# Dietary fibre

## After 21 years of study the verdict remains one of fruition and frustration

Nearly 200 new food products containing oats or oat bran were released in America last year, and according to the trade journal *Food Processing* "fiber is the most successful buzz word of today." The public has taken dietary fibre to its heart and for its heart. But does it work? Is it really the answer to heart disease as well as to all the ills of the bowel?

Some 21 years after Burkitt and Cleave blew the whistle on Western fibre depleted diets[1,2] one thing is certain: Does it work? is the wrong question. Fibre is not an "it" any more than vitamins or fats are "its." There are endless permutations of the non-starch polysaccharides (and of lignin, but I bypass that), and each one has different physicochemical properties and biological effects. All of the non-starch polysaccharides that dissolve in water to form a gel are sometimes grouped together because, in general, these soluble fibres, like guar gum and pectin, lower serum cholesterol concentration but do little to bowel function—while insoluble fibres, like cellulose and the arabinoxylans of bran, are good laxatives but ineffectual at lowering cholesterol concentration.[3] But such lumping together is to oversimplify. There are soluble fibres, such as ispaghula and xanthan gum, that are excellent laxatives[4,5] and are also good at lowering the serum cholesterol concentration[5,6] whereas karaya gum seems to do nothing either to the bowel or the blood.[7] The subtleties of polysaccharides are endless. For example, arabinoxylans, which are polymers of the five carbon sugars arabinose and xylose, may be soluble or insoluble depending on the configuration of the sugar chains.[8] Similarly, β glucans—that is, long chains of glucose joined by B links—may be freely soluble as in oats or poorly soluble as in barley depending on the stereochemistry of the intersugar links.[9]

If a chemical approach to fibre leads us into a maze perhaps we should go back to basics and ask "What is fibre there for? What is it doing in the plant?" If we leave aside gums and mucilages fibre may be equated with cell walls, and the function of cell walls is structural and mechanical. Cell walls provide the skeleton of the plant; they give to the plant—and to our food if it is plant based—integrity, solidity, and firmness. Cell walls make up only 2-3% of the weight of fruits and vegetables, yet they are wholly responsible for the difference in form between apples and apple juice, carrots and carrot juice, olives and olive oil.

Solidity and stiffening are the key words. When Eastwood called fibre "potent pith" he did not intend to raise a simile,[10] but fibre does lose some of its characteristic effects on the human body if it is treated in such a way that food becomes soft and limp. In a much quoted study Haber *et al* compared apples, apple puree, and apple juice in terms of rate of ingestion, satiating power, and insulin responses.[11] In every respect the puree behaved quite like the juice even though it contained all the fibre of the apples. Similarly, in a study with peanuts the gut reacted to peanut butter much like it reacted to peanut oil in terms of the efficiency of absorption.[12] In both studies the fibre was all there in a chemical sense—but it was well nigh gone in a physical sense, like a violin that has been stamped on.

Even the laxative properties of fibre may have a mechanical component. Tomlin and Read fed volunteers little pieces of plastic the size of wheat bran particles and found that they were just as effective as bran itself in terms of bulking the stools and speeding up intestinal transit.[13] These workers suggest that the pieces of plastic work by tactile stimulation of multimodal receptors in the colonic mucosa—Does bran work in the same way? This is a revolutionary concept and may rehabilitate the derided term "roughage." Certainly the laxative effect of wheat bran may be destroyed or reduced by milling into fine particles[14,15] or by cooking.[16] There is more at stake here than stool bulk. As transit speeds up the bacterial biomass in the colon increases,[17] fewer bacterially degraded bile salts enter the body,[18] and the composition of bile changes in a way that makes gall stones less likely.[18]

### Cancer of the large bowel

There is a cancerous twist to this story. People with premalignant adenomatous polyps of the colon are prone to make or absorb excessive amounts of deoxycholate, the main bacterially degraded bile salt,[19,20] which has mutagenic or cocarcinogenic potential.[21] As absorption of deoxycholate is sensitive to changes in the rate of intestinal transit it is an attractive possibility that bran or other agents that speed up intestinal transit will reduce the risk of cancer of the colon.

One way of testing this idea is to give bran to people whose adenomatous polyps have been removed and see if it prevents them from recurring. Several studies of this type are in progress, and the results of the first one have recently been reported.[22] To get quick results the authors selected patients with familial polyposis coli. When these patients are treated by colectomy and ileorectal anastomosis the adenomatous polyps are very prone to recur in the rectum and to turn malignant. When 19 such patients ate a bran based breakfast cereal in amounts sufficient to double their fibre intake and underwent sigmoidoscopy regularly for four years the number of rectal polyps decreased compared with that in two randomly assigned control groups. The bran was enriched with vitamins C and E, but one of the control groups received these vitamins as well, so there seems no doubt that the bran had an antineoplastic effect. It remains to be seen whether bran will be effective in patients with the far commoner non-familial type of colonic adenoma and in people with intact colons. There is a preliminary report that treatment with lactulose slows down the recurrence of such polyps,[23] which is interesting because lactulose is not simply a laxative but a fermentable carbohydrate like fibre.

There are many ways in which a high fibre intake "ought to" protect against colorectal cancer,[24] yet low fibre intakes are not a consistent feature of populations prone to this cancer nor of people who actually have it.[25,26] One of several possible explanations is that fibre is only part of the story. If fibre exerts its anticancer effect by being fermented into short chain fatty acids then any carbohydrate that enters the colon and is similarly fermented could be protective. Quite a lot of the starch we eat escapes digestion and enters the colon, where it is rapidly fermented[27] and probably has some laxative effect.[28] So perhaps starch intake matters as much as fibre intake in terms of protection from cancer. What we really

need to know, however, is not only how much starch is eaten but also what proportion gets into the colon, and this varies 10-fold from person to person.[29] One small study has suggested that people with colonic polyps are unusually efficient at digesting starch.[30] If this is confirmed it will open up the possibility that cancer might be prevented by people eating more starch and by eating it in less digestible form—for example, in whole grains such as rice rather than milled grains such as flour.[31 32] It seems that in general rice eating populations are less prone to colorectal cancer than flour eating ones.

Yet another aspect of this story is that a diet may be rich in fibre because it contains plentiful fruit and vegetables, and these carry their own anticancer benefits such as vitamin A, antioxidants, and trace elements. There are both epidemiological[33] and experimental[22] pointers to a vegetable rich diet being protective against colorectal cancer. To put all of this together leads to an anticancer regimen that sounds very like the generally accepted guidelines to healthy eating; eat lots of plant based food and eat much of it in a fresh, unprocessed form. The fibre hypothesis may have led us up the garden path, but the garden is not a bad place for a path to go.

## Heart disease

As for the question: Does a high fibre intake protect against heart disease?—again the answer has to be hedged. Four prospective studies suggest that it does,[34-37] and no prospective study has shown the opposite, but—in contrast with the links with cancer of the bowel—the mechanism is far from clear. Fibre may be a surrogate for associated dietary factors such as antioxidants,[38] but the story that hit the headlines and excited the processed food industry was the appealingly simple one "oats are hypocholesterolaemic." It looked a good story, even if it meant eating very large or very sticky bowls of porridge (the sticky ones being made with oatbran) or, for the Americans, eating lots of bran muffins. But on 18 January this year the *New England Journal of Medicine* made the story look distinctly thin. It published a carefully controlled study in which adding oat bran to the diet had an effect on total and low density lipoprotein cholesterol concentrations that was no more than that of a refined wheat product low in fibre and no more than would be predicted from the coincident fall in saturated fat and cholesterol intake.[39] The conclusions were promptly accepted,[40] but their validity must be questioned. The volunteers were slim young women with normal cholesterol concentrations (mean 4·8 mmol/l) and the results might have been different in stout middle aged people with serum cholesterol concentrations of 7-8 mmol/l. No mention was made that the important ratio of low density to high density lipoprotein cholesterol was lower in those eating oat bran than in those eating refined wheat (1·92 *v* 2·11). The dietary assessments were crude and inconsistent. Indeed, when the authors calculated the expected change in serum cholesterol concentration they compared values for fat intake derived from two different types of dietary record—a food frequency questionnaire and a four day record—so like was not compared with like. The authors' conclusion that foods rich in fibre can be beneficial in a negative way by displacing less desirable elements in the diet may well be correct. But this paper does not overturn all the studies, some with careful control of fat intake, that found oats to be hypocholesterolaemic.[41-45]

For people who wanted a simple panacea dietary fibre has proved annoyingly subtle. In one sense it is indeed a "potent pith," but in another it is "an abstraction,"[46] and, in yet another, a fertile "paradigm."[47] The same could be said of vitamins, knowledge of which has come a long way since Casimir Funk's vital amines of 1912. Future research will tend to concentrate on specific components of the dietary fibre complex, but unless it includes studies of the whole cell wall and of whole food that is histologically intact we may lose sight of the wood for the trees.

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1 Burkitt DP. Related disease—related cause? *Lancet* 1969;ii:1229-31.
2 Cleave TL, Campbell GD, Painter NS. *Diabetes, coronary thrombosis and the saccharine disease*. 2nd edn. Bristol: Wright, 1969.
3 Jenkins DJA, Reynolds D, Leeds AR, Waller AL, Cummings JH. Hypocholesterolemic action of dietary fiber unrelated to fecal bulking effect. *Am J Clin Nutr* 1979;**32**:2430-5.
4 Stevens J, Van Soest PJ, Robertson JB, Levitsky DA. Comparison of the effects of psyllium and wheat bran on gastrointestinal transit time and stool characteristics. *J Am Diet Assoc* 1988;**88**:323-6.
5 Eastwood MA, Brydon WG, Anderson DMW. The dietary effects of xanthan gum in man. *Food Addit Contam* 1987;**4**:17-26.
6 Anderson JW, Zettwoch N, Feldman T, Tietyen-Clark J, Oeltgen P, Bishop CW. Cholesterol lowering effects of psyllium hydrophilic mucilloid for hypercholesterolemic men. *Arch Intern Med* 1988;**148**:292-7.
7 Eastwood MA, Brydon WG, Anderson DMW. The effects of dietary gum karaya (Sterculia) in man. *Toxicol Lett* 1983;**17**:159-66.
8 Selvendran RR. Chemistry of plant cell walls and dietary fibre. *Scand J Gastroenterol* 1987;**22** (suppl 129):33-41.
9 Åmon P, Graham H. Mixed-linked β-(1→3), (1→4)-D-glucans in the cell walls of barley and oats—chemistry and nutrition. *Scand J Gastroenterol* 1987;**22** (suppl 129):42-51.
10 Eastwood MA. Vegetable dietary fibre—potent pith. *J R Soc Health* 1975;**95**:188-90.
11 Haber GB, Heaton KW, Murphy D, Burroughs L. Depletion and disruption of dietary fibre. Effects on satiety, plasma-glucose, and serum-insulin. *Lancet* 1977;ii:679-82.
12 Levine AS, Silvis SE. Absorption of whole peanuts, peanut oil, and peanut butter. *N Engl J Med* 1980;**303**:917-8.
13 Tomlin J, Read NW. The laxative effects of indigestible plastic particles. *Br Med J* 1988;**297**: 1175-6.
14 Wrick KL, Robertson JB, Van Soest PJ, *et al*. The influence of dietary fiber source on human intestinal transit and stool output. *J Nutr* 1983;**113**:1464-79.
15 Cummings JH. The effect of dietary fiber on fecal weight and composition. In: Spiller GA, ed. *CRC handbook of dietary fiber in human nutrition*. Boca Raton: CRC Press, 1986:211-80.
16 Wyman JB, Heaton KW, Manning AP, Wicks ACB. The effect on intestinal transit and the feces of raw and cooked bran in different doses. *Am J Clin Nutr* 1976;**29**:1474-9.
17 Stephen AM, Cummings JH. Mechanism of action of dietary fibre in the human colon. *Nature* 1980;**284**:283-4.
18 Marcus SN, Heaton KW. Intestinal transit, deoxycholic acid and the cholesterol saturation of bile-three inter-related factors. *Gut* 1986;**27**:550-8.
19 van der Werf SDJ, Nagengast FM, van Berge Henegouwen GP, Huijbregts AWM, van Tongeren JHM. Colonic absorption of secondary bile acids in patients with adenomatous polyps and in matched controls. *Lancet* 1982;i:759-62.
20 van der Werf SDJ, Nagengasts FM, van Berge Henegouwen GP, Huijbregts AW, van Tongeren JHM. Intracolonic environment and the presence of colonic adenomas in man. *Gut* 1983;**24**: 876-80.
21 Nagengast FM. Bile acids and colonic carcinogenesis. *Scand J Gastroenterol* 1988;**23**(suppl 154): 76-81.
22 DeCosse JJ, Miller HH, Lesser ML. Effect of wheat fiber and vitamins C and E on rectal polyps in patients with familial adenomatous polyposis. *J Natl Cancer Inst* 1989;**81**:1290-7.
23 Ponz de Leon M, Roncucci L, diDonato P, *et al*. Vitamins A, C and E and lactulose in the prevention of recurrence of adenomatous polyps: preliminary results of a controlled study. *Gut* 1989;**30**:A1511-2.
24 Cummings JH, Bingham SA. Dietary fibre, fermentation and large bowel cancer. *Cancer Surv* 1987;6:601-21.
25 Jacobs LR. Fiber and colon cancer. *Gastroenterol Clin North Am* 1988;**17**:747-60.
26 Rozen P, Horwitz C, Tabenkin C, Ron E, Katz L. Dietary habits and colorectal cancer incidence in a second-defined kibbutz population. *Nutr Cancer* 1987;**9**:177-84.
27 Cummings JH, Englyst HN. Fermentation in the human large intestine and the available substrates. *Am J Clin Nutr* 1987;**45**:1243-55.
28 Shetty PS, Kurpad AV. Increased starch intake in the human diet increases fecal bulking. *Am J Clin Nutr* 1986;**43**:210-2.
29 Stephen AM, Haddad AC, Phillips SF. Passage of carbohydrate into the colon. Direct measurements in humans. *Gastroenterology* 1983;**85**:589-95.
30 Thornton JR, Dryden A, Kelleher J, Losowsky MS. Super-efficient starch absorption. A risk factor for colonic neoplasia? *Dig Dis Sci* 1987;**32**:1088-91.
31 Heaton KW, Marcus SN, Emmett PM, Bolton CH. Particle size of wheat, maize and oat test meals: effects on plasma glucose and insulin responses and on the rate of starch digestion in vitro. *Am J Clin Nutr* 1988;**47**:675-82.
32 O'Dea K, Nestel PJ, Antonoff L. Physical factors influencing postprandial glucose and insulin responses to starch. *Am J Clin Nutr* 1980;**33**:760-5.
33 Slattery ML, Sorenson AW, Mahoney AW, French TK, Kritchevsky D, Street JC. Diet and colon cancer: assessment of risk by fiber type and food source. *J Natl Cancer Inst* 1988;**80**:1474-80.
34 Khaw K-T, Barrett-Connor E. Dietary fiber and reduced ischemic heart disease mortality rates in men and women: a 12-year prospective study. *Am J Epidemiol* 1987;**126**:1093-102.
35 Morris JN, Marr JW, Clayton DG. Diet and heart: a postscript. *Br Med J* 1977;ii:1307-13.
36 Kromhout D, Bosschieter EB, de Lozenne Coulander C. Dietary fibre and 10-year mortality from coronary heart disease, cancer, and all causes. The Zutphen study. *Lancet* 1982;ii:518-22.
37 Kushi LH, Lew RA, Stare FJ, *et al*. Diet and 20-year mortality from coronary heart disease. The Ireland-Boston diet-heart study. *N Engl J Med* 1985;**312**:811-8.
38 James WPT, Duthie GG, Wahle KWJ. The Mediterranean diet: protective or simply non-toxic? *European Journal of Clinical Nutrition* 1989;suppl 2:31-41.
39 Swain JF, Rouse IL, Curley CB, Sacks FM. Comparison of the effects of oat bran and low-fiber wheat on serum lipoprotein levels and blood pressure. *N Engl J Med* 1990;**322**:147-52.
40 Connor WE. Dietary fiber—nostrum or critical nutrient? *N Engl J Med* 1990;**322**:193-5.
41 Kirby RW, Anderson JW, Sieling B, *et al*. Oat-bran intake selectively lowers serum low-density lipoprotein cholesterol concentrations of hypercholesterolemic men. *Am J Clin Nutr* 1981;**34**: 824-9.
42 Anderson JW, Story L, Sieling B, *et al*. Hypocholesterolemic effects of oat bran or bean intake for hypercholesterolemic men. *Am J Clin Nutr* 1984;**40**:1146-55.
43 Van Horn LV, Liu K, Parker D, *et al*. Serum lipid response to oat product intake with a fat-modified diet. *J Am Diet Assoc* 1986;**86**:759-64.
44 Vorster HH, Lotter AD, Odendaal I. Effects of an oat fibre tablet and wheat bran in healthy volunteers. *S Afr Med J* 1986;**69**:435-8.
45 Turnbull WH, Leeds AR. Reduction of total and LDL-cholesterol in plasma by rolled oats. *J Clin Nutr Gastroenterol* 1987;2:177-81.
46 Hellendoorn EW. Dietary fiber or indigestible residue? *Am J Clin Nutr* 1981;**34**:1437-9.
47 Trowell H. Dietary fibre—a paradigm. In: Trowell H, Burkitt D, Heaton K, eds. *Dietary fibre, fibre-depleted foods and disease*. London: Academic Press, 1985:1-20.