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*Invited Commentary*

# Is the dietary fibre concept becoming too hard to digest?

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From saviour of the nation to the axis of evil – has any nutrient ever received such a mixed reception as dietary fibre? Early writings left little doubt that we should be eating more of it[1]. When I was a young student, a visit from the great Denis Burkitt left a strong impression, not only because of his apparently unlimited energy (possibly a tribute to his personal dietary fibre intake), but also because of his unashamed illustration of contents of toilet bowls as a means of demonstrating what he believed was and was not important in the dietary fibre field. His concept was simple: dietary fibre meant plant cell walls. It was beneficial to health, including reducing the risk of colorectal cancer, if and only if you could graphically illustrate the consequences of eating it. If only things still appeared that simple!

If something is good for health, then the entrepreneurs in the food industry will be looking to ways of packaging and marketing a product. However, to market something containing dietary fibre with an implied health benefit, then dietary fibre needs to be definable. A large number of health claims have come unstuck, and large numbers of expert groups have spent countless hours debating this very issue[2]. Should dietary fibre be defined on the basis of a chemical analysis or does it require a physiological definition? If chemistry is to prevail, which analytical method is to be used? And if physiology, which properties are we sure will benefit health? Is digestion and fermentation, with concomitant effects on growth of colonic cells, a virtue by means of protecting against colonic atrophy[3], or a negative effect that predicts enhanced carcinogenesis[4]?

Scientists by definition are a sceptical group, who will only accept a definition based on hypothesis if there is solid proof that it is correct. Many animal studies have been viewed with suspicion, because they typically utilise non-physiological conditions. For example, many of the animal studies considering effects of dietary fibre on protection against colon cancer have utilised carcinogens at such high doses that they overcome normal physiological effects[5]. In the nutrition field, the 'gold standard' is a randomised, double-blind, placebo-controlled trial in human subjects. However, where these have been done, at very considerable expense, results have generally been negative or show no effect[6,7]. Not only have these trials caused doubts as to whether dietary fibre is beneficial, they have added little to mechanistic understandings. The expense and practical difficulties of human studies may justify re-examining some of the fibre hypotheses in relevant animal models. The paper by Mandir *et al.*[8] in this issue of the *British Journal of Nutrition* does just that.

One of the key areas of debate surrounding physiological definitions of dietary fibre is the question as to whether or not fermentation is desirable. Resistant carbohydrates are those that are not digested, absorbed or fermented in the small intestine. Thus, it is highly appropriate to explore the role of fermentation by considering diets that are high in different sources of resistant carbohydrates, likely to be encountered in a normal human diet. This question was addressed by Mandir *et al.*[8] by comparing a normal diet with a semi-synthetic diet, and a semi-synthetic diet high (20 %) in either apple pomace (generally considered highly fermentable) or in bran (considered less fermentable). 'Apple pomace' was the pulp remaining after Rome beauty apples were pressed for juice extraction, while 'bran' was a standard wheat bran, as sold for human consumption.

A concern about the relevance of high-dose carcinogens has led to the adoption of a model for colon carcinogenesis that does not involve a carcinogen – the cancer-prone multiple intestinal neoplasia (MIN; *Apc* $^{Min/+}$) mouse. This has been optimised for cancer chemoprevention studies, and provides an excellent model that overcomes many of the objections to high-dose carcinogen studies[9]. Although Mandir *et al.*[8] found strong effects of the various diets in the MIN mice, most effects were also seen in the wild-type mice and were thus not strain specific. Therefore, the fact that either dietary fibre preparation did lead to increased polyp risk in this model must give pause for thought.

Either animals or humans fed a carbohydrate-free elemental diet show atrophy of the colon, and that is generally considered undesirable[10]. But how much cell division is necessary, and how do we relate colonic cell number expansion to adverse effects? Crypt fission has been suggested as a major mechanism by which mutant clones of cells are able to spread in the gut. The Mandir study[8] directly measures both cell division and crypt fission in gastrointestinal tissues of their mice, and relates these to polyp number and polyp diameter, as indices of colon carcinogenesis. It was certainly true that either the apple pomace or the bran prevented the slight reductions in intestinal mass, indicating colonic atrophy, in the mice fed the semi-synthetic diets. So far, so good. However, less desirably, either bran or apple pomace significantly increased polyp number and tumour burden in the colon of the MIN mouse. Although bran was used as an example of a resistant carbohydrate, it seemed to be as effective as or even more effective than apple pomace in enhancing this. Such an observation must raise a concern, since bran is a common dietary component for humans.

In putting these results into context, it is important to consider effects of bran not only on the colon, but also on other tissues. Increased spleen weight typically provides evidence of inflammation, and may indicate intestinal blood loss[11]. Although spleen weight was significantly elevated in all of the other

three MIN mouse groups compared with the wild type, the bran diet significantly protected against this effect. Additionally, there was a trend towards a lower tumour burden in the mid or distal part of the small intestine. This may suggest that there are some positive and some negative effects associated with the wheat-bran diet. In contrast, none of the effects associated with the apple pomace diet appeared positive.

The simple design of the Mandir study[8] has allowed a direct comparison of intestinal cell division parameters with tumour burden in different parts of the small intestine and colon. The take-home message is that many of the analyses being done in relation to dietary fibre may be an oversimplification. Digestion and fermentation of dietary fibre, and increased intestinal cell turnover may be good or bad, depending upon the genotype and other dietary components. This study has moved us further forward, but it may not just be dietary fibre definitions that are currently at risk[2].

Dr L. R. Ferguson

*Discipline of Nutrition, Faculty of Medical and Health Science*
*The University of Auckland*
*Private Bag 92019*
*Auckland*
*New Zealand*
*email: l.ferguson@auckland.ac.nz*

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