterol consumed. Popula-

tions that consumed diets low in cholesterol generally have lower cholesterol levels than those which consumed diets high in cholesterol and saturated fat (16, 64).

Kato et al. (64) reported in 1973 on the results of a study conducted between 1965-67 on Japanese men living in Japan, Hawaii and California. The researchers looked at the diets and serum TC and TG levels in men in these different areas. Researchers took a 24 hour dietary recall and a blood sample for TC and TG determination. Men in Japan and Hawaii also had a seven day food record taken during a second examination to validate the 24 hour recalls. Men in Japan had much lower serum TC than those living in Hawaii or California (181 mg/dl vs. 218 mg/dl and 228 mg/dl, respectively). Serum TG levels were also lower in men in Japan when compared to Japanese men living in Hawaii or California (134 mg/dl versus 231 mg/dl versus 234 mg/dl, respectively). Men in Japan had lower daily intakes of dietary cholesterol than those living in Hawaii or California (464 mg vs. 545 mg and 533 mg, respectively). Analysis indicated a significant ($P<.01$) positive linear regression between serum TC and dietary cholesterol.

Connor et al. (16) observed the diets and plasma TC and TG levels of the Tarahumara Indians of Mexico, a tribe which seldom consumed meat or animal products. The majority of their diet came from vegetable sources and was low in cholesterol and saturated fat. Estimated average cholesterol intake was 71 mg per day for men and 75 mg per day for women. Plasma TC levels were also low and averaged 116 mg/dl for children and 136 mg/dl for adults. Plasma

TG levels were also low: for children 115 mg/dl and 126 mg/dl for adults. A majority of the serum TC was carried in the LDL-C which averaged 87 mg/dl in the total population. Average HDL-C was 25 mg/dl and average VLDL-C was 21 mg/dl.

C. Clinical Studies

Many clinical studies have looked at the effect of addition of dietary cholesterol on serum TC concentrations; however, limited data is available on the effects of limiting dietary cholesterol on serum TC concentrations and the lipoprotein fractions (65, 66, 67, 68, 69).

Connor et al. (65) observed the effects of egg and crystalline cholesterol diets as well as cholesterol-free diets on serum TC and TG levels of six healthy males. The men were placed on four controlled diets for three weeks each. Subjects were initially placed on a cholesterol-free diet, then a diet with varying levels of egg cholesterol, then came a second cholesterol-free diet, and finally diets with varying amounts of crystalline cholesterol. Subjects received 3000 kilocalories, 70 g of protein derived from casein (except on the egg cholesterol diet when small amount of egg protein were added), 133 g of fat (80 g of peanut oil, 40 g of cocoa butter, and 13 g of safflower oil), and 380 g of carbohydrate. Subjects consumed 475 mg, 950 mg, or 1425 mg of egg cholesterol daily while on the egg diet. Crystalline diets contained 1200 mg, 2400 mg, or 3600 mg of crystalline cholesterol daily. Blood was drawn twice a week after a 14 hour fast for determination of serum TC and TG.

Cholesterol-free diets resulted in decreased serum TC significantly for both the first (58 mg/dl) and second (55 mg/dl) cholesterol-free period ($P<.01$ and $P<.001$, respectively). Initial removal of cholesterol from the diet produced a significant decrease ($P<.001$) in serum TG of 47 mg/dl; however, when cholesterol was removed from the diet a second time, the average fall in serum TG was 6 mg/dl (not significant).

Mattson et al. (66) reported in 1972 on the results of varying levels of dietary cholesterol on TC levels of 56 healthy men aged 21 to 48 years. The subjects were fed initially the institutional diet for seven days before being gradually placed on a zero cholesterol diet which was consumed for 21 days. Subjects were then divided among four experimental diets which contained 0, 106, 212, and 317 mg of cholesterol per 1000 kilocalories by adding egg yolks. No other changes occurred in dietary components and they stayed on these diets for 42 days. Blood samples were taken twice weekly following an overnight fast. When men were placed on the cholesterol-free diet, a decrease in serum cholesterol was attained by day 11 and maintained through day 21. The serum TC level fell from 179 mg/dl at the beginning of the period to 162 mg/dl during the cholesterol-free period. Once subjects had been on the experimental diets for 28 days, their serum cholesterol levelled off. Researchers noted that with each increase of 100 mg of cholesterol per 1000 kilocalories, there was a linear increase of 12 mg/dl in serum cholesterol.

Chenoweth et al. (67) reported in 1981 of the effects of diets

containing low and high amounts of cholesterol had on serum TC, TG and HDL-C in 32 subjects. Subjects were placed on a 10-day control diet before the experimental period. Eight subjects remained on the control diet which contained two eggs. Another eight subjects were placed on the control diet, but consumed egg substitutes instead of eggs. Groups were switched after four weeks on these diets. Another eight subjects were placed on a fat modified diet with two eggs while another group was on the fat modified diet with egg-substitutes. These groups were switched after four weeks. Blood samples were taken after an overnight fast and analyzed for serum TC, TG and HDL-C. The control diet with two eggs contained about 900 mg of cholesterol per day while substitution of the eggs decreased cholesterol intake to about 340 mg per day. The fat-modified diet with eggs contained about 730 mg of cholesterol daily and substitution for eggs reduced cholesterol intake to about 185 mg daily. On the control diet, substitution of eggs with egg-substitutes decreased TC by 26 mg/dl. Men that were placed on the fat-modified diet with two eggs following the control diet had a decrease in TC of 29 mg/dl and there was an additional decrease of 7 mg/dl when egg-substitutes replaced eggs in the diet. Men placed on the fat-modified egg-substitute diet following the control diet had an average decrease of 41 mg/dl. There were inconsistent and insignificant results seen with TG and HDL-C.

McMurray et al. (68) placed eight Tarahumara Indian men on cholesterol-free diets for three weeks before adding 1000 mg of cholesterol to the diet for three weeks. Researchers observed the

effects of these diets on plasma TC, LDL-C and HDL-C. Connor et al. (16) had reported that Tarahumara Indians consumed very low amounts of dietary cholesterol (abour 71 mg per day for men and about 75 mg per day for women). When these men were placed on a cholesterol-free diet no decrease was seen in TC (from 120 mg/dl to 113 mg/dl) and HDL-C (from 31 mg/dl to 27 mg/dl). LDL-C remained at 72 mg/dl throughout.

Cooper et al. (69) in 1982 observed the effects that a "normal" American diet versus a vegetarian-type diet had on plasma TC and TG levels of 15 volunteers. Subjects were randomly assigned to one of two diets for three weeks and then reversed to the other dietary treatment. Dietary cholesterol was present in small amounts on the vegetarian diet (about 33 mg per day) while the control or "normal" American diet contained about 596 mg per day of cholesterol. The vegetarian diet generally resulted in a decrease in all lipids. Plasma TC significantly ($P<.01$) decreased from 160 mg/dl on the American diet to 140 mg/dl on the vegetarian type diet. No significant difference was seen in TG levels when subjects were on the American diet versus the vegetarian diet (68 mg/dl versus 67 mg/dl, respectively). HDL-C levels were not significantly changed when comparing the vegetarian diet (33 mg/dl) to the American diet (37 mg/dl). LDL-C levels were significantly ($P<.025$) higher on the American diet (109 mg/dl) when compared to the vegetarian diet (93 mg/dl).

Restricting or eliminating dietary cholesterol tended to decrease TC levels in plasma. The effects of the restriction of dietary

cholesterol on TG levels and lipoprotein fractions was not as certain (65, 66, 67, 68, 69).

V. Fiber Digestibility

A. General Information

Dietary fiber has been defined as that part of the plant which is resistant to digestion (30); however, when plant fiber is exposed to the human gastrointestinal tract some digestion does take place. The amount of digestion that takes place depends on the type and composition of fiber. Some fibers appear to be completely digested while others appear to be quite indigestible. As the amount of fiber in the diet increases, apparent digestibility of the fiber decreases (70, 71, 72, 73, 74).

B. Fiber Digestibility of Foods High in Dietary Fiber

Farrell et al. (34) reported in 1978 on the digestibility of wheat bran in 14 healthy male subjects aged 22 to 46 years. In this experiment, subjects consumed a diet low or high in dietary fiber. The high fiber diet had 12 g of wheat bran added to the low fiber diet. Subjects were on each diet for 24 days. The apparent fiber digestibility decreased when going from the low fiber diet to the high fiber diet for the neutral detergent fiber (80% versus 55%, respectively), cellulose (74% versus 63%, respectively), and hemicellulose (99% versus 42%, respectively). When subjects went from the low fiber diet to the high fiber diet, apparent digestibility of acid detergent fiber (56% versus 62%), and lignin, silica, and undetermined residue (8% versus 66%, respectively) increased. Generally these researchers found that as the total fiber intake

increased, apparent digestibility of total fiber and fiber components decreased.

Kelsay et al. (70) reported on the effect of fiber from fruits and vegetables on apparent digestibility of fiber in 12 men aged 37 to 58 years. In a crossover design, half of the men were first assigned to a low fiber diet and the other half were assigned to a high fiber diet for 26 days. Apparent digestibility decreased from the low fiber diets to the high fiber diets for the neutral detergent fiber (78% versus 61%, respectively) and hemicellulose (95% versus 88% ($P<.025$), respectively). Average lignin digestibility was 18% on the high fiber diet but was found in such small amounts on the low fiber diet that determination was difficult. Cellulose had an apparent fiber digestibility of 42% on the high fiber diet. Apparent digestibility for acid detergent fiber was 33% on the high fiber diet.

Addition of high levels of wheat bran and fruits and vegetables to diets decreased apparent fiber digestibility of the diets (37, 70).

C. Fiber Digestibility of Purified Fibers Added to the Diet

Several researchers have added the purified fibers, cellulose, hemicellulose, and pectin to the diets of subjects to determine their digestibility (71, 72, 73, 74).

Holloway et al. (71) reported in 1978 on digestion of cellulose, hemicellulose and lignin in two groups of subjects. Ten subjects were healthy individuals and six subjects had an ileostomy. These two groups were used to determine whether

digestion of fibers occurs in the large or small bowel. Subjects were given a diet with known amounts of cellulose, hemicellulose and lignin. Three day fecal samples were then collected and homogenized. Ileostomy subjects excreted 84% of the ingested cellulose as compared with 22% in healthy subjects. It was determined that 28% of the ingested hemicellulose was excreted by ileostomy patients and 4% by healthy subjects. The amount of lignin excreted was about the same as the intake. From this researchers suggested that a majority of cellulose was digested in the large bowel probably by intestinal bacteria while most of the hemicellulose was digested in the small intestine.

Fetzer et al. (72) reported in 1980 on the fiber digestibility of 14.2 grams of added cellulose, hemicellulose or pectin to the diets of eight adolescent boys. The 21 day experiment involved placing subjects on a 2 day depletion period, a 3 day adjustment period and 4, 4 day randomly ordered experimental periods. Subjects were fed the basal diet or the basal diet plus 14.2 g of added cellulose, hemicellulose or pectin. Complete fecal collections were made for each period, homogenized and analyzed for cellulose, hemicellulose, and pectin. Cellulose was found to be approximately 45-46% digestible. Hemicellulose was very highly digestible and ranged from 76-90% digestible. Pectin was not found in fecal samples and so was completely digestible.

Slavin et al. (73) used the neutral detergent (NDF) method to determine digestibility of hemicellulose and cellulose. In one experiment, seven females consumed low fiber diets for one month

(5.4 g per day from fruits and vegetables and refined grains) and high fiber diets for one month (19.3 g per day) which differed only by addition of 16 g of cellulose. Average apparent digestibility of the low fiber diet was 61% which was significantly higher ($P<.01$) than the 38% digestibility of the high fiber diet. The digestibility of cellulose was 70% on the low fiber diet and was 16% on the high fiber diet. Hemicellulose was 46% digestible on the low fiber diet and 31% digestible on the high fiber diet. The second experiment looked at the digestibility of a semi-purified diet (Ensure) and the semi-purified with 16 g of cellulose added to it. Digestibility of the low fiber diet was 10% and decreased to 8% when cellulose was added. The authors suggest that any digestion that occurred in the first experiment was primarily the fruits and vegetables while cellulose was probably not digested much at all.

Cummings et al. (74) reported in 1979 on the fiber digestibility of 36 g per day of a high methoxy pectin added to the diets of five healthy males 21 to 24 years of age. In comparing the uronic acid content of feces both before and after addition of the pectin, it was found that there was no increase in the uronic acid excretion. The amount of uronic acid excretion was 0.21 g per day on the control diet and was 0.16 g per day at week 6 or 7 and 0.31 g per day at week 8 or 9. This study showed that pectin was almost completely digested in the gut probably from bacterial fermentation and concurs with the results found by Fetzer et al. (72).

Research tended to show that cellulose was relatively indigestible in the human small intestine; however, hemicellulose and pectin

tended to be quite digestible. The digestibility of cellulose and hemicellulose tended to decrease with increased fiber consumption. Experiments with healthy and ileostomy patients indicated that a majority of cellulose was digested in the large bowel. Hemicellulose was found to be very digested in both the large and small bowel (70, 71, 72, 73, 74).

VI. Dietary Fiber and Fecal Excretion

A. Dietary Fiber and Total Fecal Excretion

Dietary fiber has other effects on the human body besides lowering plasma lipids. Addition of dietary fiber to diets has resulted in laxation of the bowel. Studies have observed the effects of added wheat bran, corn bran, soy hulls, oat bran, fruits and vegetables, cellulose and bagasse in diets (34, 36, 37, 43, 51, 53, 70).

Several types of wheat fiber have been found to increase the total amount of feces excreted per day (34, 36, 37, 51). Eastwood et al. (37) added 8 g of wheat bran to the diets of 8 subjects for 3 weeks and found that fecal wet weight was increased from 107 g to 174 g ($P<.001$). Walters et al. (51) added 39 g of wheat bran to the diets of 5 volunteers for a week. Fecal wet weight increased significantly ($P<.005$) from 93 g per day to 166 g per day. Farrel et al. (34) noted an increase of fecal dry weight from 116 g to 200 g with addition of 12 g of wheat bran. Munoz et al. (36) fed 26 g of hard red spring wheat bran and soft white wheat bran to 9 and 6 subjects, respectively. Both of these fibers increased daily stool weight significantly ($P<.01$). Soft white wheat bran

increased average daily stool weight from 64 g to 99 g. Hard red spring wheat bran increased average daily stool weight from 81 g to 151 g.

Munoz et al. (36) observed the effects on the average stool weights of adding 26 g of either corn bran or soy hulls to the diets of men. Addition of corn bran increased average daily stool weight significantly ($P<.01$) from 72 g to 144 g. Addition of the soy hulls significantly ($P<.01$) increased average daily stool weight from 68 g to 128 g.

Kirby et al. (43) found that 100 g of added oat bran increased total amount of feces excreted daily from 147 g to 169 g.

Several studies have shown that fruits and vegetables tend to increase the total amount of feces excreted (48, 53, 70). Robertson et al. (48) added 200 g of raw carrot to the diets of 5 subjects for 3 weeks and found that stool wet weight increased from 142 g to 177 g ($P<.05$). Stasse Wolthius et al. (53) found that by increasing the fiber content of the diet of 46 students from 7 g per 1000 kilocalories to 16 g per 1000 kilocalories, half which was in the form of fruits and vegetables and half of which was in the form of cereals, the average fecal wet weight significantly ($P<.001$) increased from 69 g per day to 184 g per day. Kelsay et al. (70) placed 12 men either on a diet low in fiber or high in fiber from fruits and vegetables for 26 days in a crossover design. The amount of fiber consumed on each diet was not specified. The amount of fecal dry matter excreted in g per day was 23 on the low fiber diet and 52 on the high fiber diet.

Walters et al. (51) found that addition of 10.5 g of bagasse to the diets of 19 nuns significantly ($P<.005$) increased daily fecal wet weight from 88 g to 140 g.

Eastwood et al. (37) added 16 g of cellulose to the diets of 8 subjects for 3 weeks. Increased intake of cellulose significantly ($P<.001$) increased fecal wet weight from 152 g per day to 221 g per day. Increase in fecal weight was in the water content of the stool.

The addition of dietary fiber to diets have increased the amount of feces excreted by humans. This has been found to be true with a variety of fibers including wheat bran, corn bran, soy hulls, oat bran, fruits and vegetables, cellulose and bagasse (34, 36, 37, 43, 48, 51, 53, 70).

B. Dietary Fiber and Fecal Fiber Excretion

Several studies have looked at the effects of added dietary fiber on fecal fiber excretion. Farrell et al. (34) added 12 g of wheat bran to the control diets of 14 healthy male subjects aged 22 to 46 years. Subjects were placed on the control diets for 23 days and the control diet with added wheat bran for 24 days. These researchers found that the neutral detergent fiber (NDF) excreted per day increased from 7 g on the control diet to 24 g on the control diet with the added wheat bran fiber.

Kelsay et al. (70) assigned 6 men to diets low in dietary fiber (49.0 g per day) and 6 men to diets high in dietary fiber (24.9 g per day) from fruits and vegetables. They consumed these diets for 26 days before being switched to the other fiber level. The grams of fiber

excreted daily was lower on the low fiber diet (1.1 g) when compared to the high fiber diet (9.6 g).

Fetzer et al. (72) placed eight adolescent boys on a control diet, the control diet plus 14.2 g of cellulose, the control diet plus 14.2 g of hemicellulose, and the control diet plus 14.2 g of pectin. The 21 day experiment involved placing subjects on a 2 day depletion period, a 3 day adjustment period, and 4 day randomly ordered experimental periods. On the control diet, 26 g of fiber were excreted in the four day period which increased to 40 g, 57 g (significantly ($P<.05$) different from control) and 27 g while on diets that had added hemicellulose, cellulose, and pectin, respectively.

Slavin et al. (73) placed seven females on a diet low in dietary fiber (5.4 g per ady) for one month and on a diet high in dietary fiber (19.3 g per day) for one month. Diets differed only by addition of 16 g of cellulose to the low fiber diet. Addition of cellolose increased the amount of fecal fiber excreted per day from an average of 3 g on the low fiber dit to 18 g on the high fiber diet.

Addition of dietary fiber (particularly cellulose) to the diets of healthy subjects increased the amount of fecal fiber excreted by subjects in all the studies reviewed (34, 70, 72, 73).

MATERIALS AND METHODS

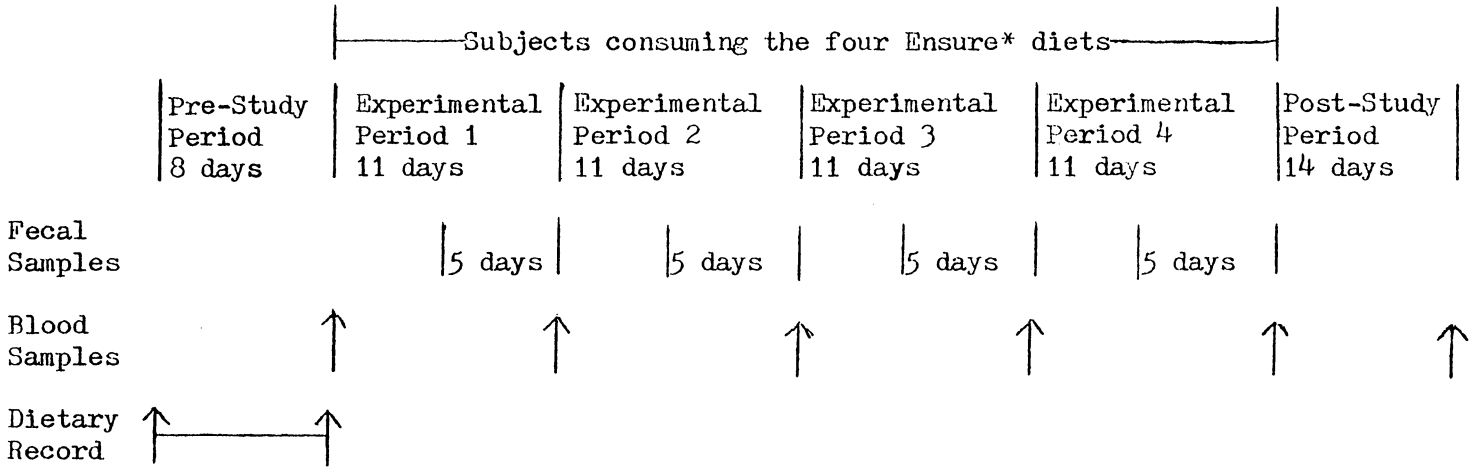
I. General Experimental Design

Twenty-two healthy male subjects, between 18 and 32 years old, were placed on Ensure* and Ensure plus a soy polysaccharide fiber added at three levels as their sole source of nutrition for four, eleven day periods. The low residue Ensure (8111) served as a control. Soy polysaccharide fiber was added to Ensure at levels of 20 grams per 1500 kilocalories (8108), 30 grams per 1500 kilocalories, and 40 grams per 1500 kilocalories (8110). Each subject was placed on the four experimental diets in a random order for each period (see Appendix A).

Subjects were divided into two groups of eleven each before being randomly assigned numbers. Subjects assigned to group one were those that would be leaving campus for Thanksgiving break. This group started the study one day before the subjects in group two.

Figure 1 shows a schematic diagram of the experimental design. During the preliminary period of eight days each participant followed his normal dietary pattern and kept daily food records for the last five days (see Appendix B). This provided information about the normal dietary pattern of the individual and the normal amount of fiber in the diet. Subjects were fed during the 44 day experiment under controlled feeding conditions. During the 44 day experiment, subjects were required to eat three meals a day, seven days a

*Ensure of Ross Laboratories, Columbus, Ohio 43216



*Ensure of Ross Laboratories, Columbus, Ohio.

Figure 1. A schematic diagram of the general experimental design.

week at Solitude, the metabolic unit of the Department of Human Nutrition and Foods. Occasionally, when necessary, subjects were given snacks (Ensure) to take with them. Subjects consumed only Ensure and distilled, deionized water during the 44 day study. All of the experimental diet (1500 kilocalories) was consumed at meals with as much of low residue Ensure as needed to maintain body weight. Subjects were given snacks, low residue Ensure, to take with them, if they were unable to consume all of their kcal at the 3 meals. Following the 44 day experimental period, subjects were asked to slowly resume their normal dietary pattern. Subjects were asked not to exercise heavily during the 44 day experimental period.

Seven blood samples were taken by venipuncture from subjects during the course of the study. Ten ml samples were taken to determine eligibility and for the follow-up sample. Twenty ml samples were taken other times. The first blood sample was taken to determine eligibility for the study. The second blood sample was taken the first day of the first experimental period prior to breakfast. During the experiment, blood samples were taken at the end of each experimental period before the breakfast that began the new experimental diet. The final blood sample was taken two weeks following the last day of the experimental diets.

Throughout the study, subjects were required to collect complete 25 hour fecal samples in wax coated container provided. Plastic lined tote bags were given to subjects to carry collections in.

Body weights were recorded daily before breakfast. Loss in body weight was adjusted by adding extra calories in the form of low residue

Ensure to the diet.

The chemical analyses performed included:

1. Fiber analysis of each of the experimental diets.
2. Fiber analysis of the homogenized fecal samples collected during the balance period of each diet.
3. Analysis of plasma total cholesterol (TC), high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL) (by difference), and triglycerides during the preliminary period, at the end of each experimental period, and at a two week follow-up period.

II. Recruitment of Subjects

Flyers providing basic information about the study were placed on bulletin boards in academic buildings throughout the campus of Virginia Polytechnic Institute and State University. Persons interested in more information about the study were requested to call the Department of Human Nutrition and Foods. During the phone conversation, more information was provided about the study. The interviewer also obtained general information from the caller, asked some general questions about their health history, and questioned the caller at length about type and amount of physical activity they engaged in.

Subjects that were interested in the study met with the interviewer to determine eligibility for the study. While meeting with the interviewer, the subject was given a questionnaire (see Appendix C). The questionnaire asked questions concerning age, height, weight, and general health. Subjects were also asked about

their use of medication, use of vitamin and mineral supplements, tobacco usage, drug usage, coffee and tea comsumption, alcohol consumption, and allergies. Questions were asked about consistency and frequency of subjects normal bowel movements. The individuals were also asked if they had difficulty with their bowel movements. Exercise and travel plans for the study duration were also asked of the subjects.

Subjects were eliminated if they had any known metabolic diseases, or family history of heart disease or high blood pressure. If persons did not fall between the 25th and 75th percentiles for weight for height as indicated by the Health and Nutrition Examination Survey (HANES), U.S. 1971-74 (75), they were not allowed to participate in the study. Subjects could not smoke, consume alcohol, take drugs or medications, or consume tea or coffee for the duration of the study. If any person had known allergies to soy products, they could not participate since the diet contained soy fiber and soy protein isolate. Individuals had to be able to consume at least 1500 kilocalories of Ensure without weight gain. Persons were eliminated if they exercised heavily. Individuals could not participate if they were found to have travel plans that took them away from campus for more than one day.

Persons eligible for the study then gave a 10 ml fasting blood sample which was analyzed for plasma TC. Individuals were not accepted if they had a plasma TC of more than 225 mg/dl.

Twenty-two eligible persons were chosen to participate in the study. All subjects were required to sign a consent form to partici-

pate in the study after hearing and reading a complete explanation of the study (see Appendix D).

III. Composition and Feeding of Diets

All subjects consumed at least 1500 kilocalories per day of Ensure at one of four fiber levels as the experimental diet for each 11 day period. As stated earlier, subjects were randomly assigned to each fiber level for each period. The only differences in each diet was addition of a soy polysaccharide fiber at three different levels to each of the diets. The diet in which soy polysaccharide fiber was not added served as a control (8111). The diet in which soy polysaccharide fiber was added at 20 grams per 1500 kilocalories was the low fiber diet (8108). The diet in which soy polysaccharide fiber was added at 30 grams per 1500 kilocalories was the intermediate fiber diet (8109). The diet in which soy polysaccharide fiber was added at 40 grams per 1500 kilocalories was the high fiber diet (8110).

Ensure is a nutritionally complete diet (see Appendix E). The protein sources in Ensure are caseinate and soy protein isolate. Ensure also contains corn starch and sucrose as the carbohydrate sources. Corn oil serves as the fat source in the diet. Ensure is supplemented with all vitamins and minerals known to be necessary for good health. Proximate analysis of Ensure is given in Appendix F.

All diets met or exceeded the subjects' requirement for essential nutrients as established by the Food and Nutrition Board of the National Academy of Sciences-National Research Council (1974). This is

based on an intake of 2000 kilocalories.

Daily caloric intake varied with the subject but was at least 1500 kilocalories for each subject each day. Additional kilocalories to maintain weight were provided in the form of low residue Ensure. Subjects that began to gain or lose weight had their caloric intake adjusted by decreasing or increasing the amount of the low residue Ensure consumed to help maintain weight.

Meals were consumed in the metabolic unit of the Department of Human Nutrition and Foods at regular meal times. Snacks (low residue Ensure) were given to subjects to consume between meals away from the metabolic unit of the Department of Human Nutrition and Foods when necessary. The amount of low residue Ensure consumed as a snack was recorded at the next meal. Some subjects took day trips. When this situation arose, they were given their meals for the day plus snacks and were asked to keep a record of the amount of Ensure consumed at each meal. Distilled, deionized water and sugarless gum were provided ad libitum. The amount of distilled, deionized water consumed and gum chewed was recorded daily.

IV. Collection and Preparation of Blood Samples

Subjects were instructed to fast for at least 12 hours prior to blood sampling. Samples were drawn after an overnight fast prior to breakfast by a licensed medical technologist. Multisample needles (21 gauge, $1\frac{1}{2}$ inch) and 10 ml vacutainers containing solid disodium ethylenediaminetetracetic acid (EDTA) were used at all times. A tourniquet was applied initially. Once the vacutainer was filled it was immediately inverted several

times to gently mix the blood with disodium EDTA. The labelled vacutainers were then placed on wet ice until being spun down.

Following collection of all blood samples, plasma was separated from red blood cells by centrifugation at 3000 rpm at 4°C for 30 minutes. Plasma was removed by pipetting and was stored in 5 ml polyethylene test tubes. Plasma was aliquoted for plasma TC and separation of plasma HDL-cholesterol. After this aliquot was made, plasma was frozen at -20°C until analysis. A test tube of blood was sent to Consolidated Biomedical Laboratories, Richmond, Virginia for analysis of plasma total triglycerides.

V. Precipitation of LDL and VLDL for Separation of HDL-Cholesterol

HDL-cholesterol was measured directly in the supernatant which had HDL isolated by selective precipitation of LDL-C and very low density lipoprotein (VLDL-C) by use of a heparin/manganese chloride solution according to the method of Albers et al. (76).

Separation of HDL was performed the day that the sample was taken. A fresh working solution of heparin/manganese chloride was prepared each day. The working solution was prepared by adding 0.6 ml of a sodium heparin solution (40,000 USP units/ml of 1.5M saline) to 1.0 ml of 1.06M manganese chloride.

One ml aliquots of plasma were pipetted into 12 ml conical tubes. To plasma, 0.1 ml of the heparin/manganese chloride solution was added. Addition of the heparin/manganese chloride solution caused precipitation of LDL and VLDL to occur immediately. The conical tubes were then vortexed after the addition of the heparin/

managanese chloride solution and allowed to sit for 10 minutes. Precipitated samples were spun at 3000rpm for 30 minutes at 4°C. Centrifugation caused formation of a hard pellet at the bottom of the conical tubes. The supernatant was pipetted into 5 ml polyethylene storage test tubes and frozen at -20°C.

The supernatant solution was later analyzed by use of the colorimetric Liebermann-Burchard reaction. The procedure for this method is outlined in Section VI.

VI. Plasma Total Cholesterol and HDL Cholesterol Determination

Determination of plasma TC and HDL-C was performed by a method using the colorimetric Liebermann-Burchard reaction in the method and reagents from Stanbio Laboratories*. All samples were analyzed in duplicate. A reference serum with a known amount of cholesterol was included in each run to check accuracy of the methods. A fresh reference serum was prepared weekly to be used in each run. The reference serum could be precipitated like the plasma samples so that an HDL-C reference could be used also.

Plasma and HDL supernatant were removed from the freezer and allowed to come to room temperature along with the reagent while the water bath increased to 37°C which was necessary for the analysis. A 0.1 ml fixed Eppendorf pipette was used to pipette samples, standards, water blanks, and reference serum in duplicate into 15 ml glass test tubes. Six ml of STANBIO cholesterol color reagent was pipetted into each test tube by use of an adjustable

*STANBIO Laboratory, Inc., SR Direct Cholesterol Test Set, San Antonio, Texas.

repipette. Samples, standards, water blanks, and reference serums were then vortexed and incubated for 20 minutes in a water bath at 37°C. Once removed from the water bath, samples, standards, blanks, and reference serums were vortexed again and read at 625 nm on a Spectronic 21** spectrophotometer within 30 minutes. Concentrations of the unknown samples were calculated from standard curves using linear regression on a calculator.

VII. Plasma Total Triglyceride Determinations

Plasma total triglycerides were determined by use of an automated SMAC system by Technicon***. Samples were sent to Consolidated Biomedical Laboratories, Richmond, Virginia for analysis. Technicon method number SG4-0023FH9 was used. The method requires the use of two independent, but interrelated sample and blank channels.

VIII. Determination of LDL Cholesterol

LDL-C was determined by calculation. The calculation method was determined by Friedewald et al. (77). In order to be able to calculate LDL cholesterol, values for TC, HDL-C, and triglycerides were needed. LDL-C was calculated by use of the following formula:

$$\text{LDL-C} = \text{TC} - (\text{Triglyceride}/5 + \text{HDL-C})$$

IX. Collection and Preparation of Fecal and Food Samples for Fiber Analysis

Complete fecal samples were collected daily from all subjects

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**Spectronic 21 spectrophotometer, Bausch and Lomb, Rochester, N.Y.

***Technicon Method No. SG4-0023FH9. Technicon Instrument Corp., Tarrytown, N.Y.

from the preliminary period through the end of the last experimental period of the study. Collections were made in paper containers that had a waxy outer coating. Containers were labelled with subject number, subject name, date and time of collection. Samples were brought into the metabolic unit of the Department of Human Nutrition and Foods and refrigerated. Daily, samples were taken out of the refrigerator to be weighed and determine the physical consistency. Samples were then frozen until they could be composited for the balance period.

The balance period consisted of the last 5 days of each eleven day experimental period and was determined by use of fecal markers consisting of brilliant blue dye and methyl cellulose in a gelatin capsule. The methyl cellulose and brilliant blue dye were in a ratio of 7:1. Subjects were given a fecal marker at breakfast of the first day and last day of the balance periods. The samples for balance periods were determined to begin with the beginning of the first fecal marker and end with the beginning of the second fecal marker.

At the end of the study, frozen samples from each balance period for each subject were composited. These samples, which began with the first fecal marker through the samples up to the second fecal marker, were placed in a 5 quart stainless steel Waring Blender with distilled, deionized water and blended for 5 minutes. An aliquot of the blended sample was immediately poured into an acid washed 250 ml polyethylene bottle, capped and frozen for later analysis. The knife, spatula, and blender used in the homogenization process

were cleaned between samples. The knife and blender were rinsed well with hot tap water and then several times with distilled, deionized water. The spatula was rinsed with hot tap water, an acid solution, and then distilled, deionized water.

A random bottle of each diet 8108, 8109, 8110, 8111 was selected for food analysis. The food was homogenized by taking a bottle of each of the diets and mixing the bottle well.

Homogeneous fecal and food aliquots were prepared for freeze-drying by being frozen in plastic containers with a large surface area on the bottom. Once frozen, samples were placed in a freeze drier for 72 hours or until completely dry. Samples were then crushed by use of a mortar and pestle to obtain a fine, homogeneous powder. Mortars and pestles were cleaned and dried between samples. Samples were then dried in an oven at approximately 50°C until no more moisture could be removed. This was approximately 24 hours. Samples were then stored in a dessicator until use for fiber analysis discussed in Section X.

X. Total Neutral Detergent Fiber Analysis of Food and Fecal Samples

Total fiber analysis of food and feces samples was performed according to the amylase modified neutral detergent method of Robertson and Van Soest (78). Samples were mixed by turning the storage container for samples before fiber analysis to obtain a homogeneous sample. Approximately 0.5 g of sample was weighed into a 600 ml Berzelius beaker. To this was added 50 ml of neutral detergent solution. Samples were digested for 30 minutes on hot plates with a refluxing condenser over the mouth of the Berzelius beaker. After

30 minutes of digestion, 50 ml of neutral detergent solution and 2 ml of alpha-amylase (derived from bacillus subtilis) were added to the samples. Samples were then digested for 30 more minutes on the hot plates with the refluxing condensers.

Samples were then filtered in preweighed scintered glass crucibles containing preweighed filter paper. Filter paper was used with the scintered glass crucibles because without filter paper the fiber particles were so small that when filtering was done, the pores of the scintered glass crucibles appeared to get clogged to the point that the sample could not be filtered. The Berzelius beakers were rinsed with hot distilled, deionized water to get the sample out of the beaker. This hot water was used to rinse the sample on the scintered glass crucible seven times to make sure all the neutral detergent solution had been rinsed from the sample. The sample was then rinsed with acetone two times. The sample was then dried in an oven at 100°C overnight. Samples were then put in a dessicator to cool and then were weighed. The percent cell wall was determined by the following formula:

$$\frac{\text{Initial sample weight - Final sample weight}}{\text{Initial sample weight}} \times 100$$

Samples were not ashed after being weighed because the fiber samples were saved to determine fiber energy.

After filter paper was removed, the scintered glass crucibles were cleaned by being ashed at 500°C for at least two hours. Crucibles were cooled before removing from the muffle furnace. Once cool, crucibles were back washed with distilled, deionized water. Crucibles were dried in an oven at 100°C overnight before using again.

XI. Fiber Digestibility

Apparent digestibility was determined by the following formula:

$$\frac{\text{Total fiber intake - Total fiber output}}{\text{Total fiber intake}} \times 100$$

Fiber intake was obtained by taking the percent of fiber as determined by the Neutral Detergent Fiber procedure in each of the experimental Ensure products and multiplying this by the amount of experimental diet consumed during the balance period. Since low residue Ensure was consumed for additional kilocalories, the fiber intake was also determined for low residue Ensure consumed during the balance period. Fiber intakes from experimental Ensure and low residue Ensure were added together to determine total fiber intake. Total fiber output was obtained by taking the percent of fiber, as determined by Neutral Detergent Fiber procedure, multiplying this by total fecal output for the balance period. These values were put into the above formula to determine apparent fiber digestibility.

XII. Statistical Analysis

Mean plasma TC, HDL-C, LDL-C and plasma total TG were expressed as mg/dl. Mean fecal fibers were expressed in g per day, percent cell wall and percent apparent fiber digestibility. Mean plasma TC, HDL-C, LDL-C, TC/HDL-C, LDL-C/HDL-C, and plasma total TG, were tested for significant differences between and within treatment groups by use of an analysis of variance model. Duncan's Multiple Range Tests were also performed on each of the above variables to determine where significant differences occurred between and within treatment

groups. Statistical tests were performed at the .05 level of significance. Mean percent cell wall, grams of fecal fiber per day, and percent apparent fiber digestibility were compared using multiple paired t-tests with a Bonferroni adjustment to maintain an overall significance level of .05. In comparisons involving the four study diets, an individual P-value $<.00833$ was required to declare statistical significance.

RESULTS

I. Subject and Diet Information

A. Subject Information

General physical characteristics of the subjects are given in Appendix G. The subjects ranged in age from 18 to 31 years with an average age of 22.95 years. Their height ranged from 1.70 meters to 1.96 meters, with an average height of 1.80 meters. Subject weights ranged from 60.00 kilograms to 101.91 kilograms and averaged 75.68 kilograms. Subjects were to be between the 25th and 75th percentiles in their age group for weight by height according to the HANES (75) data. Six subjects fell below the 25th percentile when considering their weight, age, and height. These were subjects 5, 13, 18, 19, 20, and 21. Two subjects, 16 and 22, were above the 75th percentile for weight when considering their age and height.

A few subjects indicated use of tobacco, social drugs or alcoholic beverages, but agreed to give these up for the duration of the study. Several subjects also indicated habitual use of coffee, tea, or vitamin and mineral supplements but agreed to give these up for the length of the study. No subjects were accepted for the study who regularly took medication. If any subject took medication during the study, they were asked to record this on their daily activity record form. No subject had had hypertension at any time. No subject indicated any allergies to soy products. No subject indicated regular participation in strenuous sports and they were not encouraged to exercise heavily.

Six subjects dropped out from the study because of personal reasons.

B. Dietary Information

A comparison of each of the four diets is found in Table 1. Each diet had the same composistion, varying only in amount of dietary fiber added to each diet. Addition of dietary fiber increased the amount of carbohydrate in each diet. This additional carbohydrate had a dilutional effect on the proportion of fat and protein in each diet but had no effect on grams of protein and fat in each bottle of diet. Addition of extra kilocalories in the form of the control diet had no effect on the percentage of the diet coming from fat, protein and carbohydrates. Average nutrient consumption of subjects on each diet is found in Table 2.

II. Body Weight Information

Average body weight information of subjects is found in Tables 3 and 4. These tables provide initial weight, ending weight, and weight change while on each experimental diet and over the duration of the experiment. Not much change in average weight was seen over time. The first eleven days resulted in the largest average weight change for subjects during any period. By the end of the study, the average weight was about the same as the average weight seen at the beginning of the study.

Average weights when reported by experimental diets again did not show much change over each period. The control diet had the greatest average weight change with a loss of 0.60 kilograms. Other diets had very small changes in average weight during the dietary

TABLE 1
Nutrient Composition of Low Residue Ensure* and the Ensure With Soy Fiber Added At Three Levels (g/100 ml)

	Diets			
Nutrient	Control	Low Fiber	Intermediate Fiber	High Fiber
Protein	3.76	3.88	3.88	3.92
Fat	3.76	3.76	3.71	3.63
Carbohydrate	14.39	15.23	15.68	16.33
Ash	0.68	0.72	0.76	0.76
Fiber	0.18	0.20	0.45	0.70

*Ensure of Ross Laboratories, Columbus, Ohio.

TABLE 2
Average Daily Nutrient Consumption on the Low Residue Ensure* Plus Ensure With the Soy Fiber Added At Three Levels (g/d)

	Diets			
Nutrient	Control	Low Fiber	Intermediate Fiber	High Fiber
Protein	104.11	106.97	109.61	109.11
Fat	104.11	103.48	104.84	100.90
Carbohydrate	403.42	419.74	443.20	454.04
Ash	18.93	19.77	21.44	21.12
Fiber	5.02[1]	5.36[2]	8.87[3]	12.56[4]
Kilocalories	2758.00	2907.00	2978.00	2933.00

[1]Includes 2.53 g of experimental fiber

[2]Includes 2.90 g of experimental fiber

[3]Includes 6.42 g of experimental fiber

[4]Includes 10.02 g of experimental fiber

*Ensure of Ross Laboratories, Columbus, Ohio.

TABLE 3
Subject Weight Change Over Time (Kilograms)

Weight	Period 1	Period 2	Period 3	Period 4
Initial Weight	73.8	73.1	73.9	74.1
Final Weight	73.1	72.9	73.8	74.0
Weight Change	-0.7	-0.2	-0.1	-0.1

TABLE 4
Subject Weight Change On Ensure* and Ensure Plus Soy Fiber Added At Three Levels (Kilograms)

	Diets			
Weight	Control	Low Fiber	Intermediate Fiber	High Fiber
Initial Weight	73.4	72.4	74.2	74.9
Final Weight	72.8	72.2	74.1	75.0
Weight Change	-0.6	-0.2	-0.1	+0.1

*Ensure of Ross Laboratories, Columbus, Ohio.

treatments, none greater than 0.50 kilograms. Some individuals did have great weight changes while on experimental diets (Appendix I).

Subjects that showed a consistent weight loss were given extra bottles of low residue Ensure to increase their intake of kilocalories.

III. Plasma Total Cholesterol (TC), Low Density Lipoprotein-Cholesterol (LDL-C), High Density Lipoprotein-Cholesterol (HDL-C), Plasma Total Triglycerides (TG) and Ratios of LDL-C/HDL-C and TC/HDL-C

Mean values for plasma TC, LDL-C, and HDL-C are shown in Table 5.

Regardless of the experimental diet, there were no significant differences nor trends between treatments in plasma TC. Plasma TC did decrease significantly ($P<.05$); however, when subjects went from self-selected diets onto the four different liquid formula diets containing no cholesterol. When subjects then went from the liquid formula diet to their self-selected diets for two weeks at the end of the study, plasma TC increased significantly ($P<.05$) to a level similar to the pre-study level.

LDL-C decreased significantly ($P<.05$) when participants were placed on the liquid diets. As with TC, addition of different fiber levels to the liquid diet did not result in any significant differences between treatments in LDL-C. When participants went from experimental diets back to self-selected diets, there was a significant ($P<.05$) increase in LDL-C.

HDL-C levels were affected by the liquid formula diets in a manner similar to TC and LDL-C. There were no significant differences nor trends in HDL-C between different fiber levels

TABLE 5
Effect of Ensure* and Ensure Plus Three Levels of Added Soy Polysaccharide Fiber on Plasma Total and Lipoprotein Cholesterol Levels in Young Men

Experimental Diet	TC mg/dl	LDL-C mg/dl	HDL-C mg/dl
Pre-Study Self-Selected	164±5[a]	108±5[a]	38±3[b]
Control	131±4[b]	81±4[b]	36±2[b]
Low Fiber	129±4[b]	81±4[b]	35±2[b]
Intermediate Fiber	136±4[b]	85±4[b]	38±2[b]
High Fiber	132±5[b]	82±5[b]	37±2[b]
Post-Study Self-Selected	172±7[a]	102±7[a]	54±3[a]

Values are given as the Mean±SEM.

Values with different superscripts are significantly different ($P<.05$) between diets.

*Ensure of Ross Laboratories, Columbus, Ohio.

added to the formula diet. The HDL-C values on the pre-study and experimental diets did significantly ($P<.05$) differ from post-study HDL-C values.

LDL-C/HDL-C and TC/HDL-C ratios are shown in Table 6. The ratio of LDL-C/HDL-C at the pre-study were significantly ($P<.05$) higher than LDL-C/HDL-C ratios of the experimental diets and post-study. Experimental diet ratios and post-study diet ratios did not significantly differ; however, there was an apparent decrease (not significant) in the post-study ratio when compared to the ratios of experimental diets.

TC/HDL-C ratios were significantly higher ($P<.05$) at the pre-study period when compared to experimental and post-study periods. There were no significant differences between experimental and post-study TC/HDL-C ratios. There was an apparent decrease (not significant) in the post-study ratio when compared to TC/HDL-C ratios of the four experimental diets.

Mean values for plasma total TG are given in Table 7. Plasma total TG significantly ($P<.05$) decreased when subjects were placed on the liquid formula diets. TG decreased significantly ($P<.05$) when the pre-study diet was compared with the low and intermediate fiber and the post-study diets. Though there appeared to be decreases in plasma TG for the control and high fiber diets compared to the pre-study diet, they were not significant.

IV. Fecal Fiber and Fiber Digestibility

Results of increased fiber consumption of fecal fiber in grams per day, percent fiber as well as percent fiber digestability are

TABLE 6
Effect of Ensure* and Ensure Plus Three Levels of Added Soy Polysaccharide Fiber on TC/HDL-C and LDL-C/HDL-C Ratios of Young Men

Experimental Diet	TC/HDL-C	LDL-C/HDL-C
Pre-Study, Self-Selected	4.6 ± 0.3^{a}	2.9 ± 0.2^{a}
Control	3.8 ± 0.2^{b}	2.4 ± 0.1^{b}
Low Fiber	3.9 ± 0.2^{b}	2.5 ± 0.2^{b}
Intermediate Fiber	3.8 ± 0.2^{b}	2.4 ± 0.2^{b}
High Fiber	3.8 ± 0.2^{b}	2.3 ± 0.2^{b}
Post-Study, Self-Selected	3.4 ± 0.2^{b}	2.0 ± 0.2^{b}

Values are given as the Mean±SEM.

Values with different superscripts are significantly different ($P<.05$) between diets.

*Ensure of Ross Laboratories, Columbus, Ohio.

TABLE 7
Effect of Ensure* and Ensure Plus Three Levels of Added Soy Polysaccharide Fiber on Plasma Total Triglycerides of Young Men

Experimental Diet	Triglycerides mg/dl
Pre-Study, Self-Selected	85 ± 8^{a}
Control	72 ± 7^{ab}
Low Fiber	64 ± 5^{b}
Intermediate Fiber	69 ± 6^{b}
High Fiber	72 ± 6^{ab}
Post-Study, Self-Selected	64 ± 7^{b}

Values are given as Mean±SEM.

Values with different superscripts are significantly different ($P<.05$) between diets.

*Ensure of Ross Laboratories, Columbus, Ohio.

found in Table 8.

Increased consumption of soy polysaccharide fiber from the three experimental diets containing the fiber resulted in an increase in fecal fiber, both in grams per day as well as percent fiber. Amount of fiber found in feces (grams per day) increased significantly ($P<.05$) from the control diet to intermediate and high fiber diets. Increase in fecal fiber in grams per day from the control diet to the low fiber diet was not significant.

Apparent fiber digestibility of the fiber decreased significantly ($P<.05$) from the control diet to intermediate fiber diet. Decrease from the control to low fiber diet was not significant. Apparent fiber digestibility then increased from intermediate to high (not significant) fiber diets.

TABLE 8
Effect of Ensure* and Ensure Plus Three Levels of Added Soy Polysaccharide Fiber on Fecal Fiber (g per day and %) and Apparent Fiber Digestibility

Experimental Diet	Fiber Intake g/d	Fecal Fiber g/d	Fecal Fiber %	Apparent Digestibility %
Control	5.02[1]	0.40±0.10[a]	2.40±0.70	91.70±2.50[a]
Low Fiber	5.36[2]	1.30±0.30[ab]	6.00±1.40	75.70±6.40[ab]
Intermediate Fiber	8.87[3]	2.70±0.60[b]	9.40±2.30	70.10±7.10[b]
High Fiber	12.56[4]	2.90±0.70[b]	9.50±2.30	76.90±5.90[ab]

Values are given as Mean±SEM.

Values with different superscripts are significantly different ($P<.05$) between diets.

[1]Includes 2.53 g of experimental fiber

[2]Includes 2.90 g of experimental fiber

[3]Includes 6.42 g of experimental fiber

[4]Includes 10.02 g of experimental fiber

*Ensure of Ross Laboratories, Columbus, Ohio.

DISCUSSION

I. Plasma Lipids and Lipoprotein Cholesterol

There has been much research observing the effects of natural and purified fibers on plasma lipids and lipoprotein cholesterol. Studies have been conducted under variable conditions: adding different amounts of dietary fiber, using both hyperlipidemic and normolipidemic subjects and using a variety of experimental designs. Use of soy fiber in studies has been limited and has provided inconsistent results. This study was undertaken to determine the effects of an added soy polysaccharide fiber on plasma lipids in healthy young men under controlled feeding and metabolic conditions. It was found that addition of this soy polysaccharide did not result in any significant differences between treatment in plasma TC, LDL-C, HDL-C, total TG, LDL-C/HDL-C or TC/HDL-C ratios. Several possible explanations exist as to why soy fiber did not affect plasma lipids or lipoproteins.

A possible reason why soy fiber used in this study was not effective was that not as much dietary fiber was consumed as in other studies. The diet with the highest amount of fiber in our study contained 40 grams of added soy polysaccharide fiber to Ensure and was approximately 26% dietary fiber. When the experimental fiber consumption was added to the fiber consumption in low residue Ensure, the total consumption of dietary fiber on the high fiber diet was approximately 13 grams per day. In the study by Munoz et al. (36), subjects consumed 26 grams of soy hulls daily which were found to contain 86.7% fiber. The actual

consumption of dietary fiber was then approximately 22.5 grams per day. Other differences in the diet may also account for differences in results. The diet for subjects in the study by Munoz et al. (36) had higher levels of protein (16% of total kilocalories versus 14% of total kilocalories), fat (40% of total kilocalories versus 32% of total kilocalories, and cholesterol (360 to 780 mg per day versus 0 mg per day) but lower levels of carbohydrate (44% of total kilocalories versus 54% of total kilocalories) when compared to the present study.

Another reason why this soy fiber was not effective was that addition of fiber to a zero-cholesterol diet may have made further changes in plasma lipids and lipoproteins very difficult to detect. Removal of dietary cholesterol significantly ($P < .05$) decreased plasma TC, LDL-C, HDL-C and plasma total TG in the present study. In the literature, restricting or eliminating cholesterol has been found to decrease TC, but with inconsistent changes in lipoprotein fractions (14, 44, 65, 66, 67, 68). In looking at Tarahumara Indians, Connor et al. (16) found that the level of dietary cholesterol was low (71-75 mg per day) as were their plasma lipids and lipoproteins with average values being: TC-126 mg/dl, TG-120 mg/dl, LDL-C-87 mg/dl and HDL-C-25 mg/dl. Values for TC and LDL-C were similar to values seen for subjects in the present study with TC ranging from 129±4 mg/dl to 136±4 mg/dl and LDL-C ranging from 81±4 mg/dl to 85±4 mg/dl. In a separate study McMurray et al. (68) took eight Tarahumara Indian men and placed them on a cholesterol-free diet and found that TC went from 120 mg/dl

to 113 mg/dl, HDL-C went from 31 mg/dl to 27 mg/dl, and LDL-C remained at 72 mg/dl. Since Tarahumara Indians were not on a diet that was very high in dietary cholesterol initially, this is probably why significant decreases were not seen in plasma TC, HDL-C, and LDL-C, similar to adding dietary fiber to a zero-cholesterol diet in the present study. Mattson et al. (66) and Connor et al. (65) studied the effects of a zero-cholesterol diet on TC levels and found that a significant decrease in TC occurred when placing men on zero-cholesterol diets as found in the present study. Raymond et al. (44) noted a 10 to 31% decrease in plasma TC when he placed eight healthy subjects on an essentially cholesterol-free diet (50 mg of cholesterol daily). No mention was made of how lipoprotein fractions responded to the cholesterol-free diet. Addition of the fiber mixture which contained soy hulls did not have any effect on plasma TC or any of the lipoprotein fractions. Chenoweth et al. (67) found that by decreasing the dietary cholesterol, there was a decrease in TC of 10.8% on a control diet and 15.5% to 19.0% on a fat modified diet but saw inconsistent and insignificant results with TG and HDL-C. Cooper et al. (68) found by decreasing dietary cholesterol, TC and LDL-C significantly decreased (by 20 mg/dl and 16 mg/dl, respectively) but not much change was seen in TG and HDL-C.

Additionally, subjects may not have been on the diet long enough for the fiber to influence plasma lipids or lipoproteins. It took approximately ten to fourteen days for lipids to stabilize after initiation of a new dietary treatment according to some studies

(65,66). However, one study (47) found that TC was significantly decreasing up to four weeks after administration of a new dietary treatment. Subjects in the present study were only on each of four different diets for eleven days each.

A further reason why the soy fiber may not have been effective in decreasing plasma lipids was that processing of the fiber into liquid formula diets may have, in some yet unknown manner, decreased the effectiveness of the fiber. The use of added wheat bran has resulted in conflicting data on its effects on plasma TC levels. In its raw or unprocessed form, it has been shown to decrease plasma TC by 10.1% (34), but in other forms, it has been shown to have no effect (37, 39) or even increase plasma TC (40). The bran used in studies where it has had no effect or increased plasma TC, has been processed in some fashion that may have decreased its effectiveness.

Finally, normocholesterolemic subjects may be less likely to respond to a dietary treatment than hypercholesterolemic subjects would be, especially when placed on a cholesterol-free diet. Normocholesterolemic subjects (42) placed on an oat bran diet did not have as much decrease in TC and TG (8% and no change, respectively) as subjects that were hypercholesterolemic (13% and 9%, respectively) (43). Cellulose was generally found to be ineffective in decreasing TC when added to diets (57) (151 mg/dl on the low fiber diet and 148 mg/dl on the cellulose diet) except when subjects were placed on a cholesterol containing diet which significantly increased their TC from 138 mg/dl to 226 mg/dl before addition of cellulose (58).

With addition of cellulose, TC then decreased to 170 mg/dl (58).

Based on conflicting evidence for the use of soy hulls, it would be uncertain as to whether or not this soy fiber would be effective.

II. Fecal Fiber and Apparent Fiber Digestibility

Fecal fiber, expressed as grams per day and percent cell wall, was found to increase as the amount of fiber increased. Addition of a number of different types of dietary fiber have been found to increase the total amount of feces excreted daily including wheat bran, corn bran, soy hulls, oat bran, fruits and vegetables, cellulose and bagasse (34, 36, 37, 43, 48, 51, 53, 70). Addition of 8 to 39 g of wheat bran to the diets of men was found to significantly increase fecal weight in all studies (34, 36, 37, 51). Addition of 26 g of corn bran, soy hulls or 100 g of oat bran were found to significantly increase total fecal output (36, 43). When fruits and vegetables were added to the diets of healthy subjects, there was a consistent increase in total fecal excretion (48, 53, 70). Addition of bagasse (51) and cellulose (37) to the diets of subjects were both found to significantly increase total fecal exretion. Consumption of added wheat bran (34), a diet high in fruit and vegetable fiber (37), and added cellulose, hemicellulose, and pectin (72, 73) were all found to increase the amount of fecal fiber excreted. Addition of 14.2 g of cellulose (72) was found to significantly ($P<.05$) increase the amount of fecal fiber excreted.

Apparent fiber digestibility in the present study decreased with increasing amounts of dietary fiber up to the intermediate fiber

level and then increased when going to the high fiber level. Increased apparent fiber digestibility from the intermediate to the high fiber level was inexplicable when compared to results seen in the literature. Other research studies have found that as the amount of dietary fiber increased, the fiber digestibility decreased (31, 70, 73). Farrell et al. (34) found that by adding 12 g of wheat bran to the diets of men, fiber digestibility decreased from 80% to 55%. Kelsay et al. (70) found that as the amount of fiber added as fruits and vegetables increased, the digestibility decreased from 78% to 61%. Slavin et al. (73) when adding cellulose to the diets found that the high cellulose diet caused a decrease in apparent fiber digestibility of neutral detergent fiber from 61% to 38%.

Fiber in this study appears to be quite digestible and this may be due to the presence of hemicellulose in the fiber (see Appendix H). Fetzer et al. (72) found that hemicellulose was very digestible (76-90%), comparable to the figures found in the present study (70%-92%). Similar results were seen by Kelsay et al. (70) when the apparent digestibility of hemicellulose was measured on low fiber diets (95%) and high fiber diets(88%).

SUMMARY AND CONCLUSIONS

Results of feeding soy products such as soy hulls or purified soy polysaccharide fibers and its effects on plasma lipids and lipoproteins, fecal fiber, and apparent fiber digestibility are limited, particularly under controlled feeding conditions. The present study was designed to study the effects of three levels of a soy polysaccharide fiber added to a cholesterol-free liquid formula diet on plasma lipids and lipoproteins, fecal fiber, and apparent fiber digestibility in 22 young men. All subjects were randomly rotated through four treatments in four, eleven day periods under controlled, metabolic conditions at the metabolic facility of the Department of Human Nutrition and Foods. All diets were the same except for addition of the soy fiber at the levels of 20, 30, and 40 g per 1500 kilocalories. Subjects were weighed daily before the first meal. Fasting blood samples were taken on the morning prior to starting each new dietary period to determine plasma TC, TG, and HDL-C. Complete fecal collections were made and composited from the last five days of each eleven day period for fiber analysis.

When subjects were placed on the zero-cholesterol liquid formula diet, with or without fiber, there was a significant ($P<.05$) decrease in plasma total cholesterol (TC), low density lipoprotein-cholesterol (LDL-C), and total triglyceride (TG) levels. HDL-C values remained unchanged. When different levels of soy polysaccharide fiber were consumed, no changes were seen in

plasma TC, LDL-C, HDL-C, and TG due to fiber levels.

LDL-C/HDL-C and TC/HDL-C ratios were also significantly ($P<.05$) decreased when subjects were placed on the cholesterol-free diet while addition of fiber did not affect any further changes in LDL-C/HDL-C and TC/HDL-C ratios.

Addition of the fiber to the diets may not have been effective in influencing plasma lipids and lipoproteins for several possible reasons. One possible reason was that not as much dietary fiber was consumed as in the study by Munoz et al. (36). Furthermore, a zero-cholesterol diet may have made additional changes due to dietary fiber too small to detect. Additionally, the subjects may not have been on the diets long enough to influence plasma lipids or lipoproteins. One other possible reason was that processing of the fiber into the liquid formula diets may have, in some yet unkown manner, decreased the effectiveness of the fiber. Finally, normocholesterolemic subjects may be less responsive to a dietary change than hypercholesterolemic subjects, particularly in combination with a zero-cholesterol diet.

As found in the literature, as the levels of soy polysaccharide fiber increased in the diets, the amount of fiber excreted daily and the percentage of fiber excreted daily increased. Apparent fiber digestibility decreased up to the intermediate fiber level. Why the highest level of dietary fiber did not decrease the apparent fiber digestibility beyond the intermediate level of fiber is unkown.

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APPENDICES

APPENDIX A

DIET RANDOMIZATION TABLE

Subject	Period 1	Period 2	Period 3	Period 4
1	8109	8110	8111	8108
2	8110	8111	8108	8109
3	8109	8108	8111	8110
4	8110	8111	8109	8108
5	8111	8108	8110	8109
6	8108	8110	8111	8109
7	8111	8108	8109	8110
8	8110	8111	8108	8109
9	8111	8109	8110	8108
10	8110	8108	8111	8109
11	8110	8108	8109	8111
12	8110	8108	8109	8111
13	8109	8108	8111	8110
14	8111	8109	8108	8110
15	8109	8111	8110	8108
16	8108	8111	8109	8110
17	8109	8108	8110	8111
18	8110	8108	8111	8109
19	8110	8108	8109	8111
20	8111	8108	8110	8109
21	8110	8109	8108	8111
22	8108	8109	8110	8111

APPENDIX B

FOOD RECORD

FOOD AND DRUG INTAKE RECORD DAY: Sat Sun Mon Tues Wed (Circle One) CODE#____/____/____

(Record starts when you get up in the morning. Add night-time snacks, drinks, drugs.)

Food Code (leave blank)	Time	Location	Description of Food & Beverages (include preparation)	Amount	Description of Drug Taken	Amount
	7:30 a.m.	Home	Fried eggs in margarine	2 medium eggs 1 Tbsp. margarine	Marijuana - joint	4 hits
	7:30 a.m.	Home	Kroger Enriched White Bread, toast	2 slices	Bayer Aspirin	2 tablets
	7:30 a.m.	Home	Frozen Concentrate Orange Juice	8 ounces		
	7:30 a.m.	Home	Whole Milk	8 ounces		
	10:15 a.m.	Squires Snack Bar	Coke	12 ounces		
	10:15 a.m.	Squires Snack Bar	Wise Potato Chips - Plain	about 20 chips		
	12:30 p.m.	Cafeteria	Broiled ground beef hamburger	6 ounces (2)		
	12:30 p.m.	Cafeteria	Hamburger roll - white bread	2 rolls		
	12:30 p.m.	Cafeteria	Tomato catsup	4 Tbsp.		
	12:30 p.m.	Cafeteria	Dill pickle slices	6 slices		
	12:30 p.m.	Cafeteria	French Fries	25 strips		
	12:30 p.m.	Cafeteria	American cheese	2 slices		
	12:30 p.m.	Cafeteria	Lowfat milk	12 ounces		
	12:30 p.m.	Cafeteria	Raw red apple	1 medium		
	12:30 p.m.	Cafeteria	Commercial Chocolate Chip Cookies	4 cookies		
	3:00 p.m.	Department office	Coffee - instant	8 ounces		
	3:00 p.m.	Department Office	Cream, half-n-half	1 Tbsp.		
	6:30 p.m.	Restaurant	12" Cheese Pizza	6 slices		
	6:30 p.m.	Restaurant	Miller Lite Beer - 12 oz. can	3 cans		
	11:45 p.m.	Home	Skippy Smooth Peanut butter	3 Tbsp.		
	11:45 p.m.	Home	Graham Crackers, 2 1/2"	8 crackers		
	11:45 p.m.	Home	Chocolate Ice Cream	1 cup		
	12:30 a.m.	Home	Whole Milk, warmed	4 ounces		

Did you remember to record everything?

Please answer these questions.*

1. Have you had any exposure to pesticides, solvents or other chemicals today? Yes No Don't know
2. Was today's intake different from your usual habits? Yes No Don't know
3. Did you engage in any unusually strenuous exercise today? Yes No Don't know
4. Are you on a special diet? Yes No Don't know

*If you answered Yes to any above please explain on back.

APPENDIX B

FOOD RECORD (CONTINUED)

FOOD AND DRUG INTAKE RECORD DAY: Sat Sun Mon Tues Wed (Circle One) CODE# ____ / ____ / ____

(Record starts when you get up in the morning. Add night-time snacks, drinks, drugs.)

Food Code (leave blank)	Time	Location	Description of Food & Beverages (include preparation)	Amount	Description of Drug Taken	Amount

Please answer these questions.*

1. Have you had any exposure to pesticides, solvents or other chemicals today?
   Yes No Don't know
2. Was today's intake different from your usual habits? Yes No Don't know
3. Did you engage in any unusually strenuous exercise today? Yes No Don't know
4. Are you on a special diet?
   Yes No Don't know

*If you answered Yes to any above please explain on back.

Did you remember to record everything?

APPENDIX C

PRE-EXPERIMENTAL QUESTIONNAIRE

Name_____ Age_____

Address_____

Phone_____ Student #_____

1. Weight_____lbs.

   Height_____ft._____in.

2. Health History

   Have you, at any time, had any of the following conditions? (check where appropriate)

   diabetes____ irritable bowel syndrome____
   hypertension____ Crohn's____
   anemia____ diverticulosis____
   kidney disease____ colitis____
   liver disease____ other_____

3. Are you taking any prescribed medication on a regular basis?
   Yes____ No____

   If yes, specify the name of the medication and the reason for its use.

   _____

4. Do you supplement your diet with either vitamin(s) or mineral(s)?
   Yes____ No____

   If yes, how frequently do you take the supplement(s)? (check where appropriate)

   1/week____
   3/week____
   5/week____
   every day____
   other (explain)_____
   Name and type of supplements_____

   _____

5. Do you habitually use any of the following items? (check where appropriate)

   cigarettes____ marijuana or other social drugs____
   cigars____ coffee____
   pipe____ tea____
   chewing tobacco____
   beer, wine or other alcoholic beverages____

APPENDIX C

PRE-EXPERIMENTAL QUESTIONNAIRE (CONTINUED

6. Are you allergic to any specific food items or medications?
   Yes _____ No _____

   If yes, list those you are allergic to

   _____

7. How often do you normally have bowel movements?
   2 times or more a day ____
   1 time each day ____
   3 times each week ____
   5 times each week ____
   Other _____

8. What is the normal consistency of your stool?
   dry and firm ____
   moist and soft (retains shape) ____
   soft (loses shape) ____
   watery ____

9. Do you often have pain or difficulty in moving your bowels?
   Yes_____ No _____ Seldom_____

10. Do you exercise on a regular basis?
    Yes _____ No _____

    If yes, what activity do you participate in and how heavily are you involved?
    jogging _____
    biking _____
    tennis _____
    raquetball_____
    football_____
    other _____

11. Do you intend to leave town for more than 1 day during the period of September 28 to November 22? Yes_____ No_____

    Additional comments:

APPENDIX D

CONSENT FOR PARTICIPATION IN THE LIQUID FORMULA, BOWEL FUNCTION, FECAL COMPOSITION AND BLOOD PROFILES STUDY

DEPARTMENT OF HUMAN NUTRITION AND FOODS
VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
BLACKSBURG, VA. 24061

You are invited to participate in a study of four liquid formula diets. These diets consist of normal food ingredients, generally recognized vitamins and minerals. All ingredients have been approved as food additives by the Food and Drug Administration. We hope to learn more about the effects of these formulas on stool production and their effects on the digestion and absorption of nutrients from the gastrointestinal tract. You were selected for this study because of your willingness to participate, willingness to follow the rules of the project, and assumed good health.

What You Will Be Asked to Do

1. Starting September 28 or 29 and ending October 5 to 6, 1981 you will keep a daily record of all foods and beverages consumed. You will also collect all urine and feces during this period and record times of bowel movements and any physical discomfort experienced. You will consume an inert dye on the 5th day of this period and again on the 11th day and record and notify the investigators the time when the dye appears in your feces. Two small blood samples will be drawn by a Certified Medical Technician during this time.
2. You will taste various samples of the liquid formula diet at the beginning and end of the study and communicate your preferance.

APPENDIX D

CONSENT FOR PARTICIPATION IN THE LIQUID FORMULA,
BOWEL FUNCTION, FECAL COMPOSITION AND BLOOD PROFILES STUDY
DEPARTMENT OF HUMAN NUTRITION AND FOODS
VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
BLACKSBURG, VA. 24061 (CONTINUED)

3. Starting October 6 or 7 you will consume liquid formula diets which have all nutrients required for maintenance of good health for 44 days ending with the appearance of the dye in your feces from the final period. This will be from November 19 to November 21. These 44 days will be divided into four 11 day periods where you will consume different formulations of the basic liquid formula diet. You will not consume any other food during this period and you will be required to consume only the water supplied to you by the investigators. You will consume at least 2 bottles of the test formula at exactly 7:30 a.m., 12:00 p.m. and 5:30 p.m. each day and you will maintain you body weight by supplementing this diet with extra liquid formula preferably at meal times.
4. You will not drink alcohol, tea or coffee during the study. Over-the-counter, or social drugs may not be taken although aspirin or other analgesic may be used occasionally but the investigators must be informed. No use of tobacco products should occur during the study period. Sugarless gum may be chewed. If the above mentioned substances are taken for any reason the investigators must be informed.
5. During each 11 day liquid formula period you will be expected to supply the following:

APPENDIX D

CONSENT FOR PARTICIPATION IN THE LIQUID FORMULA,
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VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
BLACKSBURG, VA. 24061 (CONTINUED)

a. all urine

b. all feces

c. daily body weight

d. daily information concerning bowel habits and hunger

e. a small sample of blood (40-50 ml or 2 ounces) at the end of each period drawn by a Certified Laboratory Technician

6. You will be asked at the beginning of the study to fill out various forms related to your health and health habits. These and the tests of blood samples will be used to monitor your general health as a safety measure and to fulfill the objectives of the study.

Possible Risks

This is a low risk study in that the formulas are designed to meet nutritional needs of humans. However the formulas must be consumed slowly at spaced intervals to avoid the possibilities of gastrointestinal disturbances. The formulas might cause some bloating and/or gas formation. Small risks may be associated with the drawing of blood samples and there is a slight chance for food allergies. If formulas are left unrefrigerated, some rises of food poisoning are possible.

If you have an adverse reaction, contact Dr. Phyllis Bowen at the Department of Human Nutrition and Foods, Virginia Tech 961-5540 or

APPENDIX D

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VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
BLACKSBURG, VA. 24061 (CONTINUED)

evenings at 951-8518.

Possible Benefits

This study is designed for research purposes and is not expected to give benefits to the participants.

Due to the objectives of the study, alternative procedures are not appropriate.

Confidentiality

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. If you give us your permission by signing this document, we plan to disclose results of this study but not your name to the scientific community via scientific journal articles and to Ross Laboratory in order that the information obtained may contribute to total nutrition knowledge and may be put to practical use.

Compensation

Compensations for participation include the following: 1) provision of food for the liquid diet period of the study, 2) payment of $7.00 per day during participation in the liquid diet period and $3.00 per day during participation in the preliminary period; all compensation due to be received at the completion of the entire study. In addition, some subjects receive some sense of fulfillment for having

APPENDIX D

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VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
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made a personal contribution to science.

Rights and Responsibilities

It is your responsibility to report to the principal researcher or research staff any pre-existing medical problems and any medical problems that might arise during the course of the study. All prescripted medication and all other medication that you might need to take during the course of the study must be reported to the principal researcher or noted on daily forms that you will receive and fill out.

No compensation or medical treatment is available from Virginia Polytechnic Institute and State University if injury should be suffered as a result of this research.

You are free at anytime to withdraw your consent to discontinue participation in the project without prejudice.

You have a right to ask any question at anytime with an expectation to receive a truthful answer.

If you have any questions, we expect you to ask us. If you have any additional questions later, Dr. Phyllis Bowen, Department of Human Nutrition and Foods, 206 Wallace Hall, 961-5540, Dr. Forrest Thye, Department of Human Nutrition and Foods, 207 Wallace Hall, 961-6620 or Dr. Janette Taper, Department of Human Nutrition and Foods, 206 Wallace Hall, 961-5549 will be happy to answer them.

APPENDIX D

CONSENT FOR PARTICIPATION IN THE LIQUID FORMULA
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VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY
BLACKSBURG, VA. 24061 (CONTINUED)

You will be given a copy of this form to keep. You are making a decision whether or not to participate. YOUR SIGNATURE INDICATES THAT YOU HAVE AGREED TO PARTICIPATE HAVING READ THE PRECEEDING INFORMATION AND HAVING ALL QUESTIONS ANSWERED TO YOUR SATISFACTION.

I have read this consent document and have reviewed it with _____. All questions have been answered to my satisfaction. I agree to participate in the study.

_____ _____
Signature of Participant

_____
Signature of Investigator(s)

_____
Signature of Witness

Contact the following people if there are any problems or questions concerning your participation in this study.

Dr. Phyllis E. Bowen
Project Director
Department of Human Nutrition & Foods
VPI&SU
Blacksburg, VA 24061
703-961-5540

Dr. Milton P. Stombler
Chairman of University Internal Review Board for Human Research
339 Burruss Hall
Associate Dean, Research Division
VPI&SU
Blacksburg, VA 24061
703-961-5283

APPENDIX E

COMPOSITION OF ENSURE

Proximate Analysis of Low Residue Ensure (g/100 ml)

Protein	3.71
Fat	3.71
Carbohydrate	14.47
Ash	0.72
Kilocalories	107.59

Vitamins and Minerals (per 100 ml)

Vitamin A (IU)	263.71
Vitamin D (IU)	21.10
Vitamin E (IU)	3.16
Vitamin K_1 (mcg)	14.77
Vitamin C (mg)	16.03
Folic Acid (mcg)	21.10
Thiamine (mg)	0.16
Riboflavin (mg)	0.18
Vitamin B_6	0.12
Vitamin B_{12} (mcg)	0.63
Niacin (mg)	2.11
Choline (g)	0.05
Biotin (mcg)	16.03
Pantothenic Acid (mg)	0.53
Sodium (g)	0.08
Potassium (g)	0.13
Chloride (g)	0.10
Calcium (g)	0.05
Phosphorus (g)	0.05
Magnesium (mg)	21.10
Copper (mg)	0.10
Zinc (mg)	1.58
Iron (mg)	0.95

APPENDIX F

PROXIMATE ANALYSIS OF THE EXPERIMENTAL DIETS (G/100 ML)

Nutrient	Diets			
	Control	Low Fiber	Intermediate Fiber	High Fiber
Protein	3.76	3.88	3.88	3.92
Fat	3.76	3.76	3.71	3.63
Carbohydrate	14.39	15.23	15.68	16.33
Ash	0.68	0.72	0.76	0.76

APPENDIX G

MALE SUBJECT INFORMATION IN EXPERIMENT TO STUDY THE EFFECT OF THREE LEVELS OF SOY POLYSACCHARIDE FIBER IN ENSURE*

Subject Number	Age	Height	Weight
	Years	Meters	Kilograms
1	19	1.73	70.59
2	19	1.85	76.00
3	24	1.78	67.82
4	19	1.83	71.41
5	18	2.00	61.82
6	20	1.80	71.32
7	18	1.70	69.00
8	28	1.78	74.91
9	25	1.83	82.91
10	21	1.83	79.82
11	25	1.75	70.91
12	25	1.75	73.59
13	20	1.73	60.00
14	28	1.85	88.91
15	25	1.88	78.32
16	18	1.70	83.18
17	21	1.78	67.00
18	27	1.73	65.59
19	23	1.90	68.50
20	30	1.70	67.09
21	21	1.80	64.68
22	31	1.80	101.91

*Ensure of Ross Laboratories, Columbus, Ohio.

APPENDIX H

SOY FIBER COMPOSITION

	Fiber Methodology			
	Southgate		Van Soest	
Neutral Detergent Fiber	-[1]	40.0[1]	25.0[2]	26.0[3]
Hemicellulose	59.0	(30.0)	-	-
Cellulose	16.0	(10.0)	-	-
Lignin	-	0.5	-	-
Protein	12.0	12.0	-	-
Ash	5.5	5.5	-	-
Fat	1.0	1.0	-	-
Total	93.5	59.0	25.0	26.0

[1]Data reported by Ross Laboratories before product formulation from analysis supplied by Ralston Purina.

[2]Present value reported in personal communication.

[3]Results obtained from laboratory analysis at Virginia Tech.

APPENDIX I

SUBJECT WEIGHT CHANGE ON THE EXPERIMENTAL DIETS (KILOGRAMS): INDIVIDUAL DATA

Subject Number	Control	Low Fiber	Intermediate Fiber	High Fiber
1	-0.3	-0.1	-2.7	-0.8
2	-0.4	-1.3	+0.8	+0.3
3	+0.3	+0.8	+1.3	0.0
4	-0.9	-0.8	+1.2	-0.5
5	-1.9	-0.5	-	-
6	+0.6	+0.4	+2.0	+0.3
7	-0.5	-0.5	0.0	-
8	-0.6	-	-	0.0
9	-1.0	-0.9	-0.4	-0.3
10	+0.2	-0.2	-0.5	-0.3
11	-0.9	+0.2	-0.2	-0.1
12	+0.2	-0.3	-0.2	-2.1
13	-0.2	+0.5	-0.1	+1.1
14	-2.8	-0.9	-0.9	+1.0
15	-1.2	-0.8	-1.1	-0.6
16	+0.6	-	-1.5	+0.1
17	-2.4	-0.4	-0.7	0.0
18	-0.4	-0.4	+0.8	-0.3
19	+0.4	-1.0	0.0	+3.2
20	+3.2	0.0	-	-
21	-0.2	+1.0	0.0	-
22	-0.3	-1.2	-0.8	+0.3
Mean	-0.6	-0.2	-0.1	+0.1

APPENDIX J

PLASMA TOTAL CHOLESTEROL ON EXPERIMENTAL DIETS (MG/DL): INDIVIDUAL DATA

Subject Number	Pre-Study Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study Self-Selected
1	170	104	113	128	120	139
2	109	95	103	99	99	126
3	135	117	101	108	100	125
4	218	140	165	170	168	214
5	171	120	94	-	-	-
6	180	158	145	137	137	163
7	157	131	115	136	-	-
8	156	139	-	-	125	-
9	180	135	133	156	137	187
10	138	131	123	126	142	160
11	202	178	159	175	193	211
12	155	116	121	133	116	165
13	205	134	135	140	121	169
14	157	135	135	133	128	165
15	160	135	116	137	125	138
16	157	132	122	122	116	205
17	177	123	129	132	132	225
18	158	147	134	135	145	200
19	153	128	151	153	145	174
20	154	129	138	-	-	-
21	160	122	132	125	-	151
22	165	136	140	147	135	187
Mean±SEM	164±5	131±4	129±4	136±4	132±5	172±7

APPENDIX K

PLASMA TOTAL TRIGLYCERIDES ON EXPERIMENTAL DIETS (MG/DL): INDIVIDUAL VALUES

Subject Number	Pre-Study, Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study, Self-Selected
1	-	127	89	73	82	93
2	44	72	30	56	42	48
3	47	43	32	29	37	50
4	151	53	80	85	52	34
5	69	42	52	-	-	-
6	74	44	52	68	63	35
7	106	62	42	73	-	-
8	48	72	-	-	86	-
9	62	35	36	36	26	50
10	122	128	53	62	68	55
11	64	58	56	46	83	40
12	57	34	65	70	71	56
13	49	64	63	67	88	102
14	81	73	93	69	66	48
15	-	59	72	60	116	59
16	154	151	-	130	107	-
17	137	66	79	75	98	141
18	129	46	76	97	51	75
19	71	70	53	52	55	52
20	74	100	83	-	-	-
21	76	58	75	50	-	60
22	-	128	106	108	97	95
Mean±SEM	85±8	72±7	64±5	69±6	72±6	64±7

APPENDIX L

PLASMA LDL-C ON EXPERIMENTAL DIETS (MG/DL): INDIVIDUAL VALUES

Subject Number	Pre-Study, Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study, Self-Selected
1	-	55	57	83	72	80
2	60	44	51	51	51	65
3	74	66	51	64	45	60
4	132	79	112	116	123	143
5	111	71	46	-	-	-
6	118	112	100	93	97	105
7	104	84	82	74	-	-
8	108	84	-	-	84	-
9	106	95	97	97	94	104
10	75	71	82	82	91	92
11	127	111	97	116	119	110
12	117	82	72	76	75	108
13	158	95	93	96	69	105
14	116	94	91	94	73	113
15	-	86	71	83	67	71
16	97	65	-	62	58	-
17	111	72	81	93	78	156
18	106	104	82	64	89	130
19	102	75	103	118	107	111
20	112	77	82	-	-	-
21	112	74	80	74	-	84
22	-	84	96	82	76	-
Mean±SEM	108±5	81±4	81±4	85±4	82±5	102±7

APPENDIX M

PLASMA HDL-C ON EXPERIMENTAL DIETS (MG/DL): INDIVIDUAL VALUES

Subject Number	Pre-Study, Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study, Self-Selected
1	27	24	38	30	32	40
2	40	37	46	37	40	51
3	52	42	44	38	48	55
4	56	50	37	37	35	64
5	46	41	38	-	-	-
6	47	37	35	30	27	51
7	32	35	25	47	-	-
8	38	41	-	-	24	-
9	62	33	29	52	38	73
10	39	34	30	32	37	57
11	62	55	51	50	57	93
12	27	27	36	43	27	46
13	37	26	29	31	34	44
14	25	26	25	25	42	42
15	37	37	31	42	35	55
16	29	37	-	34	37	36
17	39	38	32	24	34	41
18	26	34	37	52	46	55
19	37	39	37	25	27	53
20	27	32	39	-	-	-
21	33	36	37	41	-	55
22	27	26	23	43	40	-
Mean±SEM	38±2	36±2	35±2	38±2	37±2	54±3

APPENDIX N

LDL-C/HDL-C RATIOS ON EXPERIMENTAL DIETS: INDIVIDUAL VALUES

Subject Number	Pre-Study, Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study, Self-Selected
1	2.4	2.3	1.5	2.8	2.2	2.0
2	1.5	1.2	1.1	1.4	1.3	1.3
3	1.4	1.6	1.2	1.7	0.9	1.1
4	2.4	1.6	3.0	3.1	3.5	2.2
5	2.4	1.7	1.2	-	-	-
6	2.5	3.0	2.9	3.1	3.6	2.0
7	3.3	2.4	3.3	1.6	-	-
8	2.8	2.0	-	-	3.5	-
9	1.7	2.9	3.3	1.9	2.5	1.4
10	1.9	2.1	2.7	2.6	2.4	1.6
11	2.0	2.0	1.9	2.3	2.1	1.2
12	4.3	3.0	2.0	1.8	2.8	2.3
13	4.3	3.6	3.2	3.1	2.0	2.4
14	4.6	3.6	3.6	3.8	1.7	2.7
15	-	2.3	2.3	2.0	1.9	1.3
16	3.3	1.8	-	1.8	1.6	-
17	2.8	1.9	2.5	3.9	2.3	3.8
18	4.1	3.0	2.2	1.2	1.9	2.4
19	2.8	1.9	2.8	4.7	4.0	2.1
20	4.1	2.4	2.1	-	-	-
21	3.4	2.0	2.2	1.8	-	1.5
22	-	3.2	4.2	1.9	1.9	-
Mean±SEM	2.9±0.2	2.4±0.2	2.5±0.2	2.4±0.2	2.3±0.2	2.0±0.2

APPENDIX O

TC/HDL-C RATIOS ON EXPERIMENTAL DIETS: INDIVIDUAL VALUES

Subject Number	Pre-Study, Self-Selected	Control	Low Fiber	Intermediate Fiber	High Fiber	Post-Study, Self-Selected
1	6.3	4.3	3.0	4.3	3.8	3.5
2	2.7	2.6	2.2	2.7	2.5	2.5
3	2.6	2.8	2.3	2.8	2.1	2.3
4	3.9	2.8	4.5	4.6	4.8	3.3
5	3.7	2.9	2.5	-	-	-
6	3.8	4.3	4.1	4.6	5.1	3.2
7	4.9	3.7	4.6	2.9	-	-
8	4.1	3.4	-	-	5.2	-
9	2.9	4.1	4.6	3.0	3.6	2.6
10	3.5	3.8	4.1	3.9	3.8	2.8
11	3.3	3.2	3.1	3.5	3.4	2.3
12	5.7	4.3	3.4	3.1	4.3	3.6
13	5.5	5.2	4.7	4.5	3.6	3.8
14	6.3	5.2	5.4	5.3	3.0	3.9
15	4.3	3.6	3.7	3.3	3.6	2.5
16	5.4	3.6	-	3.6	3.1	5.7
17	4.5	3.2	4.0	5.5	3.9	5.5
18	6.1	4.3	3.6	2.6	3.2	3.6
19	4.1	3.3	4.1	6.1	5.4	3.3
20	5.7	4.0	3.5	-	-	-
21	4.8	3.4	3.6	3.0	-	2.8
22	7.5	5.2	6.1	3.4	3.4	-
Mean±SEM	4.6±0.3	3.4±0.2	3.8±0.2	3.8±0.2	3.8±0.2	3.8±0.2

APPENDIX P

FECAL FIBER ON EXPERIMENTAL DIETS (G/D): INDIVIDUAL DATA

Subject Number	Control	Low Fiber	Intermediate Fiber	High Fiber
1	0.55	1.77	0.23	0.20
2	-	0.64	-	-
3	0.16	1.18	3.42	4.43
4	2.85	3.16	4.65	1.73
5	0.24	2.30	-	-
6	1.71	1.19	4.84	0.50
7	0.28	0.42	0.31	-
8	0.14	-	-	1.00
9	0.00	3.82	6.03	5.61
10	0.29	0.62	5.71	0.00
11	0.40	3.50	4.27	6.34
12	0.64	0.00	0.62	1.90
13	0.24	0.00	0.48	5.44
14	0.14	3.56	4.35	5.87
15	0.26	0.06	0.23	0.48
16	1.78	-	3.39	5.72
17	0.37	0.25	0.02	0.86
18	0.40	0.39	4.66	0.38
19	0.00	0.36	0.31	0.76
20	0.53	0.87	-	-
21	0.17	3.31	5.51	-
22	0.00	0.43	1.56	2.24
Mean	0.53	1.39	2.81	2.56

APPENDIX A

FECAL FIBER ON EXPERIMENTAL DIETS (%): INDIVIDUAL DATA

Subject Number	Control	Low Fiber	Intermediate Fiber	High Fiber
1	2.43	8.63	1.26	0.80
2	-	15.42	-	-
3	0.80	4.57	7.47	15.30
4	12.94	16.03	20.16	5.29
5	1.77	15.36	-	-
6	7.67	4.99	13.86	1.69
7	2.49	3.77	2.17	-
8	0.80	-	-	3.81
9	0.00	17.80	25.51	17.36
10	1.69	2.97	16.40	0.00
11	2.57	13.77	14.54	16.61
12	2.91	0.00	2.45	6.03
13	1.59	0.00	2.59	20.73
14	0.73	14.84	17.32	18.87
15	1.92	0.45	1.07	0.98
16	9.80	-	18.89	18.27
17	1.58	2.07	0.13	4.12
18	2.28	2.74	16.88	1.68
19	0.00	1.60	1.13	2.45
20	4.04	4.50	-	-
21	1.72	15.37	18.39	-
22	0.00	3.73	5.95	8.07
Mean	2.84	7.43	10.34	8.36

APPENDIX R

APPARENT FIBER DIGESTIBILITY ON EXPERIMENTAL DIETS (%): INDIVIDUAL DATA

Subject Number	Control	Low Fiber	Intermediate Fiber	High Fiber
1	89.79	69.15	97.11	98.38
2	-	86.67	-	-
3	97.04	80.88	63.43	66.32
4	57.53	53.86	56.22	87.39
5	93.46	52.30	-	-
6	72.07	80.95	52.19	96.25
7	94.13	93.07	96.77	-
8	96.76	-	-	92.14
9	100.00	27.09	32.53	55.35
10	94.83	88.96	40.57	100.00
11	91.25	31.05	50.83	48.50
12	89.99	100.00	93.73	84.69
13	94.52	100.00	94.30	55.20
14	97.05	33.25	49.99	55.34
15	94.37	98.74	97.30	96.11
16	64.10	-	56.41	52.24
17	89.65	94.50	99.81	92.81
18	92.02	90.95	44.22	96.59
19	100.00	93.47	96.43	93.84
20	83.29	76.68	-	-
21	96.29	36.83	37.26	-
22	100.00	92.00	83.21	83.32
Mean	89.91	74.02	69.02	79.67

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THE EFFECT OF THREE LEVELS OF A SOY POLYSACCHARIDE FIBER ON PLASMA LIPIDS, FECAL FIBER, AND APPARENT FIBER DIGESTIBILITY

By Ellen Kennedy

(ABSTRACT)

Twenty-two adult male subject were randomly assigned to four complete liquid diets at four different fiber levels; one without added soy polysaccharide fiber which served as a control. All subjects were rotated through four, eleven day controlled feeding periods. Complete fecal samples were collected and composited from the last five days of each eleven day period for fiber analysis. Fasting blood samples were taken on the morning prior to starting each new dietary period for plasma total cholesterol (TC), total triglycerides (TG), and high density lipoprotein-cholesterol (HDL-C) analyses. Low density lipoprotein-cholesterol (LDL-C) was determined by calculation.

Increased mean dietary fiber intake led to a significant ($P<.05$) increase in fecal fiber from 0.4 g per day to 2.9 g per day. Mean apparent fiber digestibility significantly ($P<.05$) decreased from 92% on the control diet to 70% on the intermediate fiber diet. There was a nonsignificant increase in apparent fiber digestibility from the intermediate fiber diet (70%) to the high fiber diet (76%). When subjects went from self-selected diets to the zero-cholesterol, liquid formula diets, significant ($P<.05$) decreases were seen in plasma TC (from 164 mg/dl to a range of 129 to 136 mg/dl), LDL-C (from

108 mg/dl to a range of 81 to 85 mg/dl), TG (from 85 mg/dl to a range of 64 to 72 mg/dl), TC/HDL-C ratios (from 4.6 to an average of 3.8), and LDL-C/HDL-C ratios (from 2.9 to an average of 2.4). HDL-C levels did not change from the self-selected diet to the zero-cholesterol liquid formula diets (from 38 mg/dl to a range of 35 to 38 mg/dl). The addition of 20, 30, and 40 g of soy polysaccharide fiber to the low residue control diet did not result in any further changes in plasma TC (131 mg/dl versus 129 mg/dl, 136 mg/dl, and 132 mg/dl, respectively), TG (72 mg/dl versus 64 mg/dl, 69 mg/dl, and 72 mg/dl, respectively), HDL-C (36 mg/dl versus 35 mg/dl, 38 mg/dl, and 37 mg/dl, respectively), LDL-C (81 mg/dl versus 81 mg/dl, 85 mg/dl, and 82 mg/dl, respectively), TC/HDL-C ratios (3.8 versus 3.9, 3.8, and 3.8, respectively), and LDL-C/HDL-C ratios (2.4 versus 2.5, 2.4, and 2.3, respectively). At the end of the study when subjects went back to self-selected diets plasma TC, LDL-C, and HDL-C all significantly ($P<.05$) increased.