FOR PRIVATE CIRCULATION For the use of a Registered Medical Practitioner only

QUARTERLY MEDICAL REVIEW

Vol. 56, No. 4 **October - December 2005**

DIETARY FIBER IN HEALTH AND DISEASE

CONTENTS

Published and Issued by

RAPTAKOS, BRETT & CO. LTD., WORLI, MUMBAI 400 030.

QUARTERLY
MEDICAL REVIEW

Vol. 56, No. 4 October - December 2005

"DIETARY FIBER IN HEALTH AND DISEASE"

G. SUBBULAKSHMI*

INTRODUCTION

Dietary fiber has long been a topic both for clinicians and nutritionists. Lately there has been a growing interest in dietary fiber resulting from the works of Trowell and Burkitt. The fiber hypothesis proposed by them suggested that high dietary fiber content helps protect against certain diseases that are common in the Western World. Faulty diets are found to play an important role in the development of heart disease, hypertension, cancer, osteoporosis and other chronic diseases. Diets high in animal fat and low in vegetables and fruits are strongly related to the development of these and other non communicable diseases (NCDs).

In today's fast world many changes have occurred in the time honored food habits of almost all the cultures. The displacement of fiber-rich plant foods by novel dietary staples, introduced during the Neolithic and Industrial periods, was instrumental in changing the diets that our species had traditionally consumed—a diet that would have almost always been high in fiber.

Food staples and food-processing procedures introduced during these periods have fundamentally altered 7 crucial nutritional characteristics of ancestral hominin diets: *1*) glycemic load, *2*) fatty acid composition, 3) macronutrient composition, *4*) micronutrient density, *5*) acid-base balance, *6*) sodium-potassium ratio, and *7*) fiber content. The evolutionary collision of our ancient genome with the nutritional qualities of recently introduced foods may underlie many of the chronic diseases of Western civilization[1].

The "western" diet is defined by low fiber and high fat, sucrose and animal protein intakes and the pattern of disease that accompanies it. People in Japan for example are known to live longer than anyone else in the world. However, Japanese in United States who have adopted the western diet were found to have a higher incidence of NCDs as compared to their counterparts in Japan[2]. The human body functions best on a diet that is low in sodium, contains a moderate amount of protein and is high in fiber, complex carbohydrates and vegetables and fruits. Studies that tracked how disease patterns and rates change as people adopt a "western" style of eating provided evidence for the benefits of high fiber, low fat, low sodium diets. A recent analysis of mortality trends suggests that large increases in non-communicable diseases (NCDs) have occurred in developing countries, particularly those in rapid transition (e.g. Brazil, China and India). According to these estimates, at least 40% of all deaths in developing countries are attributable to NCDs (vs.75% in industrialized countries)[3].

The rise in CVD is a reflection of a significant change in diet habits, physical activity levels, and tobacco consumption worldwide as a result of industrialization, urbanization, economic development and food

*Director, Research Unit, College of Home Science, Nirmala Niketan, Mumbai-20.
Email: subbulakshmigurumurthy@yahoo.com

market globalization. People are consuming a more energy-dense, nutrient-poor diet and are less physically active. These are no longer only diseases of the developed world: some 80% of all CVD deaths world-wide took place in developing, low and middle-income countries, while these countries also accounted for 86% of the global CVD disease burden. In developing countries people are being exposed to these risk factors for longer periods and a high proportion of disease takes place in people of working age.

A complex range of factors interact to determine the nature and course of this epidemic in developing societies like India which are rapidly urbanizing. Increases in energy intake, dramatic increases in fat intake along with increased levels of sedentary life style are the rapid quantitative changes in developing countries indicating an increase in per capita availability of food which is also accompanied by qualitative changes in the diet. Food balance data from the Food and Agriculture Organization (FAO) show that the change in energy intake in Asian countries has been small, but there have been large changes in consumption of animal products, sugars, fats and refined carbohydrates with decreased consumption of coarse grains. The net effect has been a marked shift in the diet with energy from fat (both animal and vegetable) increasing each year and a decline in complex carbohydrates and dietary fiber. Data from India show that higher-income groups consumed a diet with 33% of the energy from fat while the very low-income groups consumed only 17% energy from fat[4].

What are Dietary Fibers?

The American Association of Cereal Chemists (AACC) defines dietary fiber as **"The edible parts of plants or analogous carbohydrates that are resistant to digestion and absorption in the human small intestine with complete or partial fermentation in the large intestine. Dietary fiber includes polysaccharides, oligosaccharides, lignin, and associated plant substances. They promote beneficial physiological effects including laxation, and / or blood cholesterol attenuation, and/or blood glucose attenuation."** The non-starch polysaccharides (NSP) include celluloses, hemicelluloses such as arabinoxylans and arabinogalactans, pectins, modified celluloses, fructans (oligomers and polymers of fructose, i.e. inulin), gums, and mucilages.

At various times in history, links between insufficient dietary fiber consumption and constipation, diverticular disease, hiatus hernia, appendicitis, varicose veins, hemorrhoids (piles), diabetes, obesity, coronary heart disease, cancer of the large bowel, gallstones, duodenal ulcers, breast cancer, and blood clotting have been hypothesized. Of these, three physiological impacts characteristic of insufficient dietary fiber consumption have been proven to be constipation, increased risk of coronary heart disease, and increased fluctuation of blood glucose and insulin levels[5].

COMPONENTS OF DIETARY FIBER

COMPONENT	SOURCE
Cellulose	All food plants
Hemicellulose	All food plants, especially cereal bran
Pectin	Mainly fruits
Lignin	Mainly cereals and 'woody' vegetables
Gums and some food thickeners	Food additives in processed foods

RECOMMENDED FIBER INTAKES THROUGH THE LIFE CYCLE

Recommendations for adult dietary fiber intake generally fall in the range of **20 to 35 g/day**. Other recommendations for dietary fiber intakes are based on energy intake and are 10 to 13 g of dietary fiber per 1000 kcal. Nutrition facts labels use 25 g dietary fiber per day for a 2, 000 kcal/day diet or 30 g/day for a 2,500 kcal/day diet as goals for American intake. Two to three servings of whole grains, five servings of fruits and vegetables a day and one or two servings of legumes every week would supply the required amount of fiber.

Dietary fiber has important health benefits in childhood, especially in promoting normal laxation. Currently, children consume amounts of dietary fiber that appear to be inadequate for optimal health promotion and disease prevention. No published studies have defined desirable fiber intakes for infants and children younger than 2 years. Until there is more information about the effects of dietary fiber in the very young, a rational approach would be to introduce a variety of fruits, vegetables, and easily digested cereals as solid foods in the diet. Children aged 3-18 need less fiber than adults, and they need different amounts at different ages. It is prudent to recommend that **children older than 2 years of age increase dietary fiber intake to an amount equal to or greater than their age + 5 g/day.** According to the "age + 5" rule dietary fiber intake would increase from 8 g/day at age 3 years to 25 g/day by age 20 years. After age 20, dietary fiber levels of 25 to 35 g/day are recommended. Dietary fiber intake should be increased gradually in childhood by increasing consumption of a variety of fruits, vegetables, legumes, cereals, and other whole-grain products. Although very high fiber intake in childhood could have adverse effects, the potential health benefits of a moderate increase in dietary fiber substantially outweigh the possible risks[6].

Specific recommendations for the elderly have not been published, although a safe recommendation would be intakes of 10 to 13 g dietary fiber per 1,000 kcal. Adequate fluid intake should be advised when recommendations for dietary fiber are made. Caution should be used when recommending fiber to those with gastrointestinal diseases, including constipation.

The usual daily intake of dietary fiber in Europe and the USA amounts to only 15-20 g, while health authorities and nutrition societies recommend a reference value of at least 30 g. Dietary fibers are applied as food-integrated, as supplement and as purified substances.

Daily Requirement of Dietary Fiber in Different Age Groups

Age	Adequate Intake (g/day)	
	Male	Female
1-3 years	19	19
4-8 years	25	25
9-13 years	31	26
14-18 years	38	26
19-30 years	38	25

Age	Adequate Intake (g/day)	
	Male	Female
31-50 years	38	25
51-70 years	30	21
> 70 years	30	21
Pregnancy	-	28
Lactation	-	29

SOURCES OF DIETARY FIBER

Dietary fiber is found in foods of plant origin only - cereals, vegetables, fruit, dried peas, beans, lentils and nuts. Because of their water content, fruits and vegetables are more dilute sources (1.5 - 5 %). Legumes, nuts and dried fruits are higher in fiber (greater than 7%). Foods such as meat, fish, eggs, milk and cheese, table sugar and alcohol do not provide dietary fiber.

Servings of commonly consumed grains, fruits, and vegetables contain only 1 to 3 g of dietary fiber[7]. Though the vegetarians feel that they consume adequate vegetables and fruits in their diet, 73% of individuals had a mean fiber intake below 20 g/day[8].

Fiber content of certain foods per 100 gms

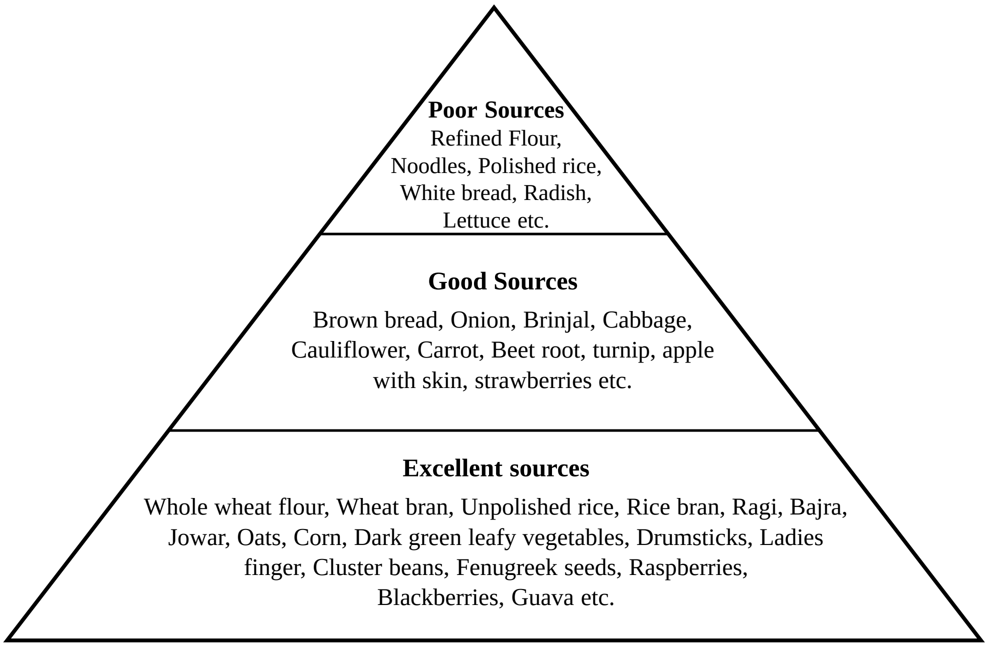
Food stuff	Fiber (gms)
Ragi	3.6
Rice bran	4.3
Green gram whole	4.1
Moth beans	4.5
Amaranath	6.1
Drumstick	4.8
Guava	5.2
Passion fruit	9.6
Bran bread	8.5
White bread	1.9
Cornflakes	2.0
Oatmeal	10.6
Wheat flakes	9.0
Oat bran, raw	6.6
Rice, raw (brown)	3.5
Rice, raw (white)	1.0-2.8
Wheat bran	15.0
Apple (with skin)	2.8
Apricots (dried)	7.8

Food stuff	Fibre (gms)
Figs (dried)	9.3
Banana	2.8
Orange	3.1
Kiwi fruit	3.4
Pears (raw)	2.6
Prunes (dried)	7.2
Raisins	5.3
Broccoli, raw	7.7
Brussels sprouts, (cooked)	2.6
Cabbage (white) raw	2.4
Cauliflower, raw	2.4
Sweet potato, cooked	3.0
Tomatoes, raw	1.3
Coconut, raw	9.0
Peanuts, dry-roasted	8.0
Pistachios	10.8
Tahini	9.3
Tofu	1.2

Source: Provisional table on the dietary fiber content of selected foods (Washington, D.C.: U.S. Department of Agriculture, 1988).

The food pyramid, showing a healthy diet, became familiar to many. It recommends 6+ servings of cereals and breads a day, 5+ of fruit and vegetables, 2 servings of milk, cheese and yoghurt, and one of meat, fish, chicken, beans, nuts or eggs. Food at the apex of the pyramid, fats, oils, sugars, alcohol and salt are usually components of foods in lower levels so no servings are recommended. Similarly, the fiber pyramid could be a good guide for disease prevention and health promotion.

FIBER PYRAMID



Fruit and vegetable intake varies considerably among countries, in large part reflecting the prevailing economic, cultural and agricultural environments. WHR 2002 analysis assessed the levels of mean dietary intake of fruit and vegetables (excluding potatoes) in each region, measured in grams per person per day. Low intake of fruit and vegetables is estimated to cause about 19% of gastrointestinal cancer, and about 31% of ischemic heart disease and 11% of strokes worldwide. 2.7 million deaths are attributable to low fruit and vegetable intake. Of the disease burden attributable to low fruit and vegetable intake, more than four fifths is from heart diseases and the balance from cancers.

TYPES OF DIETARY FIBER

Dietary fiber is a heterogeneous group of substances which have only one common characteristic: the non-digestability in the small bowel. With one exception all fibers are carbohydrates (poly- or disaccharides). Some fibers are water-soluble – **Soluble fibers** while others are insoluble and called **Insoluble fibers**. This property is associated with physiological effects[9]. Insoluble fiber comes from the remnants of plant cell walls while soluble fiber is present in the space between the cell walls called the middle lamella. Insoluble fiber is composed of cellulose, hemicellulose and lignin. Cellulose, hemicellulose and lignin are structures, pseudo crystalline polymers held together by strong molecular forces of hydrogen bonding that are not disrupted by water and are therefore insoluble in gastrointestinal fluid and therefore poorly fermented by intestinal flora.

Soluble fibers are composed of complex polysaccharides such as beta glucans, pectins, gums and some hemicelluloses. Soluble fiber is hydrophilic, non crystalline and easily wetted by the aqueous gastrointestinal fluid forming viscous colloidal dispersions or gels when hydrated and is extensively fermented by intestinal flora[10].

Foods high in soluble fiber include oat bran, oatmeal, beans, peas, rice bran, barley, citrus fruits, strawberries and the edible part of apples. Oat bran has the most soluble fiber of any grain; however, the whole oat grain is a good source of insoluble fiber as well. Fruit pectin, for example, is a highly solube fiber. Psyllium seed, the commonest source of bulk laxatives, contains fiber that is moderately soluble. Wheat bran consists of relatively insoluble fiber that is most readily evident as "roughage". Although all fiber adds bulk to bowel movements, the chemical effects of the different types of fiber can be opposite. Foods high in insoluble fiber include wholewheat breads, wheat cereals, wheat bran, rye, rice, barley, most other grains, cabbage, beets, carrots, Brussels sprouts, turnips, cauliflower and apple skin[11]

The fiber available from rice and wheat is predominantly insoluble while that from fruits and vegetables is mainly soluble; hence while total fiber intake probably changed little, in the old world agriculture generally increased the insoluble: soluble dietary fiber ratio[12].

Resistant starch

While not traditionally thought of as fiber, resistant starch acts in a similar way. Resistant starch, the sum of starch and starch-degradation products not digested in the small intestine[13], contributes to the pool of microbial substrate or dietary fiber reaching the large intestine. It is found in many unprocessed cereals and grains, firm bananas, potatoes and lentils, and is added to bread and breakfast cereals as Hi-Maize. It can also be formed by cooking and manufacturing processes such as snap freezing. Resistant starch is also important in bowel health. Bacteria in the large bowel ferment and change the resistant starch into short-chain fatty acids, which are important to bowel health and may protect against cancer. These fatty acids are also absorbed into the bloodstream and may play a role in lowering blood cholesterol levels.

Legumes are a primary source of resistant starch, with as much as 35% of legume starch escaping digestion[14]. Small amounts of resistant starch are produced by the processing and baking of cereal and grain products. The amount of resistant starch in a typical Western diet is not known and meaningful tables of the resistant starch content of foods are not available.

Different Fibers and their properties

Types of Fiber	Physicochemical property	Physiological effect
Resistant starch, β-Glucans, Pectin, Guar	Fermentation	Energy source. Increase in biomass. Short chain fatty acid production Reduction in pH of colon (inhibition of 7-α dehydroxylase), Anti-neoplastic activity of butyrate
Non-fermentable portion of hydrocolloids *e.g.* Cellulose, Arabinoxylans, Algal hydrocolloids	Water Holding Capacity	Increased stool bulk Shorter gut transit times
Pectin, Guar	Viscosity	Delayed gastric emptying. Slower transit time through small bowel
Guar, Locust bean gum, Alginate	Gel Formation	Reduced rate of nutrient absorption (*eg.* glucose, bile acids)
Hydrocolloids with large hydrophobic surface area *e.g.* β-Glucans, Arabinoxylans, Methyl cellulose	Binding of organic molecules	Binding of bile acids, carcinogens and mutagens

Addressing the Australian Gastroenterology Week conference in Adelaide, Dr Graeme McIntosh said researchers had already identified some new dietary fibers, including a specific oligosaccharide that behaves like an antibiotic. He said while an antibiotic killed the bacteria, *mannan oligosaccharides* performed a similar function by absorbing certain pathogens (gastrointestinal bugs) and *oligosaccharides* had been used successfully to treat disorders such as diarrhea. Some *oligosaccharides* had already been commercially developed in Europe and the United States with the manufacturer claiming it improved digestive tract function and primed the immune system. The health benefits of fiber necessitate improvement in food labeling standards to provide more reliable measurements of fiber to assist consumers with food choices.

MECHANISM OF ACTION OF SOLUBLE FIBERS

Although studied extensively, the mechanism by which soluble fibers like oat beta-glucan reduces cholesterol is unclear. However, it has been demonstrated that development of viscosity in the gut, is an essential precursor[15]. Because soluble fibers like beta-glucan form a hydrocolloid, they have the capacity to form highly viscous solutions at low concentrations in vivo and this can lead to binding and increased excretion of bile acids, reduced absorption of fat and cholesterol, and a reduced absorption rate of available carbohydrates. Any or all of these events can result in attenuated cholesterol levels. Oat beta-glucan may also promote cholesterol clearance from the plasma via reverse cholesterol transport. This also suggests that soluble fibers like beta-glucan are likely to be more effective when taken with meals[16]. When these viscous fibers are added to a diet, the rate of glucose appearance in the blood is slowed, and insulin secretion is subsequently decreased. These beneficial effects on blood glucose and insulin concentrations are most evident in individuals who have diabetes mellitus.

For most soluble fiber sources, the potential mechanisms are the following:

- Alter energy intake and gastric emptying
- Alter emulsification of dietary fat in the conditions prevailing in the stomach and small intestine,
- Alter the secretion rate of pancreatic enzymes and reduce the activity of gut lipases
- Slow and/or reduce the intestinal absorption of dietary fatty acids and cholesterol as well as glucose absorption and insulin response
- Bind bile salts in the small intestine and as a result increase ileal and fecal excretion of fat, cholesterol and bile salts.
- Alter chylomicron secretion and thus, postprandial lipid and lipoprotein metabolism.
- Alter hepatic levels of LDL receptor. Most soluble viscous dietary fiber sources lower fasting plasma and LDL cholesterol after chronic intake.
- Favorably alter risk factors such as hyperinsulinemia or thrombotic indices.

Depending on the particular type of fiber, some mechanisms can have more or less importance[17].

DIETARY FIBER CONTENT OF SELECTED FRUITS AND VEGETABLES

Food	Serving	Soluble fiber	Insoluble fiber
Apple	1	0.84	1.96
Banana	1 medium	0.64	1.36
Pear	1	1.00	2.00

Effect of processing on fiber

The industrial era and food processing techniques have further distanced nutrition from its primate and Paleolithic antecedents. Roller-milling has reduced the fiber content of cereal-based foods so that total fiber intake has plummeted to levels much below that consumed by agriculturists, hunter-gatherers, or primates.

There is, however, de-polymerization of the dietary fiber polysaccharides where degradation is related to the severity of the heat treatment. Souring, freezing and mild microwave treatments have no effects. The viscosity is in general related to the extent of polymerization. Microwave treatment has different effects on various cultivars of green beans, and the addition of salt (NaCl and $CaCl_2$) to the boiling water changes the physico-chemical properties of soluble fiber in carrots, depending on the cation. The higher viscosity of the soluble fiber in raw carrots may partly explain the lower glucose and hormonal responses observed in healthy subjects when compared with blanched and microwave-cooked carrots.

Dietary Fiber in Health and Disease

Accumulating evidence suggests that dietary fiber could help prevent major diseases such as CVD and certain cancers, principally of the digestive system. There are several mechanisms by which these protective effects may be mediated, involving antioxidants and micronutrients, such as flavonoids, carotenoids, vitamin C and folic acid, as well as dietary fiber. These and other substances block or suppress the action of carcinogens and, as antioxidants, prevent oxidative DNA damage.

BENEFICIAL EFFECTS OF DIETARY FIBER

ORGAN	ACTION	HEALTH EFFECT
Teeth	Requires more chewing. Increases the secretion of saliva.	Protects against dental caries. Keeps gums healthy.
Stomach	Increases the secretion of saliva and gastric juice. Decreases the rate of evacuation of stomach contents into small intestine >> prolongs the feeling of satiety.	Enhances satiety prevents overeating and weight gain
Digestive tract	Shortens intestinal transit time. Dilutes harmful substances. Beneficial for the bacterial population in the large intestine. Interrupts the enterohepatic circulation of estrogens, reducing estrogen levels	Prevents constipation. Decreases risk of breast and colon cancer. Alters bile acid metabolism in the gut in a favorable way.
Cardiovascular system	Inhibits the absorption of dietary holesterol.Increases the release of bile acids into the intestine. Influences the plasma triglyceride levels and blood clotting properties	Lowers blood cholesterol levels. Decreases the risk of heart disease and gallstones.
Blood glucose	Slows down the absorption of carbohydrates.	Stabilizes blood glucose levels, especially in diabetic individuals.

Ecologic, observational and laboratory studies generally agree that eating a diet high in vegetables, fruits, and other plant-based foods; low in animal fats and low in salt content, along with maintaining a healthy weight, not using tobacco in any form, and being physically active can reduce the risk of cancer, CHD, and other chronic diseases[18].

In the gastrointestinal tract, they modify the absorption of nutrients (particularly carbohydrates and lipids) in the small bowel. They accelerate the gut transit time and determine stool composition and quantity. They are the main nutritional source for the colonic microflora. During the bacterial fermentation, short-chain fatty acids are formed which are essential for nutrition and integrity of the colonocytes and for colonic function. Moreover detoxicating enzymes, antioxidants and carcinogen-inactivating compounds are also formed as a result of bacterial fermantation of dietary fiber. The most important fibers are cellulose, hemicellulose, pectin, guar, psyllium, beta-glucan, lignin and digestion-resistant starch; they are present in varying amounts in plant foods and in fiber preparations.

Dietary fiber lowers the energy density of food, while increasing satiety and reducing hunger. In addition, fiber increases the viscosity of the gastrointestinal fluid, impeding mass flow and absorption of molecules in the small intestine, slowing the digestion of fat and starch and the transport of glucose into the blood stream[19]. The net result is that increased consumption of dietary fiber as part of a healthy diet, either in foods naturally high in fiber or from foods supplemented with fiber, can lead to the gradual loss of weight by reducing caloric intake[20].

Soluble fibers (those found primarily in fruit and vegetables) modestly reduce total and LDL cholesterol concentrations beyond those achieved by a diet low in saturated fat and fiber, by slowing gastric emptying, may reduce the appetite and help to control caloric intake. Diets low in dietary fiber may underlie or exacerbate constipation, appendicitis, hemorrhoids, deep vein thrombosis, varicose veins, diverticulitis, hiatal hernia, and gastroesophageal reflux[21]. As components in foods are digested and absorbed from the small intestine, fiber becomes a major component in the gut lumen, making the viscosity evident. This viscosity interferes with bile acid absorption from the ileum[22]. In response, LDL cholesterol is removed from the blood and converted into bile acids by the liver to replace the bile acids lost in the stool. Some evidence also indicates that changes in the composition of the bile acid pool accompanying ingestion of some viscous fibers dampen cholesterol synthesis. Because endogenous synthesis accounts for about three-quarters of total body cholesterol pool, slowing synthesis, as do the “statin” drugs, could have a favorable impact on blood cholesterol concentrations [23,24].

CLINICAL BENEFITS OF DIETARY FIBER SUPPLEMENTATION

- **GASTRO INTESTINAL DISORDERS**
- **OBESITY**
- **DIABETES MELLITUS**
- **CORONARY ARTERY DISEASE**
- **HYPERCHOLESTEROLEMIA**
- **CANCERS**

DIETARY FIBER SUPPLEMENTATION AND GASTRO INTESTINAL DISORDERS

Laxation is a very important physiological effect that results from increasing the dietary fiber component of one's diet in place of other food components. The discomfort of constipation and the potential for increasing the risk of other diseases resulting from constipation such as diverticular disease and hemorrhoids cannot be understated. Positive nutrition effects include improved body function and increased overall body comfort. Improved laxation fits in both categories. Increased dietary fiber in the diet results in an increase in fecal bulk, reduced transit time of fecal material through the large intestine, increased frequency of defecation, improved regularity of defecation, and reduced hardness of stools. Accompanying this there is typically a shift to a lower colonic pH, an increase in intestinal microflora populations and a change in intestinal microflora species distributions, all considered beneficial. Softer stools result in less discomfort to the colon and anus at time of elimination and less strain on the muscles involved in defecation.

Due to high intake of refined foods, the expenditure on laxatives had increased to $870 million in the USA in 1999. An Australian health survey (conducted via the postal service) completed by 14,761 young (18-23 years), 14,070 middle-aged (45- 50 years), and 12,893 older (70-75 years) women reported in 2000 showed constipation as a problem for 14.1% of the young, 26.6% of the middle-aged, and 27.7% of the older women. Hemorrhoids affected 3.2% of the young, 17.7% of the middle-aged and 18.3% of the older women. One third of the young women and half of the middle-aged and older women had sought help for their constipation[25]. An overall constipation rate of 14.7% was found with some 45% of the individuals with constipation reporting to be suffering from the complaints for 5 years or more in 1997 in a telephonic interview of 10,018 individuals in the United States[26].

Wheat bran is a very good source of dietary fiber as it is not soluble in water. This gives wheat bran its main beneficial action of being a good (and cheap) laxative - because the fiber increases the mass of the stools. Wheat bran has few other actions that are considered important. Oat bran contains some insoluble fiber plus a larger amount of soluble fiber, both of which can help laxation.

Burkitt et al[27], compared various population groups and found that those on high fiber diets produced stools of 150- 980 (average 275 for children, 470 for adults) grams/day, with transit times of 19-68 hours (average 33.5 for children, 35.7 for adults). Those on low fiber produced stools of 39-195 (average 173 for children on a high fruit diet, 110 for teenagers, and 104 for adults) grams/day with transit times of 28-144 hours (average 48 for children on a high fruit diet, 76.1 for teenagers, and 83.4 for adults). The group consuming a mixed diet in terms of dietary fiber content produced stools of 48-488 (average 165 for children, 185 for teenagers, 155 for nurses, 175 for hospital patients, and 225 for vegetarians) grams/day with transit times of 18-118 hours (average 45.2 for children, 47.0 for teenagers, 44.0 for nurses, 41.0 for hospital patients, and 42.4 for vegetarians). The consensus report of the European Non-Digestible Oligosaccharides concluded there is convincing evidence that consumption of non-digestible oligosaccharides stimulates bowel habit[28].

Partially-hydrolyzed guar gum given 3 times a day in 12 gram dosages as a beverage after every meal, increased fecal weight and output frequency in eight healthy men over the course of a four week study[29]. Fifteen women aged 18-48 years suffering from constipation consumed 8.2 grams/day of partially hydrolyzed guar gum in addition to an average daily intake of 9.2 grams/day of other dietary fibers. Defecation frequency increased by 37% and fecal moisture increased from 69.1 to 73.8%[30]. Again adding 20 grams of partially-hydrolyzed guar gum per liter of enteral feeding solution resulted in a significant decrease in cases of diarrhea in a study involving 100 patients on total or supplemental enteral nutrition[31].

Epidemiological evidence suggests that low dietary fiber intake may predispose to irritable bowel syndrome (IBS) [32]. Administration of dietary fiber supplements in these patients leads to an improvement in colonic motility. In a controlled trial, intake of extra fiber led to significant decrease in pain and improvement in bowel habits and colon motility[33]. In studies on rats the amount of butyric acid in the distal colon has been shown to be higher with dietary components containing high amounts of resistant starch. Further, the fermentability is lower and the butyric acid concentration higher with composite foods than with the corresponding purified fiber fractions. In human studies the faecal concentration of butyric acid has been shown to increase in patients with ulcerative colitis when [beta]-glucan-enriched oat bran (20 g fiber) is added to the diet for 12 weeks. Also, an improvement of symptoms was reported[34].

DIETARY FIBER SUPPLEMENTATION IN OBESITY

Globally, there are more than 1 billion overweight adults, at least 300 million of them obese. Obesity and overweight pose a major risk for chronic diseases, including type 2 diabetes, cardiovascular disease, hypertension and stroke, and certain forms of cancer. The key causes are increased consumption of energy-dense foods high in saturated fats and sugars, and reduced physical activity.

The obesity epidemic is not restricted to industrialized societies; and is often faster in developing countries than in the developed world. The health consequences range from increased risk of premature death, to serious chronic conditions that reduce the overall quality of life. Of especial concern is the increasing incidence of child obesity.

The rising epidemic reflects the profound changes in society and in behavioral patterns of communities over recent decades. While genes are important in determining a person's susceptibility to weight gain, energy balance is determined by calorie intake and physical activity. Thus societal changes and worldwide nutrition transition are driving the obesity epidemic. Economic growth, modernization, urbanization and globalization of food markets are just some of the forces thought to underlie the epidemic.

The distribution of BMI is shifting upwards in many populations. And recent studies have shown that people who were undernourished in early life and then become obese in adulthood, tend to develop conditions such as high blood pressure, heart disease and diabetes at an earlier age and in more severe form than those who were never undernourished[35]. The "Nurses Health Study" found that compared to lean women (BMI < 22 Kg per m2) those with BMI > 35 had a 93 fold increased risk of developing type 2 diabetes mellitus[36, 37]. For men with BMI >35, the risk was increased 40- fold[38].

The non-fatal, but debilitating health problems associated with obesity include respiratory difficulties, chronic musculoskeletal problems, skin problems and infertility. The more life-threatening problems fall into four main areas: CVD problems; conditions associated with insulin resistance such as type 2 diabetes; certain types of cancers, especially the hormonally related and large-bowel cancers; and gallbladder disease. The likelihood of developing Type 2 diabetes and hypertension rises steeply with increasing body fat. Confined to older adults for most of the 20th century, this disease now affects obese children even before puberty. Approximately 85% of people with diabetes are type 2, and of these, 90% are obese or overweight. And this is increasingly becoming a problem in developing world. Chronic overweight and obesity contribute significantly to osteoarthritis, a major cause of disability in adults.

Although obesity should be considered a disease in its own right, it is also one of the key risk factors for other chronic diseases together with smoking, high blood pressure and high blood cholesterol. In the analyses carried out for *World Health Report 2002*, approximately 58% of diabetes and 21% of ischemic heart disease and 8-42% of certain cancers globally were attributable to a BMI above 21 kg/m^2.

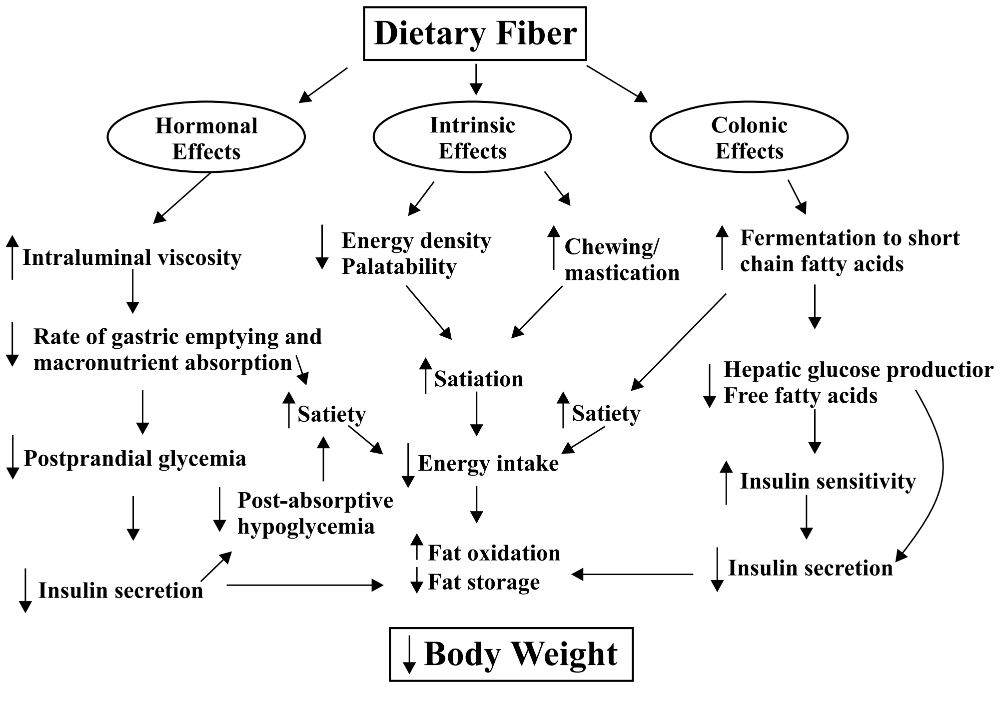
The prevalence of obesity among young adult males in urban areas was between 10.7% to 53.1% [39]. In a similar study in Delhi of 13,414 adults aged 25-64 years in urban areas, the prevalence of obesity was found to be 27.8%. The incidence of obesity was higher in females 33.4% as compared to 21.3% among males and obesity was found to be associated with hypercholesterolemia, hyperlipidemia and lower levels of physical activity[40]. The Nutrition Foundation of India reports that the prevalence of obesity in the upper strata of urban society is 32.2% among males and 50% among females and in the middle class the prevalence is 16.2% among males and 30.3% among females[41]. Obesity, both in children and adults, and its contribution to NCDs are likely to become a serious health problem in India[42].

Childhood obesity is already epidemic in some areas and on the rise in others. An estimated 17.6 million children under five are estimated to be overweight worldwide. The prevalence of obese children aged 6-to-11 years has more than doubled since the 1960s. Obesity prevalence in youths aged 12-17 has increased dramatically from 5% to 13% in boys and from 5% to 9% in girls between 1966-70 and 1988-91 in the USA. The problem is global and increasingly extends into the developing world. For example, in Thailand the prevalence of obesity in 5-to-12 year old children rose from 12.2% to 15.6% in just two years. The probability of continuing obesity or overweight in adulthood, when acquired during childhood, varies with the child's age and the presence of obesity or overweight in parents: it is the primary risk of childhood obesity. Thus, approximately 20% to 50% of children who were obese before puberty will remain so in adulthood, and 50% to 70% of obese adolescents will retain this obesity in adulthood. According to the WHO's global database, India has a preschool childhood obesity prevalence of 1% [43]. Joshi et al[44] from Asian Health Care have found obesity as an emerging problem in pre-school, school going and adolescent children.

Although the majority of obese or overweight children have no medical complications, specific disorders can occur in the case of severe obesity and some subclinical disorders (hypertension, hypercholesterolemia, and hypertriglyceridemia, etc.) are more common in obese children. Furthermore, childhood obesity has psychosocial consequences, relating to a loss of self-esteem, worsened by the stigmatization of obesity. The presence of atherosclerotic risk factors in 237 school children aged 13-17 years in western India was examined. Obesity was observed in 10% of the population, borderline hypertension in 27%, borderline hypercholesterolemia in 32.9% and definite hypercholesterolemia in 6.8%. Fats provided nearly 40% of the diet and fiber intake was less than 10 gm/day. The study showed a high prevalence of metabolic and dietetic coronary risk factors among adolescents of the middle- and upper-middle class in India[45].

Heaton[46] proposed that fiber acts as a physiologic obstacle to energy intake by at least three mechanisms: 1) fiber displaces available calories and nutrients from the diet; 2) fiber increases chewing, which limits intake by promoting the secretion of saliva and gastric juice, resulting in an expansion of the stomach and increased satiety; and 3) fiber decreases the absorption efficiency of the small intestine. Diets high in energy density increase food consumption when compared with diets lower in energy density. High-fiber foods have much less energy density compared with high-fat foods.

Rolls[47] suggested that as humans consume a constant weight of food and, hence, lower energy (i.e., high fiber) food intake per unit weight may promote a decrease in weight. Thus, high-fiber foods can displace energy or calories. Further, eating a low energy or high-fiber food increases satiety. The bulking and viscosity properties of dietary fiber are predominantly responsible for influencing satiation and satiety[48].



Physiological effects of dietary fiber[49]

Fiber rich foods usually are accompanied by increased efforts and/or time of mastication, which leads to increased satiety through a decreased rate of ingestion due to intrinsic, hormonal, and colonic effects of dietary fiber. Dietary fiber may also influence fat oxidation and fat storage.

A diet of different fiber-containing foods also is usually richer in micronutrients. Theoretically, if dietary fiber could block or limit the absorption of macronutrients, it could aid in weight control. Even a small change in absorption could have long-term significance in weight maintenance.

As per the World Health Organisation[50], some of the key elements for effective weight management apart from reduction of fat and calories include:

- Creating supportive population-based environments through public policies that promote the availability and accessibility of a variety of low-fat, high-fiber foods, and that provide opportunities for physical activity.
- Promoting healthy behaviors to encourage motivate and enable individuals to lose weight by eating more fruit and vegetables, as well as nuts and whole grains.

Populations that report higher fiber consumption also demonstrate lower obesity rates[51].A multidisciplinary study of more than 5000 individuals associated obesity with increased energy intakes and decreased consumption of fiber rich foods such as fruits and vegetables[52]. Again, in cross-sectional observational studies, fiber intake was reported to be inversely associated with body weight[53 – 55] and sub scapular skin fold thickness[56] as well as body mass index among young adults[57].

It is interesting to note that obese men and women have significantly lower dietary fiber intakes than do lean mean and women[58]. Again in the Nurses' Health Study, weight gain was inversely associated with intake of high fiber, whole grain foods and positively associated with intake of refined grain food[59]. Large intakes of dietary fiber at breakfast are associated with less food intake at lunch. Levine et al. found less food intake at lunch when a high-fiber breakfast cereal was consumed earlier in the day[60]. A higher-fiber breakfast with a low glycemic index was associated with less food intake at lunch for normal and overweight children[61].

When postmenopausal women consumed higher-fiber diets they showed a significant weight loss[62]. Birketvedt et al[63] found that the addition of dietary fiber to a low-calorie diet significantly improved weight loss, with the placebo group losing 5.8 kg and the fiber-supplemented group losing 8.0 kg. With ad libitum energy intake, the average effect of increasing dietary fiber across all these studies indicated that an additional 14 g/d of fiber resulted in a 10% decrease in energy intake and a weight loss greater than 1.9 kg through approximately 3.8 months of intervention. The effects of increasing dietary fiber were reported to be even more impressive in obese individuals[64]. The effect of one week of supplementation with a water-soluble fiber (guar gum) in obese women (40 g in group 1 and 20 g in group 2) and no supplementation in group 3 resulted in reduction in energy intake by soluble fiber under free living conditions and in hunger-reducing effect of fiber at the low energy intake level indicating the usefulness of fiber in the treatment of obesity[65]. Delargy et al[66] compared the effects on short-term (24 h) appetite of two equienergetic high (22 g) fiber breakfasts, an equienergetic low fiber breakfast and a low energy 'light' breakfast. Psyllium gum (the soluble fiber) and wheat bran (the insoluble fiber) were incorporated into breakfast cereals. Hunger ratings showed a trend towards the subjects being less hungry and they consumed significantly less energy at snack time after the high insoluble than after the high soluble fiber breakfast cereal. The results indicate that different types of fiber modulate the time course of appetite control and may produce alterations in the experience of motivation and patterns of eating without necessarily effecting total energy intake. According to Pereira and Ludwig[67] out of 27 experimental human studies published between 1984 and 2000, seventeen studies showed a beneficial effect of dietary fiber on energy intake and seven reported mixed effects. The results of the CARDIA study[57] also provide strong support for a significant role of dietary fiber in weight regulation.

According to Holt et al[68] the strongest predictor of the breads' satiety index scores was their portion size, total carbohydrate, fiber content and thus energy density. Warren et al[61] found that low glycemic breakfasts with higher dietary fiber decreased food intake in preadolescent children.

Increasing dietary fiber in the diet, as a part of a health promotion plan of action may be an important public health strategy in preventing obesity.

HYPOGLYCEMIC EFFECT OF DIETARY FIBER SUPPLEMENTATION

The WHO has projected an increase in the incidence of diabetes in the Indian population to 57 million by 2025 from the current level of about 26 – 27 million people. The Chennai Urban Rural Epidemiology Study (CURES) conducted by the Madras Diabetic Research Foundation with the objective of estimating the prevalence of diabetes, hypertension and related complications in the population covering a sample size of about 26,000 people observed that more than 16% of the metro population above the age of 20 was diabetic and about 22.3% were obese. There was also a three fold increase in the prevalence of diabetes in obese as compared to the lean population[69]. All India Institute of Medical Sciences[70] observed a high predisposition to diabetes among children in Delhi. It was observed that 24% of male and 36% of female children had insulin resistance, hypertension, high triglycerides, glucose intolerance (Syndrome X) and 13% had sub-clinical inflammation, a clear indicator for future diabetes and heart diseases. The children also had low levels of HDL cholesterol. The dietary habits of these children revealed a lower consumption of fiber and monounsaturated fat and higher consumption of saturated fat.

Although a direct and irrefutable linkage between insufficient dietary fiber intake and diabetes has not been established, significant research since that time has indicated decreased risk of the disease with increased dietary fiber consumption[71]. Shortly thereafter, several researchers designed and carried out a series of studies that showed beneficial effects of high fiber diets for individuals afflicted with the disease. Beneficial effects of increased dietary fiber consumption were shown for both Type 1 and Type 2 diabetics and included improved glucose tolerance, reduced insulin requirements, increased peripheral tissue insulin sensitivity, decreased serum cholesterol, decreased serum triglycerides, better weight control, and potentially consistently lower blood pressure[72].

Of the 19 studies reviewed, 17 studies reported decreased fasting blood glucose levels ranging from 6-39%, 13 of which were statistically significant[73]. According to Jenkins et al[74] soluble dietary fibers, either as part of a food or as a supplement well mixed with food appear to exhibit the greatest therapeutic effect. Jenkins et al[75] also showed that of guar, pectin, gum tragacanth, methylcellulose and wheat bran each flattened the glucose response, with the reduction in mean peak rise in blood glucose concentration being positively correlated to viscosity. The reduction in the postprandial blood glucose was 44% with guar gum, 29% with pectin, 29% with psyllium, 23% with other gelling fibers (gum tragacanth, methyl cellulose, locust bean gum, agar, Konjac mannan), and 27% wheat bran. A nonlinear inverse correlation between the glycemic index and the dietary fiber content of a food was shown[76,77].

Reduction in postprandial glucose with dietary fiber supplement[75]

Type of fiber	Percentage reduction in postprandial glucose levels
Guar gum	**44%**
Pectin	**29%**
Psyllium	**29%**
Wheat bran	**27%**

Further similar correlations between the glycemic index and the insoluble dietary fiber component and the soluble dietary fiber component with a stronger dependency on the soluble dietary fiber component were demonstrated[75]. The high fiber diet lowers significantly the fasting as well as the postprandial glucose[78.] In a randomized crossover study[79]of 13 patients a high intake of dietary fiber (50g/day), particularly of the soluble type was found to **improve glycemic control** – mean plasma glucose concentration was lower by 8.9%, **decrease hyperinsulinemia** – plasma insulin concentrations were 12% lower and **decrease plasma lipid concentrations** – fasting plasma total cholesterol was 6.7% lower, plasma triglyceride was 10.2% lower, plasma VLDL concentration was 12.5% lower and plasma LDL was 6.3% lower, in patients with Type 2 diabetes. The high fiber diet also decreased gastrointestinal absorption of cholesterol by 10% and increased fecal acidic sterol excretion by 41%. Epidemiological studies[80] have shown the healthful effects of diets in Mediterranean countries that are high in fruits, vegetables and grains.

Total fiber intake (g/day) was found to be inversely related to HbA1c ($p = 0.02$), independently of carbohydrate intake, total energy intake and other factors regarding lifestyle and diabetes management. Severe ketoacidosis risk fell significantly with higher fiber intake ($p = 0.002$). Furthermore, increased fiber intake may reduce the risk of severe ketoacidosis[81]. Similarly there is a reduction of 14.4% in total cholesterol, 14% in triglycerides and 28.7% in LDL and an increase in HDL cholesterol by 12 mg/dl in 90 days with supplements containing guar gum, pectin and oat fiber (10-15g)[82]. Passi et al[83] also found similar results of 13% reduction in total cholesterol, 16% reduction in triglycerides and 15% reduction in LDL cholesterol. Guar gum supplementation was also found to increase HDL cholesterol by 6 mg/dl.

It is hypothesized that fiber-depleted foods will lead to higher glucose levels and stimulate excessive insulin secretion. Hyperinsulinaemia, in turn, may result in down regulation of the insulin receptors, lower levels of relevant signaling molecules and thus an elevation of the fasting blood glucose. Once insulin resistance is established there may be an even greater sensitivity to the deleterious effect of low-fiber, high Glycemic-Index foods, thus maintaining a vicious cycle. Viscous fiber may act to break this cycle by reducing the rate of absorption and lowering the postprandial glycaemic and insulinaemic responses. To achieve these effects, viscous fibers convert the small intestine into a storage organ for the slow release of glucose to the portal circulation. Viscous fiber may impede bulk diffusion of the products of luminal digestion to the mucosal surface.

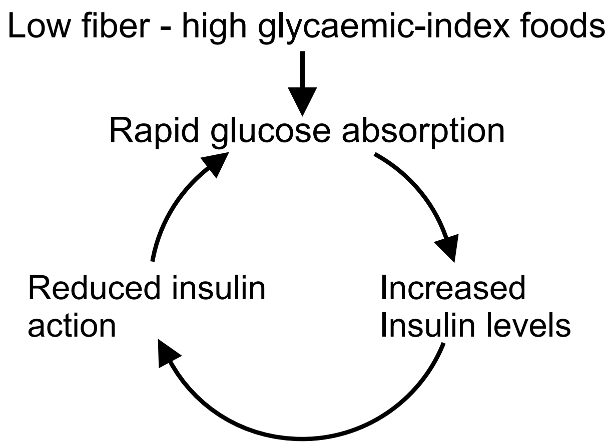


Fig. 1. Potential mechanisms for the relationship between dietary fiber and insulin resistance.

Effect on Gut hormones

Beyond the effects of dietary fiber on postprandial glucose, insulin, and satiety discussed above, dietary fibers alter responses and actions of the gut hormones gastric inhibitory peptide, glucagon-like peptide-1[84,85] and cholecystokinin (CCK). CCK is a peptide hormone and a neurotransmitter secreted by cells in the upper part of the small intestine after ingestion of food. This regulates gut motility, gallbladder contraction, and pancreatic enzyme secretion. CCK may mediate postprandial glycemic and insulinemic responses to viscous fibers[85]. A direct correlation between postprandial CCK and subjective satiety scores after ingestion of foods that differed in the amount of fiber was reported by Holt et al[87]. Plasma CCK was associated with subjective measurements of satiety in women[88]. Bean intake, as a source of dietary fiber, also increased CCK in male subjects[89].

Effect on Gastric Emptying

Dietary fiber affects gastrointestinal physiology and functions including delaying gastric emptying[90]. Consumption of viscous fibers delays gastric emptying, which may cause an extended feeling of fullness and may delay absorption of glucose and other nutrients[91]. Yao and Roberts[92] summarized the effect of an increase in energy density on the rate of gastric emptying in humans and consistently found that high-fiber diets slowed gastric emptying. They also speculated that there is a link between palatability, gastric emptying, and glycemic index. Dietary fiber also by delaying gastric emptying and/or slowing down the energy and nutrient absorption, leads to lower postprandial glucose and lipid levels.

DIETARY FIBER AND CORONARY HEART DISEASE (CHD)

Recently the American Heart Association (AHA) and the Food and Drug Administration (FDA) confirmed that coronary heart disease is the leading cause of death in the United States, killing more people than any other disease. It causes heart attack and angina (chest pain). The AHA ranks stroke as the third most fatal disease in America, causing paralysis and brain damage. Reduced risk of developing and consequently dying from CHD was amongst the earliest observations of workers in dietary fiber research, with subsequent inclusion in the "dietary fiber hypothesis". Scientific consensus on the evidence for the role of dietary fiber in reducing the risk of CHD has thus been acknowledged. In 1993 the regulations for the Nutrition Labeling and Education Act allowed claims that high fiber foods, i.e. whole grains, fruits and vegetables, may be effective in reducing CHD risk. Subsequently, specific claims for oat and oat bran products and psyllium products have also been allowed, based in part on the results of extensive *meta*-analyses of the research data accumulated on these two foods in recent decades. Epidemiologic data suggests that the intake of complex carbohydrate and dietary fiber is associated in an inverse manner to risk for coronary artery disease (CAD).

Since 1979 studies have repeatedly shown that fiber, and cereal fiber in particular, results in a reduced risk of CHD. It has been estimated that an additional 5 g of cereal fiber per day will reduce the risk of CHD by 37%[93]. In a Harvard study of over 40,000 male health professionals, researchers found that a high total dietary fiber intake was linked to a 40 percent lower risk of coronary heart disease, compared to a low fiber intake[94]. Raised plasma cholesterol is a major risk factor for early coronary artery disease. Studies in America have shown that a 1% reduction in cholesterol produces a 2% reduction in the risk.

It has been known for many years that oats and oat products can help to lower blood cholesterol levels in the body. The soluble fiber component of oat bran helps in lowering the cholesterol. This probably occurs through the increased conversion of cholesterol to bile acids in the liver. Because oat bran binds bile acids in the intestine, more bile acids are lost when oat bran is eaten; therefore more cholesterol is used to replace the bile acids.

Short term, controlled clinical trials indicate that oat bran or beans, in a metabolic ward setting, decreased serum cholesterol concentrations of hypercholesterolemic individuals by 10 to 12%. Studies of free-living hypercholesterolemic individuals and animal studies have documented that incorporation of oat products, psyllium or guar gum and pectin into the diet decreases serum cholesterol by 6 to 8% due to the viscous, soluble fibers in these foods which have substantial hypocholesterolemic effects.

Feeding experiments in humans have shown that about 60 to 90 grams of oat bran per day (depending on the type of bran) is required in order to lower blood cholesterol levels appreciably. This amount may provide up to 10 grams of the total fiber intake required per day. The quantity of rolled oats needed to produce the same effect is larger because oat bran is a more concentrated product. However, one food product alone in the diet cannot be expected to lower cholesterol levels by itself and should be coupled with a reduction in the intake of total and saturated fats.

According to the American Heart Association, for every gram increase in soluble fiber from sources such as oat, LDL cholesterol may decrease by an average of 2.2 mg/dl[95]. Long term clinical trials have indicated that increasing soluble fiber intake as part of a low fat, low cholesterol diet reduces serum cholesterol concentration from 3 to 5% below that for the low fat, low cholesterol diet.

Other studies suggest that increased fiber intake may decrease blood pressure slightly, assist in weight management, alter blood clotting factors, and increase insulin sensitivity. Intake of dietary fiber and complex carbohydrate appear to have a protective role for coronary artery disease (CAD). A 12-year study of 859 southern California men and women showed that a 6-gram increment in daily fiber intake was associated with a 25% reduction in ischemic heart disease mortality[96].

A twelve-week study utilizing 208 healthy men and women demonstrated that consuming oat products along with the AHA fat-modified eating style resulted in approximately 3% lower serum cholesterol than the reduced-fat diet alone[97]. A diet of various levels of beta-glucan (in the form of oat meal and oat bran) an acceptable form of water-soluble fiber, is effective in lowering serum cholesterol levels in conjunction with a low-fat diet in a dose dependent manner[98]. Beta glucan in oats and psyllium husk, have been sufficiently studied for the FDA to authorize a health claim that foods meeting specific compositional requirements and containing 0.75g or 1.7g of soluble fiber per serving, respectively, can reduce the risk of heart disease[99]. Consequently, these two dietary fibers are specifically included in the most recent National Cholesterol Education Program American Heart Association guidelines[100]. Glucomannan, the fiber from the Konjac root, and a staple of the traditional Japanese diet is also valuable for reducing cholesterol.

The characteristics such as solubility in water, viscosity, fermentability, and the kinds and amounts of protein and tocotrienols have been explored as possible bases for this physiological effect and the mechanism by which these fiber sources lower blood cholesterol levels has been the focus of many investigations[101]. Viscosity has been one characteristic common to all cholesterol-lowering fibers[101]. Indeed, when a soluble fiber that is not viscous is evaluated or the fiber is treated to reduce viscosity sufficiently, the cholesterol-lowering ability is lost[102-104]. As components in foods are digested and absorbed from the small intestine, fiber becomes a major component in the gut lumen, making the viscosity evident. This viscosity interferes with bile acid absorption from the ileum[105-106]. In response, LDL cholesterol is removed from the blood and converted into bile acids by the liver to replace the bile acids lost in the stool. Some evidence also indicates that changes in the composition of the bile acid pool accompanying ingestion of some viscous fibers dampen cholesterol synthesis [106-107]. Because endogenous synthesis accounts for about three-quarters of total body cholesterol pool, slowing synthesis, as do the "statin" drugs, could have a favorable impact on blood cholesterol concentrations. Increasing soluble fiber intake by consuming a wide variety of foods may[108] or may not[109] have a hypocholesterolemic effect; this variable effectiveness may depend on the composition of the rest of the diet.

A *meta*-analysis of 67 controlled studies focusing on soluble dietary fibers through oat products, psyllium, pectin and guar gum showed a significant reduction in serum cholesterol with increased dietary fiber intake[110]. Significant reductions in serum cholesterol with 50 g/day of oat bran and 42.5 g/day of processed oat bran were demonstrated in free-living men and women with hypercholesterolemia[111-113]. Oda et al[114] showed that rats on diets supplemented with soluble fiber fractions from oat, barley or wheat had lower liver cholesterol even though their plasma cholesterol was not significantly lower. Insoluble dietary fiber fractions also lowered liver cholesterol, but not significantly.

Evidence is also accumulating that a diet rich in fiber may protect against diseases associated with raised clotting factors. Pectin supplementation caused significant decreases in total cholesterol, low-density lipoprotein cholesterol, apolipoprotein A & B and lipoprotein-a. Significant changes in the characteristics of fibrin networks developed in the plasma of the pectin supplemented group indicated that networks were more permeable and had lower tensile strength. These network structures are believed to be less atherogenic. It is suspected that pectin modified network characteristics by a combination of its effects on metabolism and altered fibrin conversion[115]. This confirms the therapeutic possibilities of dietary intervention.

A 19-year follow-up study reported in the November 2001 issue of Archives of Internal Medicine indicated that increasing bean and legume intakes may be an important part of a dietary approach to preventing coronary heart disease[116]. Soybeans and legumes are high in protein and soluble fiber. Liu et al[117] reported that increasing our consumption of fiber-rich foods like whole grains, fruits and vegetables, can significantly lower the risk of heart disease. Thus, it is proved that eating a high-fiber diet can significantly lower our risk of heart attack, stroke and colon cancer.

ANTICARCINOGENIC EFFECT

Case-control studies that compared non-vegetarian and vegetarian diets and alcohol and tobacco use in India have reported that vegetarians have a reduced risk of oral, oesophageal, and breast cancers[118]. In India, the incidence of breast cancer is increasing, with an estimated 80,000 new cases diagnosed annually. The incidence of breast cancer increased by approximately 50% between 1965 and 1985[119].

A recent comprehensive analysis by World Cancer Research Fund & American Institute for Cancer Research[120], showed that vegetables exert a convincing preventive effect for five cancers, a probable preventive effect for four others, and a possible preventive effect for another seven. Fruits revealed four convincing, four probable and four possible preventive relationships. In short, the best available evidence suggests that vegetables and fruits have far more cancer-preventing potential than do grains. This probably reflects the phytochemical content of fruits and vegetables, phytochemicals to which current human biology became adapted through many million years of evolutionary interrelationships.

Obesity and lack of physical activity are associated with increased risk at various cancer sites, including breast and endometrial cancer. In India, increases in the rates of obesity, central adiposity, and waist-hip ratio associated with urbanization are seen in every region and are highest among those with the highest levels of education and income[121]. Large epidemiological studies have identified a possible association between increased dietary fiber and a decreased risk for cancers of the colon and breast. The nutrition guidelines follow the World Cancer Research Fund recommendations that advocate having a diet containing vegetables and fruits in large amounts, reducing the intake of saturated fats, and increasing physical activity. Additionally, results from recent studies at the American Institute of Cancer Research indicate high-fiber protein-rich soy-based products, such as textured soy protein and tempeh, help in preventing and treating colon cancer.

Extensive epidemiologic evidence supports the theory that dietary fiber may protect against large bowel cancer. Correlation studies that compare colorectal cancer incidence or mortality rates among countries with estimates of national dietary fiber consumption suggest that fiber in the diet may protect against colon cancer. Data collected from 20 populations in 12 countries showed that average stool weight varied from 72 to 470 g/day and was inversely related to colon cancer risk[122].

When results of 13 case-control studies of colorectal cancer rates and dietary practices were pooled, the authors concluded that the results provided substantive evidence that consumption of fiber-rich foods is inversely related to risks of both colon and rectal cancers[123].

Despite the inconsistency in the results of fiber and colon cancer studies, the scientific consensus is that there is enough evidence that dietary fiber protects against colon cancer and that health professionals should be promoting increased consumption of dietary fiber[124].

Ingestion of prebiotics (non-digestible food ingredient that selectively stimulates bacteria in the colon) results in a different spectrum of fermentation products, including the production of high concentrations of short chain fatty acids, leading to a decrease in pH. A low pH in faeces was associated with a reduced incidence of colon cancer in various populations[125,126].

Butyrate is associated with many biological properties in the colon[127]. One of the first observed effects of butyrate on the degree of DNA methylation is probably associated with modified gene expression, the consequences of which are yet unknown, particularly in relation to colon cancer. However, butyrate may also directly enhance cell proliferation in normal cells and suppress proliferation in transformed cells by improving cell differentiation. This is an important step in suppressing cancer cells. In addition, apoptoses may be increased in transformed cells but inhibited in normal cells when butyrate is present [128-130].

Colon cancer, which in a high proportion of the population is due to somatic mutations occurring during the lifetime of an individual, could be retarded by preventing these mutations. Prebiotics have been shown to deactivate genotoxic carcinogens. DNA damage had been prevented and chemopreventive systems may be stimulated in vivo in colon tissues.

International comparisons show an inverse correlation between breast cancer death rates and the consumption of fiber-rich foods[131]. An interesting exception to the high-fat diet hypothesis in breast cancer was observed in Finland, where intake of both fat and fiber is high, and the breast cancer mortality rate is considerably lower than in the United States and other Western countries where the typical diet is high in fat[132]. The large amount of fiber in the rural Finnish diet may modify the breast cancer risk associated with a high-fat diet.

A pooled analysis of 12 case-control studies revealed that high dietary fiber intake is associated with reduced risk of breast cancer[133]. Dietary fiber intake has also been linked to lower risk of benign proliferative epithelial disorders of the breast[134]. Excess estrogen levels can occur as one gets older and the estrogen ratios get out of balance. Eating a diet high in fiber can bind or soak up the excess estrogen, increasing the rate at which it is excreted from the body[135,136]. Eating lots of fiber also speeds up digestion through the stomach, small intestine, and large intestine (or colon), limiting the amount of time cancer causing chemicals can get into the bloodstream. Some fibers have been shown to actually absorb cancer-causing substances that then leave the body in the stool[137].

MECHANISM OF ACTIONS OF DIETARY FIBER

In Obesity

It causes satiety by forming a bulk in stomach & sense of fullness.
The person eats less & looses weight.
Also help in lowering cholesterol

In Hypercholesterolemia & Coronary Heart Disease

Lowers cholesterol levels by interfering with reabsorption of bile acids.
It prevents atheromatous plaque development & also helps in diet control

In Diabetes

By forming a bulk in stomach it prolongs the gastric emptying time and therefore postprandial rise of sugar

In Constipation

Forms a bulk in the intestine and draws water into lumen.
Also it stimulates motility of intestines
Both these actions help in smooth evacuation of stools

INDICATIONS FOR DIETARY FIBER SUPPLEMENTATION

- Obesity
- Diabetes
- Hyperlipidemia
- Constipation
- Irritable Bowel Syndrome

POTENTIAL ADVERSE EFFECTS OF DIETARY FIBER

- **Feeling of Fullness**
- **Bloating**
- **Gastritis**
- **Flatulence**

Precautions with dietary fiber intake

It should always be taken along with **adequate amount of water** to prevent impaction & intestinal obstruction.

It should be used with caution in people with gastrointestinal diseases

CONCLUSION

It is a challenge for the clinicians and dietetic professionals to look into the changing pattern in the dietary intake and the nutrition transition in relation to its contribution to the emerging epidemics of non communicable diseases and to act with an insight into the diagnosis, pathogenesis, prevention and management of the same.

Dietary fiber as a preventive as well as curative agent in the daily diet has been well established. Different types of fiber have different functions with different mechanisms of action. The results of increased dietary fiber from high-fiber foods or from fiber supplements were similar. In addition, the beneficial effect of dietary fiber was seen for soluble and insoluble dietary fibers

Food technologists should find ways and means of reducing refined flour in the food products and bring out fiber rich food products in the market.

REFERENCES

1. Loren Cordain, S Boyd Eaton, Anthony Sebastian, Neil Mann, Staffan Lindeberg, Bruce A Watkins, James H O'Keefe, and Janette Brand-Miller, Origins and evolution of the Western diet: health implications for the21st Century; Am J Clin Nutr 2005;81:341–54.
2. Nutrition Now, Brown JE, 2nd Edition, 1999, Wadsworth Publishing Company).
3. Murray CJL, Lopez AD. Global Comparative Assessments in the Health Sector. Geneva: World Health Organization, 1994.
4. National Nutrition Monitoring Bureau, 1987. cf. Shetty PS 2002 Nutrition transition in India, Public Health Nutrition: 5(1A), 175–182.
5. AACC Report - The Definition of Dietary Fiber**;** Cereal Foods World March 2001, 46(3):112 – 126.
6. Williams CL Importance of dietary fiber in childhood J Am Diet Assoc. 1995 Oct; 95(10):1140-6, 1149; quiz 1147-8, Williams CL, Bollella M, Wynder EL: A new recommendation for dietary fiber intake in childhood. Pediatrics. 1995;96(5 Suppl S):985-988.

7. Marlett JA. Cheung T-F. Database and quick methods of assessing typical dietary fiber intakes using data for 228 commonly consumed foods. J Am Diet Assoc. 1997; 97:1139-1148,1151.

8. Position of the American Dietetic Association: Health implications of dietary fiber, Journal of The American Dietetic Association 2002, 102(7):993-1000.

9. Trepel F,Dietary Fiber: more than a matter of dietetics. I. Compounds, properties, physiological effects; Wien Klin Wochenschr, 2004 Jul 31; 116(14):465-76.

10. Ang JF and Crosby GA, Formulating reduced-calorie foods with powdered cellulose, Food Technology, 2005; 59(3):35-38.

11. Decker Weiss,Fiber and Health, Ask the Doctor publications; www.ATDonline.org. Accessed on 8.6.2005.

12. Eaton SB. Fiber intake in prehistoric times. In: Leeds AR, ed. Dietary Fiber perspectives 2 reviews and bibliography. London: John Libbey. 1990: pp. 27-40.

13. Asp N-G. Nutritional classification and analysis of food carbohydrates. Am J Clin Nutr 1994; 59:679S-681S.

14. Marlett JA, Longacre MJ. Comparisons of in vitro and in vivo measures of resistant starch in selected grain products. Cereal Chem 1996;73:63-68.

15. Freiburger LM, Gallaher DD. Association between intestinal contents viscosity and cholesterol lowering in rats fed fermentable and nonfermentable dietary Fibers. FASEB J 2000; 14:A291.

16. Gregory Stephens Optimising cardio health with oat beta-glucans April 2005, Functional Foods and Nutraceuticals.

17. Denis Lairon. INSERM, 18 avenue Mozart, Unite 476, 13009 Marseille, France Soluble dietary fibers and lipid metabolism: mechanisms of action. Abstract presentation, 2001 AACC Annual Meeting Charlotte, North Carolina, October 14-18, 2001; Charlotte Convention Center.

18. Schaefer EJ. Lipoproteins, nutrition, and heart disease. Am J Clin Nutr 2002;75:191-212

19. Ang J.F and Crosby G.A,Formulating reduced-calorie foods with powdered cellulose, Food Technology, 59(3):35-38,2005.

20. Bucholz A.C and Schoeller, D.A 2004 Is a Calorie a Calorie? Am J Clin Nutr. 79:899S – 906S.

21. Anderson JW, Smith BM, Gustafson NJ. Health benefits and practical aspects of high-fiber diets. Am J Clin Nutr 1994;59(suppl):1242S–7S.

22. Everson GT, Daggy BP, McKinley C, Story JA. Effects of psyllium hydrophilic mucilloid on LDL-cholesterol and bile acid synthesis in hypercholesterolemic men. J Lipid Res. 1992; 33:1183-1192.

23. Position of the American Dietetic Association: Health implications of dietary fiber Journal of The American Dietetic Association 2002. 102 (7) 993 – 1000.

24. Hillman LC, Peters SG, Fisher CA, Pomare EW. Effects of Fiber components pectin, cellulose, and lignin on bile salt metabolism and biliary lipid composition in man. Gut. 1986; 27:29-36.

25. Chiarelli, P., Brown, W., and McElduff, P. 2000. Constipation in Australian women: prevalence and associated factors. Int. Urogynecol. J.11(2):71-78.

26. Stewart, W.F., Liberman, J.N., Sandler, R.S., Woods, M.S., Stemhagen,A., Chee, E., Lipton, R.B., and Farup, C.E. 1999. Epidemiology of constipation (EPOC) study in the United States: relation of clinical subtypes to sociodemographic features. Am J Gastroenterol 94(12):3530- 3540.

27. Burkitt, D.P., Walker, A.R.P., and Painter, N.S. 1972. Effect of dietary Fiber on stools and transit-times, and its role in the causation of disease. Lancet ii:1408-1412.

28. Van Loo, J., Cummings, J., Delzenne, N., Englyst, H., Franck, A., Hopkins,M., Kok, N., Macfarlane, G., Newton, D., Quigley, M., Roberfroid,M., van Vliet, T., and van den Heuvel, E. 1999. Br. J. Nutr.81:121-132.

29. Takahashi, H., Yang, S.I., Hayashi, C., Kim, M., Yamanaka, J., and Yamamoto, T. 1993. Effect of partially hydrolyzed guar gum on fecal output in human volunteers. Nutr. Res. 13:649-657.

30. Takahashi, H., Wako, N., Okubo, T., Ishihara, N., Yamanaka, J., and Yamamoto, T. 1994. Influence of partially hydrolyzed guar gum on constipation of women. J. Nutr. Sci. Vitaminol. 40:251-259.

31. Homann, H.H., Kemen, M., Fuessenich, C., Senkal, M., and Zumtobel,V. 1994. Reduction in diarrhea incidence by soluble fiber in patients receiving total or supplemental enteral nutrition. J. Parenteral Enteral Nutr. 18(6):486-490.

32. Burkitt DP, Walker ARP, Painter NS. Effects of dietary Fiber on stools and transit times and its role in the causation of diseases. Lancet 1972;ii:1408-11.

33. Hahden JM, Blacksham E, Amate M, Parkains AC, Spiller RC. Abnormalities of GI transit in bloated irritable bowel syndrome: effect of bran on transit and symptoms. Am J Gastroenterol 2002;79:2315-20.

34. Margareta, E.; Nyman, G.-L 2003.. Importance of processing for physico-chemical and physiological properties of dietary Fiber**.** Proceedings of the Nutrition Society. Royal College of Physicians, Edinburgh, UK, 27-30 MAY 2002. 62(1):187-192, February 2003.

35. World Health Organization (WHO). Obesity: Preventing and Managing the Global Epidemic. Report of a WHO consultation. WHO Technical Report Series No. 894. Geneva: WHO, 2000.

36. Pinkney J. Treating diabeties and CHD:What should we do about obesity? Cordio-diabetes. Cardiovascular Disease and Diabetes Management 2002; 2: 9-14.

37. Manson JE, Colditz GA, Stampfer MJ *et al*. A prospective study of obesity and risk of coronary heart disease in women. *N Eng J Med* 1990; 322: 882-9.

38. Chan JM, Rimm EB, Colditz GA *et al*. Obesity, fat distribution and weight gain, risk factors for clinical diabetes in men. *Diabetes Care* 1994; 17: 961-9.

39. Dhurandhar NV, Kulkarni PR. Prevalence of obesity in Bomaby. Int. J. Obes. Relat. Metab. Disord. 1992; 16: 367–75.

40. Gopinath N, Chadha SL, Jain P, Shekawat S, Tandon R. An epidemiological study of obesity in adults in the urban population of Delhi. J. Assoc. Physicians India 1994; 42: 212–5.

41. Gopalan C. Obesity in the urban middle class. NFI Bull. 1998;19: 1–4.

42. Shetty PS. 2002 Nutrition transition in India, Public Health Nutrition: 5(1A), 175–182.

43. World Health Organization (WHO). Obesity: Preventing and Managing the Global Epidemic. Report of a WHO consultation. WHO Technical Report Series No. 894. Geneva: WHO, 2000.

44. Joshi SR, Joshi SS. Obesity therapeutics – the Indian Consensus. Gupta SB, (ed). API Medicine Update 2002, Association of Physicians of India, 2002; 12: 492-504.

45. Gupta R, Goyle A, Kashyap S, Agarwal M, Consul R, Jain BK Prevalence of atherosclerosis risk factors in adolescent school children, Indian Heart J. 1998 Sep-Oct;50(5):511-5.

46. Heaton KW. Food Fiber as an obstacle to energy intake. Lancet 1973; 2:1418 –21.

47. Rolls BJ. The role of energy density in the over consumption of fat. J Nutr 2000; 130 suppl):268S–71.

48. Burton-Freeman, B. Dietary fiber and energy regulation. J Nutr 2000; 130(suppl):272S–5.

49. Slavin JL,Dietary Fiber and Body Weight, Nutrition, 2005;21:411-418.

50. World health organization - global strategy on diet, physical activity and health, 2003.

51. Kimm S. The role of dietary fiber in the development and treatment of childhood obesity. Pediatrics 1995;96:1010–4.

52. Lissner L, Lindroos AK, Sjostrom L. Swedish obese subjects (SOS): an obesity intervention study with a nutritional perspective. Eur J Clin Nutr 1998;52:316 –22.

53. Alfieri M, Pomerleau J, Grace DM, Anderson L. Fiber intake of normal weight, moderately obese and severely obese subjects. Obesity Res 1995;3:541–7.

54. Appleby PM, Thorogood M, Mann JI, Key TJ. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary Fiber and alcohol. Int J Obesity Relat Metab Disord 1998;22:454–60.

55. Nelson LH, Tucker LA. Diet composition related to body fat in a multivariate study of 293 men. J Am Diet Assoc 1996; 96:771–7. 2004; 23:5–17.

56. Kromhout D, Bloemberg B, Seidell JC, Nissinen A, Menotti A. Physical activity and dietary fiber determine population body fat levels: the Seven Countries Study. Int J Obesity 2001; 25:301– 6.

57. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, Jacobs DR. Dietary fiber, weight gain, and cardiovascular risk factors in young adults. JAMA 1999; 282:1539–46.

58. Miller WC, Niederpruem MG, Wallace JP, Lindeman AK. Dietary fat, sugar and fiber predict body fat content. J Am Diet Assoc 1994;94:612–5.

59. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz G. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. Am J Clin Nutr 2003;87: 920 –27.

60. Levine AS, Tallman JR, Grace MK, Parker SA, Billington CJ, Levitt MD. Effect of breakfast cereals on short-term food intake. Am J Clin Nutr 1989;50:303–7.

61. Warren JM, Henry CJK, Simonite V. Low glycemic index breakfasts and reduced food intake in preadolescent children. Pediatrics 2003;112:414.

62. Mueller-Cunningham WM, Quintana R, Kasim-Karakas SE. An ad libitum, very low-fat diet results in weight loss and changes in nutrient intakes in postmenopausal women. J Am Diet Assoc 2003;103:1600–6.

63. Birketvedt GS, Aaseth J, Florholmen JR, Ryttig K. Long-term effect of Fiber supplement and reduced energy intake on body weight and blood lipids in overweight subjects. Acta Med 2000;43:129 –32.

64. Howarth NC, Saltzman E, Roberts SB. Dietary fiber and weight regulation. Nutr Rev 2001;59:129 –39.

65. Pasman WJ, Saris WH, Wauters MA, Westerterp-Plantenga MS Effect of one week of Fiber supplementation on hunger and satiety ratings and energy intake Appetite. 1997 Aug;29(1):77-87.

66. Delargy HJ, O'Sullivan KR, Fletcher RJ, Blundell JE Effects of amount and type of dietary Fiber (soluble and insoluble) on short-term control of appetite. Int J Food Sci Nutr.1997 Jan; 48(1):67-77.

67. Pereira MA, Ludwig DS. Dietary fiber and bodyweight regulation. Observations and mechanisms. Pediatr Clin North Am 2001;48:969– 80.

68. Holt S, Brand J, Soveny C, Hansky J. Relationship of satiety to postprandial glycaemic, insulin and cholecystokinin responses. Appetite 1992; 18:129–41.

69. The Hindu – Business Line, Internet Edition, Feb 24, 2003.

70. AIIMS study on diabetes warns kids over diet, The Indian Express, Mumbai ed., April 20, 2005.

71. Trowell, H.C. 1974. Diabetes Mellitus death rates in England and Wales, 1920-1970 and food supplies. Lancet 2:998-1001.

72. Anderson JW, Dietary Fiber, complex carbohydrate and coronary artery disease Can J Cardiol. 1995 Oct;11 Suppl G:55G-62G

73. Anderson, J.W., Gustafson, N.J., Bryant, C.A., and Tietyn-Clark, J. 1987. Dietary fiber and diabetes: a comprehensive review and practical application. J Am Diet Assoc 87(9):1189-1197.

74. Jenkins, D.J.A., Leeds, A.R., Gassull, M.A., Cochet, B., and Alberti, K.G.M.M. 1977. Decrease in Postprandial Insulin and Glucose Concentrations by Guar and Pectin. Ann. Int. Med. 86:20-23.

75. Jenkins, D.J.A., Wolever, T.M.S., Leeds, A.R., Gassull, M.A., Haisman,P., Dilawari, J., Goff, D.V., Metz, G.L., and Alberti, K.G.M.M. 1978. Dietary Fibers, Fiber analogues, and glucose tolerance: importance of viscosity. British Medical Journal 1:1392-1394.

76. Nishimune, T., Yakushiji, T., Sumimoto, T., Taguchi, S., Konishi, Y., Nakahara, S., Ichikawa, T., and Kunita, N. 1991. Am. J. Clin. Nutr. 54:414-419.

77. Trout, D.L., Behall, K.M., and Osilesi, O. 1993. Prediction of glycemic index for starchy foods. Am J Clin Nutr 58:873-878.

78. Rivellese, A., Riccardi, G., Giacco, A., Pacioni, D., Genovese, S., Mattioli, P.L., and Mancini, M. 1980. Effect of dietary Fiber on glucose control and serum lipoproteins in diabetic patients. Lancet 2:447-450.

79. Chandalia M et al, Beneficial effects of high dietary fiber intake in patients with Type 2 diabetes mellitus 2000, N Engl J Med;342:1392-8.

80. Nestle M 1995, Mediterranean diets: Historical and research overview, Am J Clin Nutr; 619 Suppl:1313S- 1320S.

81. Buyken AE, Toeller M, Heitkamp G, Vitelli F, Stehle P, Scherbaum WA, Fuller JH Relation of Fiber intake to HbA1c and the prevalence of severe ketoacidosis and severe hypoglycaemia. EURODIAB IDDM Complications Study Group. Diabetologia. 1998 Aug;41(8):882-90.

82. Peter J Verdegem, Unicity International, Orem, UT; Steven H Freed, David J Joffe; DiabetesInControl.com, Deerfield, IL, Abstracts of the Sixth Annual Conference on Arteriosclerosis,

Thrombosis and Vascular Biology; The Clinical Impact of Fiber Supplementation on Cardio-Vascular Risk Parameters in Type 2 Diabetes Arterioscler Thromb Vasc Biol 25 (5): e43. 2005.

83. SJ Passi, SC Manchanda, S Suri, S Bhandari and H Kalra, Lipid lowering effect of dietary Fiber supplementation through food based approach *ICCN* Poster Presentations;Clinical nutrition: diagnosis and management *Asia Pacific Journal of Clinical Nutrition (2004) 13 (Suppl)* S155.
84. Tseng CC, Kieffer TJ, Jarboe LA, Usdin TB, Wolfe MM. Postprandial stimulation of insulin releases by glucose-dependent insulinotropic polypeptide (GIP): Effect of a specific glucose-dependent insulinotropic polypeptide receptor antagonist in the rat. J Clin Invest 1996;98:2440 –5.
85. Van Dijk G, Thiele TE. Glucagon-like peptide-I (7–36) amide: a central regulator of satiety and interoceptive stress. Neuropeptides 1999;33:406 –14.
86. Bourdon I, Olson B, Rackus R, Richter BD, Davis PA, Schneeman BO. Beans, as a source of dietary fiber, increase cholecystokinin and apolipoprotein B48 response to test meals in men. J Nutr 2001;131:1485–90.
87. Holt SHA, Brand-Miller JC, Stitt PA. The effects of equal-energy portions of different breads on blood glucose levels, feelings of fullness and subsequent food intake. J Am Diet Assoc 2001;101:767–73.
88. Burton-Freeman B, Davis PA, Schneeman BO. Plasma cholecystokinin is associated with subjective measures of satiety in women. Am J Clin Nutr 2002;76:659–67.
89. Bourdon I, Yokoyama W, Davis P, Hudson C, Backus R, Richter D, et al. Postprandial lipid, glucose, insulin, and cholecystokinin responses in men fed barley pasta enriched with b-glucan. Am J Clin Nutr 1999;69:55– 63.
90. Schneeman BO. Gastrointestinal physiology and functions. Br J Nutr 2002;88(suppl 2):S159–63.
91. Bergmann JF, Chassany O, Pepit A, Triki R, Caulin C, Segrestaa JM. Correlation between echographic gastric emptying and appetite: influence of psyllium. Gut 1992;33:1042–3.
92. Yao M, Roberts SB. Dietary energy density and weight regulation. Nutr Rev 2001;59:247–58.
93. David J. A. Jenkins, Mette Axelsen, Cyril W. C. Kendall, Livia S. A. Augustin, Vladimir Vuksan and Ulf Smith, Dietary Fiber, lente carbohydrates and the insulin-resistant diseases British Journal of Nutrition (2000), 83, Suppl. 1, S157–S163.
94. Rimm EB, Ascherio A, Giovannucci E, Spiegelman D, Stampfer MJ, Willett WC.Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. JAMA 1996; 275:447-51.
95. Krauss et al., Circulation, 102:2296-2311, 2000.
96. Khaw, K.T., and Barrett-Connor, E. 1987. Dietary fiber and reduced ischemic heart disease mortality rates in men and women: a 12-year prospective study. Am J Epidemiol 126(6):1093-1102.
97. Brown, L., Rosner, B., Willett, W.W., and Sacks, F.M. 1999. Cholesterol lowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr 69:30-42.
98. Davidson, M.H., Dugan, L.D., Burns, J.H., Bova, J., Story, K., and Drennan, K.B. 1991. The hypocholesterolemic effects of beta-glucan in oatmeal and oat bran, a dose controlled study. JAMA 265(14):1833- 1839.
99. US Food and Drug Administration Health Claims: Soluble fiber from certain foods and risk of heart diseases. Code of Federal Regulations 2001;21:101.81.

100. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. 2001. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel Ill). JAMA 2002;285:2486-2497.

101. Marlett JA. Sites and mechanisms for the hypocholesterolemic actions of soluble dietary fiber sources. In *Dietary Fiber in Health and Disease*, eds Kritchevsky D and Bonfield C. Plenum Press, New York, NY 1997, 109-121.

102. Anderson SA, Fisher KD, Talbot JM. *Evaluation of the Health Aspects of Using Partially Hydrolyzed Guar Gum as a Food Ingredient*, Federation of American Societies for Experimental Biology, Bethesda, MD 1993. 1-61.

103. Lia Å, Hallmans G, Sandberg A-S, Sundberg B, Aman P, Andersson H. Oat B-glucan increases bile acid excretion and a fiber-rich barley fraction increases cholesterol excretion in ileostomy subjects. Am J Clin Nutr 1995;62:1245-1251.

104. Davidson MH, Dugan LD, Stocki J, Dicklin MR, Maki KG, Coletta F, Cotter R, McLeod M, Hoersten K. A low-viscosity soluble-fiber fruit juice supplement fails to lower cholesterol in hypercholesterolemic men and women. J Nutr 1998;128:1927-1932.

105. Everson GT, Daggy BP, McKinley C, Story JA. Effects of psyllium hydrophilic mucilloid on LDL-cholesterol and bile acid synthesis in hypercholesterolemic men. J Lipid Res 1992;33:1183-1192.

106. Marlett JA, Hosig KB, Vollendorf NW, Shinnick FL, Haack VS, Story JA. Mechanism of serum cholesterol reduction by oat bran. Hepatology 1994;20:1450-1457.

107. Hillman LC, Peters SG, Fisher CA, Pomare EW. Effects of Fiber components pectin, cellulose, and lignin on bile salt metabolism and biliary lipid composition in man. Gut 1986;27:29-36.

108. Jenkins DJ, Wolever TM, Rao AV, Hegele RA, Mitchell SJ, Ranson TP, Boctor DL, Spadafora PJ, Jenkins AL, Mehling C. Effect of blood lipids of very high intakes of fiber in diets low in saturated fat and cholesterol. N EnglJMed 1993;329:21-26.

109. Haack VS, Chesters JG, Vollendorf NW, Story JA, Marlett JA. Increasing amounts of dietary fiber provided by foods normalizes physiologic response of the targe bowel without altering calcium balance or fecal steroid excretion. Am J Clin Nutr 1998;68:615-622.

110. Van Horn, L.V., Kiang Liu, R.D., Parker, D., Emidy, L., Liao, Y., Pan, W.H., Giumetti, D., Hewitt, J., and Stamler, J. 1986. Serum lipid response to oat product intake with a fat-modified diet. J Am Dietetic Assoc. 86(6):759-764.

111. Demark-Wahnefried, W., Bowering, J., and Cohen, P.S. 1990. Reduced serum cholesterol with dietary change using fat-modified and oat bran supplemented diets. J Am Diet Assoc 90:2223-2229.

112. Kirby, R.W., Anderson, J.W., Sieling, B., Rees, E.D., Chen, W-J.L., Miller, R.E., and Kay, R.M. 1981. Oat-bran intake selectively lowers serum low-density lipoprotein cholesterol concentrations of hpercholesterolemic men. Am J Clin Nutr 34:824-829.

113. Van Horn, L., Emidy, L. A., Liu, K., Liao, Y., Ballew, C., King, J., and Stamler, J. 1988. Serum lipid response to a fat-modified, oatmeal enhanced diet. Preventive Medicine 17:377-386.

114. Oda, T., Aoe, S., Sanada, H., and Ayano, Y. 1993. Effects of soluble and insoluble fiber preparations

isolated from oat, barley, and wheat in liver cholesterol accumulation in cholesterol-fed rats. J Nutr Sci Vitaminol 39:73-79.

115. Veldman FJ, Nair CH, Vorster HH, Vermaak WJ, Jerling JC, Oosthuizen W, Venter CS. Dietary pectin influences fibrin network structure in hypercholesterolaemic subjects Thromb Res. 1997 May 1;86(3):183-96.

116. Bazzano, L. A., He, J., Ogden, L. G., Loria, C., Vupputuri, S., Myers, L., Whelton, P. K., Legume consumption and risk of coronary heart disease in US men and women: NHANES I Epidemiologic Follow-up Study." Archives of Internal Medicine 2001 Nov 26;161(21):2573-2578.

117. Liu, S., Buring, J. E., Sesso, H. D., Rimm, E. B., Willett, W. C., Manson, J. E.,A prospective study of dietary fiber intake and risk of cardiovascular disease among women." Journal of the American College of Cardiology 2002 Jan 2; 39(1):49-56.

118. Rao DN, Ganesh B, Rao RS, Desai PB. Risk assessment of tobacco, alcohol and diet in oral cancer-a case-control study. Int J Cancer 1994;58:469-73 . Sinha R, Anderson DE, McDonald SS, Greenwald P Symposium on Cancer Risk and Diet in India, 2003 49 (3), 222 – 228.

119. Saxena S, Szabo CI, Chopin S, Barjhoux L, Sinilnikova O, Lenoir G et al. BRCA1 and BRCA2 in Indian breast cancer patients. Hum Mutat 2002;20:473-4

120. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition, and the prevention of cancer: a global perspective. Washington DC:American Institute for Cancer Research.1997: pp. 506-7.

121. Gopalan C. Rising incidence of obesity, coronary heart disease and diabetes in the Indian urban middle class. Possible role of genetic and environmental factors. World Rev Nutr Diet 2001;90:127-43.

122. Cummings JH, Bingham SA, Heaton KW, Eastwood MA. Fecal weight,colon cancer risk and dietary intake of nonstarch polysaccharides (dietary fiber). Gastroenterology.1992;103:1783-1789.

123. Howe GR, Benito E, Castelleto R, Cornee J, Esteve J, Gallagher RP, Iscovich JM, Deng-ao J, Kaaks R, Kune GA. Dietary intake of fiber and decreased risk of cancers of the colon and rectum: Evidence from the combined analysis of 13 case-control studies. J Natl Cancer Inst. 1992; 84:1887-1896.

124. Kim YI. AGA technical review: Impact of dietary fiber on colon cancer occurrence. Gastroenterol. 2000;118:1235-1257.

125. Malhotra SL. Dietary factors in a study of cancer colon from cancer registry, with special reference to the role of saliva, milk and fermented milk products and vegetable Fiber. Med Hypotheses 1977; 3:122-34.

126. Segal I, Hassan H, Walker ARP, Becker P, Braganza J. Faecal short chain fatty acids in South African urban Africans and whites. Dis Colon Rectum 1995;38:732-4.

127. Pool-Zobel BL, Neudecker C, Domizlaff I, et al. Lactobacillus - and Bifidobacterium - mediated antigenotoxicity in the colon of rats. Nutr Cancer 1996;26:365-80.

128. Hague A, Elder DJE, Hicks DJ, Paraskeva C. Apoptosis in colorectal tumour cells: induction by the short chain fatty acids butyrate, propionate and acetate and by the bile salt deoxycholate. Int J Cancer 1995;60:400-6.

129. Marchetti C, Migliorati G, Moraca R, et al. Deoxycholic acid and SCFA-induced apoptosis in the human tumor cell-line HT-29 and possible mechanisms. Cancer Lett 1997;114:97-9.

130. Hass R, Busche R, Luciano L, Reale E, Engelhardt W. Lack of butyrate is associated with induction of bax and subsequent apoptosis in the proximal colon of guinea pig. Gastroenterology 1997;112:875-81.

131. Prentice RL. Future possibilities in the prevention of breast cancer: fat and fiber and breast cancer research. Breast Cancer Res. 2000;2:268-276.

132. Adlercreutz H. Evolution, nutrition, intestinal microflora, and prevention of cancer: a hypothesis. Proc. Soc Exp Biol Med. 1998; 217:241-246.

133. Howe GR, Hirohata T, Hislop TG, Iscovich JM, Katsouyanni K, Lubin F,Marubini E, Modan B, Rohan T. Dietary factors and risk of breast cancer:Combined analysis of 12 case-control studies. J Natl Cancer Inst. 1990;82:561-569.

134. Baghurst PA, Rohan TE. Dietary fiber and risk of benign proliferative epithelial disorders of the breast. Int J Cancer. 1995;63:481-485.

135. Rose DP, Lubin M, Connally JM. Effects of diet supplementation with wheat bran on serum estrogen levels in the follicular and luteal phases of the menstrual cycle. Nutrition. 1997;13:535-539.

136. Haggans CJ, Travelli EJ, Thomas W, Martini MC, Slavin JL. The effect of flaxseed and wheat bran consumption on urinary estrogen metabolites in premenopausal women. Cancer Epidemiol Biomarkers Prev. 2000;9:719-725.

137. La Vecchia C, Ferraroni M, Franceschi S, Mezzetti M, Decarli A, Negri E. Fibers and breast cancer risk. *Nutr Cancer.* 1997;28: 264-269.

Dear Doctor,

It his been our endeavour to provide the medical fraternity, the latest thinking on a variety of medical topics, a tradition that we have been following for over 55 years through our Quarterly Medical Reviews.

This booklet is presented to you by Raptakos Team & Eidikos Team of Raptakos Brett, & Co. Ltd.

We would very much like to have your valuable suggestions and comments to make our future issues more meaningful to you.

We will appreciate if you could spend a few minutes to fill in your comments and mail the same to us.

Thanking you,

MEDICAL ADVISOR

FEED BACK October - December 2005

1. Your comments on this issue of the Q.M.R.

   ....

   ....

2. Please suggest medical topics for our QMRs which could be printed in future.

   ....

   ....

3. Any other suggestions / comments.

   ....

   ....

Name: Dr. .... M / F

Clinic Address: ....

....

City: .... State: ....Pin: ....

Tel: ....E-mail: ....

Qualifications: ....

Please mail this form to:

Vice President (Medical)
RAPTAKOS, BRETT & CO. LTD.
Dr. Annie Besant Road, Worli,
Mumbai 400 030.