Dietary fiber in health and disease: An overview

David Kritchevsky PhD

The Wistar Institute, Philadelphia, Pennsylvania, USA

What we now know as the Vahouny Symposia on Dietary Fiber were originally called the George Washington University Symposia on Dietary Fiber. These Symposia were conceived by George Vahouny and renamed in Vahouny's honor after his untimely death in 1986. The first Symposia centered on definitions, terminology, mineral availability, diabetes, obesity, lipid absorption, and bile acids and cancer.[1] The only holdovers for the second Symposia were obesity and the bile acid–cancer connection.[2] New topics were analytical methodology, glycemic index and gallstones. The first Vahouny Symposia consisted of a review of mineral absorption, mechanisms underlying the hypocholesterolemic action of fiber, bile acid turnover, colon cancer epidemiology and short chain fatty acids (SCFA).[3] The second Vahouny Symposia examined structure–function relationships, lipids, bile acids, mineral metabolism and SCFA.[4] The most recent Vahouny Symposia, the third in the series, examined energy regulation, lipids, oligosaccharides, SCFA and reviewed what we know about fiber and heart disease, and about fiber and cancer.[5]

The remaining major topics to be covered are aspects of sterol metabolism, nutrient absorption and SCFA. We have a wealth of observational data but have not been able to arrive at any intellectually satisfying general mechanisms. We have concentrated on the effects of fiber on colon cancer, partly because the colon is the organ where fiber fermentation takes place and because of the emphasis placed on fiber and colon cancer by Burkitt.[6] The ways in which fiber can influence colon cancer have been discussed at length and include physical dilution of colon content; absorption of bile acids and carcinogens; decreased transit time; altered bile acid metabolism; and the effects of fermentation, namely, the production of SCFA, lowering of pH and stimulation of bacterial growth.

It is fitting to recall that Hellendoorn in 1978 suggested that fermentation was the principal cause underlying the physiologic activity of indigestible food residues.[7] He cited the principal effects as being due to the following: gas formation leading to production of CO_2, H_2, CH_4, flatulence, accelerated transit and soft voluminous stools; acid production, which accelerated transit, increased stool weight and provided SCFA; and other reactions such as a reduction of cholesterol to coprostanol, increased production of secondary bile acids and binding of secondary bile acids by bacteria.

It is now fairly well accepted that diets high in cereal grains offer protection against colon cancer. The data have been reviewed and discussed by Hill.[8,9] There is interest in the possible effects of fiber on cancers other than those of the large bowel. There are data which suggest that dietary fiber may influence breast cancer. The effects of fiber in breast cancer reflect another property of fiber, namely, the ability to bind organic compounds. The binding of estrogens to fiber may affect the course of mammary cancer. Rose pointed out that vegetarians excrete more estrogen in their faeces than do omnivores, who excrete more urinary estrogen than do vegetarians.[10] The possibility was suggested that estrogen binding to fiber affects estrogen metabolism and excretion patterns. Rose *et al.* fed premenopausal women wheat bran, oat bran or corn bran for several months.[11] Wheat bran in the diet resulted in a 15% reduction in plasma estradiol and a 20% reduction in plasma estrone; both differences were statistically significant. Oat bran increased estradiol and estrone levels by 13% and 6%, respectively. Corn bran had little effect on estradiol levels (0.6% increase) and increased estrone levels by 8%.

Baghurst and Rohan studied 451 breast cancer patients and an equal number of controls.[12] They found a significant negative correlation between relative risk and fiber intake. Attempts to link fiber intake to protection against other forms of cancer have yielded weak connections, if any. The association between dietary fiber and pancreatic cancer is weak,[13,14] and there is no connection between diet and prostate cancer.[15]

A high fiber diet is not a low fiber diet to which fiber, in some form, has been added. It is a diet high in plant foods which contain a large number of substances which have been shown to have anticancer activity. A partial list is presented in Table 1. Phenyl isothiocyanate, ellagic acid and flavonoids may inhibit binding of carcinogens to DNA. Carotenoids, tocopherols and flavonoids are antioxidants and can prevent oxidative damage. Phytosterols and terpenes, as well as fiber, can prevent action of steroid hormones. However, it may be that no single substance is effective in preventing cancer but rather that an interaction of several substances results in protective action. The studies with beta carotene should be viewed as cautionary. Low plasma levels of beta carotene have been associated with increased all-cause mortality[16] but administration of beta carotene to smokers has resulted in an increased incidence of lung cancer.[17] The plasma levels of beta carotene could be attributed to a diet which contained a

Correspondence address: Dr David Kritchevsky, The Wistar Institute, 3601 Spruce Street, Philadelphia, PA 19104, USA.
Tel: 1 215 898 3713; Fax: 1 215 898 3868
Email: kritchevsky@wistar.upenn.edu

Table 1. Phytochemicals present in a fiber-rich diet

Allium compounds
Carotenoids
Flavonoids
Folic acid
Glucosinolates
Isoflavones
Isothiocyanates
Phytosterols
Saponins
Selenium
Thiocyanates
Vitamins C and E

number of substances including beta carotene. This could be an interactive or promoting effect.

The role of fiber in reducing risk of coronary disease may go beyond the hypocholesterolemic properties of soluble fibers. Humble recently summarized the available data.[18] All the studies showed a protective effect of fiber when the data were unadjusted or age adjusted.[19–24] In two of the studies the correlation of protection with fiber weakened or disappeared after further adjustment for caloric intake.[20,23] The daily fiber intakes in the studies cited ranged from 12 to 30 g/day. Khaw and Barrett-Connor found a marked reduction of risk in a cohort of 503 women aged 50–79 years.[22] Humble *et al.* studied 1801 male hypercholesterolemics aged 45–59 years for 9 years.[25] The trend for reduction of relative risk going from the lowest to highest quintile of intake was significant ($P < 0.003$). It will be interesting to follow developments in this area.

The area of fiber research which should be given greater attention is that of interactions among the nutrients in fiber-rich diets and interactions between fiber and other macronutrients.

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