The good that fibre does — new findings

Nearly everybody these days has heard about fibre: the clear message from nutritionists around the world is that we should eat more of it. But ideas about what exactly happens to fibre in the human gut have recently shifted quite dramatically.

One definition of fibre is everything of plant origin that we cannot digest — it's the 'skin and bones' of plants. But we should not think of it only as a single foodstuff: merely a smattering of processed bran. Nor is it just indigestible roughage or 'bulk' that goes right through us essentially unchanged. Within the broad term fibre, nutritionists distinguish several different types — each with its own characteristics.

Wheat bran, beloved of health food shops, is mainly lignin and cellulose. The latter is a large chain-like molecule composed of sub-units of glucose. Cellulose is the main component in the protective wall around plant cells, and contributes to the supporting tissue in leaves and stems. It is one form in which plants can store glucose — starch is another — and both resemble our own glucose-storing molecule, glycogen. Lignin is also an important plant structural component, and very indigestible.

Other types of fibre include: the gums, widely used in the food industry; pectin, a 'glue' that holds plant cells together; and a group of chemicals termed the hemicelluloses. Many people do not realise that the gums and pectins, even though they don't have the fibrous texture of cellulose-containing structures, are in fact fibre. Fruit is a good source of pectins, which are used in jam-making.

Nutritionists came to the conclusion that fibre is 'a good thing' after studying the incidence of various diseases in populations with different dietary habits. They noticed clear associations between certain patterns of nutrition and certain diseases. Conditions such as coronary heart disease, a major health problem in Australia, were rare among people consuming diets high in fibre and starch and low in saturated fat. However, this correlation does not prove that eating fibre will prevent heart disease, but if by eating more fibre you eat less fat (and that is generally the case), then certainly it will help.

Does fibre go straight through us, undigested, as has long been thought? Almost certainly no.

Diabetes may also be linked with fibre. Certainly, evidence is accumulating to suggest that large quantities of fibre in the diet can help some sufferers (see the box on page 6).

People who eat a high-fibre diet have a far lower incidence of a bowel disease called diverticulitis than a population for whom a low-fibre diet is the accepted norm. Probably part of the reason is that the chronic constipation commonly experienced with a low-fibre diet results in a build-up of pressure within the bowel, and this, coupled with straining to relieve the constipation, can damage the bowel wall.

Also, some scientists think that large amounts of fibre may act to dilute certain biologically active chemicals, formed by bacterial action on bile acids and other faecal components, that over long periods of time could induce cancer of the bowel; furthermore, fibre also speeds up the passage of faeces through the bowel, preventing long-term accumulation of these active compounds.

But does fibre go straight through us, undigested, as has long been thought? The answer that has emerged from recent research is, for most of the important types of fibre, almost certainly no.

Despite the fact that cellulose has such a strong chemical similarity to starch and glycogen, it has a slight difference that makes animals' enzymes unable to break it down into its usable glucose sub-units. An animal living off a food source rich in cellulose — as most herbivores do — relies upon bacteria and micro-organisms to produce the necessary enzymes, and keeps these useful bugs in a large warm chamber where they do their work of fermenting — or digesting — cellulose. The anatomy of the animal's digestive tract is adapted accordingly, with enlargements either at the front end (the ruminants) or the hind end (horses, rabbits, and koalas).

Although our human gut has a large bacterial population in the hind end, for years people assumed that these bacteria could only be a source of trouble for us. Later it was conceded that they had a role confined merely to supplying us with some vitamin B12 and discouraging colonisation by disease-causing bacteria. Our digestive system doesn't look much like a herbivore's, and we cannot live on grass, so it was thought that any cellulose we consume must remain undigested, contributing to the mass of our faeces.

Now scientists, among them Dr David Topping of CSIRO's Division of Human Nutrition, are finding that a great deal of the fibre we eat is actually being broken down and digested. They are certain that we cannot produce the enzymes necessary for this, so it must be the work of our resident bacteria. (Don't think that the bacteria inside you are too few and too trivial to accomplish much — in fact they weigh almost as much as your liver, the largest internal organ, and number as many as all your body cells!)

The message from nutritionists is that we should be eating more of this (above) ... and less fatty foods.

Working initially with rats and then pigs (omnivores whose digestive systems are similar to our own), Dr Topping and Mr Richard Illman examined the blood leaving the gut. They found that, in animals fed diets high in various types of fibre, this contained compounds called volatile fatty acids (VFAs) — not to be confused with fats proper.

In cows and sheep, as well as the non-ruminant herbivores such as horses, VFAs — acetic acid, propionic acid, and butyric acid — are produced by bacterial fermentation, absorbed into the bloodstream, and used as an energy source by the cells. In this way the animal profits from the cellulose, which it cannot itself digest.

Scientists had thought that fibre fermentation in other animals, such as humans and rats, does not release VFAs in any significant quantity. They reached this conclusion because they found a very low concentration of VFAs, as measured in blood. But the blood that was sampled was always peripheral venous blood — that is, easily taken from a surface vein. When Dr Topping and his colleagues sampled the hepatic portal vein, deep inside the animals, that carries blood from the gut, they found significant quantities of VFAs. Why then, was their concentration so low in peripheral blood?

Evidently, some organs in the animals' bodies were avidly removing the VFAs and using them as an energy source. Further studies at the Division, with isolated rat livers, showed that liver tissue can metabolise substantial amounts of VFAs. The heart is also known to do this.

Measuring VFAs in the hepatic portal vein of healthy humans is, of course, not possible; Dr Topping, however, was becoming convinced that these had far more importance as an energy source for us than had ever been supposed, despite their extremely low concentration in our peripheral blood.

One way to find out more was to take a sample from inside the human colon and see whether VFAs were present there. It was possible to do this in patients having bowel surgery. With the helpful collaboration of Dr Brenton Mitchell, Dr Michael Lawson, and Dr William Roediger, of Adelaide's Queen Elizabeth Hospital, Dr Topping was able to analyse samples from patients and found extremely high levels of VFAs occurring in the contents of the transverse colon, and lower levels of them further down the line in the sigmoid colon (see the diagram). His data agree closely with those obtained in the experimental animals, and the concentration of VFAs in human bowels was even greater than in the fluid of the sheep's rumen!

Further support for the idea that VFAs are produced in the human colon came from a group in Britain, who found that patients who had a significant part of the large bowel cut out, and so had less bacterial fermentation and absorption of its products, became lighter in weight than normal people with an identical diet, suggesting that the fermentation provided energy in a form the body can use.

So the evidence is fairly conclusive that bacterial fermentation of fibre to produce volatile fatty acids, which are then absorbed and used as an energy source, occurs in the human colon much as it does in the cow's rumen. Various other studies in which controlled amounts of various types of fibre were fed to human volunteers, and the fibre remaining in the faeces analysed, have shown that up to 70% of the energy in fibre may be extracted by fermentation in our large bowel. The amount of energy is not great in itself, but it may contribute about 8–10% of the total energy we receive from our food, and is thus quite significant.

New role for the colon

For decades, students of medicine and biology have learnt that the gut is a kind of assembly — or disassembly — line, with each area specialising in a particular function: storage (the stomach); digestion of all the different components of food (the stomach and the duodenum); absorption of the resulting simpler compounds (the duodenum and ileum); and finally the slow absorption of water by the unglamorous colon before the voiding of what's left. But now we find that the colon too is a site of digestion (by bacteria) and of absorption of the resulting VFAs.

Food takes several hours to reach the colon, and once there the process of fermentation also requires hours. In other words, the energy supplied from the colon arrives much later than the energy in the compounds absorbed from the duodenum, which is available within 2–4 hours of eating. So this fibre digestion provides a valuable 'spreading' over time of our energy input.

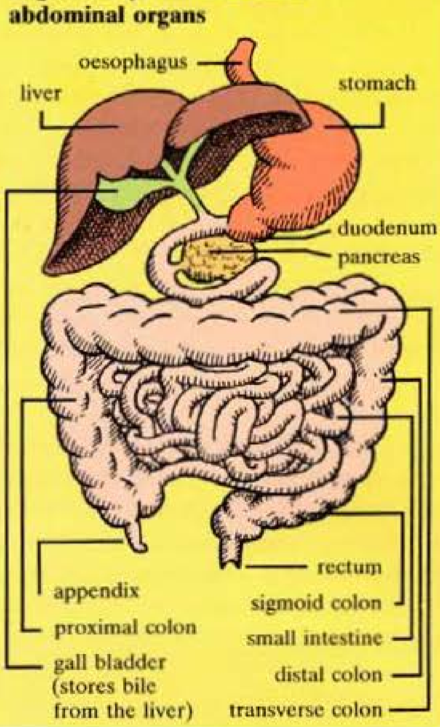
The discovery that fibre metabolism by bacteria can release energy has also upset the traditional kilojoule or calorie tables so popular with weight-watchers. These were calculated on the assumption that fibre either contributed nothing in the way of energy or actually contributed to energy loss.

How do the new findings explain the beneficial effects that we have all been led to expect of fibre? Firstly, some fibre — especially cellulose, of which we only digest about 30–40%, and lignin, which remains completely undigested — is still important as 'roughage', providing the much-vaunted 'bulk' component of faeces so necessary in ensuring that the nation has a regular bowel habit. The gums and pectins, which our bacteria digest to a greater extent than cellulose, provide bulk too, but in a different fashion. They stimulate bacterial growth, and bacteria make up about 50% of the mass of faeces. Bacteria have the additional virtue of helping retain water in the faeces (being largely water themselves), and this makes for easier defecation.

Secondly, studies in Australia and Japan have shown that VFAs, especially butyric acid, provide an energy source for the cells that compose the wall of the large bowel, and a supply of VFAs appears to help maintain the health of these cells and stimulate blood flow in the region. The Japanese study showed that one of the VFAs — propionic acid — actually stimulates the nervous and muscular activity of the colon wall, improving peristalsis (the action that propels matter along the gut). This provides an attractive new explanation for why dietary fibre is so good at relieving constipation.

Thirdly, the right type of fibre can lower the level of cholesterol in the blood. (Cutting down on fat intake is, of course, the most important way of reducing body cholesterol.) Too much cholesterol, a lipid that the body both manufactures and takes in from the diet, is strongly implicated in causing a major 'disease of affluence' — namely cardiovascular disease. Excess cholesterol (in a modified form) ends up

Digestive system and other abdominal organs



The human digestive system.

coating the inside of our arteries and narrowing them, eventually cutting off the blood flow within a vital organ such as the heart or brain.

The liver uses cholesterol to make bile acids, the principal component of bile. After manufacture in the liver, bile is stored in the gall bladder, and enters the gut at the duodenum. Dr David Oakenfull, a scientist at the CSIRO Division of Food Research in Sydney, has shown that a class of fibre compounds called saponins (for the technically minded, these are plant glycosides), common in peanuts, spinach, and soybeans, will lower blood cholesterol by soaking up bile acids in the intestine. The result is that more bile has to be produced, and so more cholesterol is removed from the body. (See *Ecos* 19 and 47.)

A similar effect occurs in rats fed oat bran, which contains a viscous gum as well as some saponin. Dr Topping has shown that the excretion (in the faeces) of cholesterol-derived bile acids causes a noticeable decrease in the amount of cholesterol in the blood. Although oats are well fermented, this effect is not due to that, but is a physical property of the type of fibre involved. Other workers have now demonstrated that oats can lower blood cholesterol levels in humans.

Let them eat grass?

So the established beneficial effects of fibre still remain, even though much of it that occurs in natural foodstuffs is digested. The old idea of a sprinkling of processed bran as a sort of health supplement or natural-looking 'vitamin pill' to take with your corn flakes has lost ground. Wheat bran, being mainly cellulose and lignin (the latter is

Fibre and old folk

Many old people, particularly those living in institutions, suffer severe and chronic constipation. This is more than just unpleasant for the sufferers: constipation for long periods is associated with various serious disorders of the bowel. Furthermore, the consumption of large quantities of laxatives to remedy the problem is itself a less than healthy habit, as well as being expensive for the nursing institutions. In severe cases, even laxatives may eventually fail.

Could increased consumption of fibre be the answer? Dr Katrine Baghurst, of the CSIRO Division of Human Nutrition, together with Sisters Alison Hope and Elizabeth Down, of the Southern Cross nursing home near Adelaide, decided to put the idea to the test.

They carried out a year-long study at the nursing home. Twenty-nine old people, with an average age of 83 years, volunteered for inclusion in the study, and 18 of them completed the year.

The old folk were offered supplementary fibre in various forms (biscuits, porridge, prune juice), and encouraged to increase their fibre intake. This was measured, along with the components of their regular diet. To assess the effectiveness of the program, the nurses recorded the number of bowel movements, the faecal consistency, the consumption of laxatives, and the volunteers' body weights.

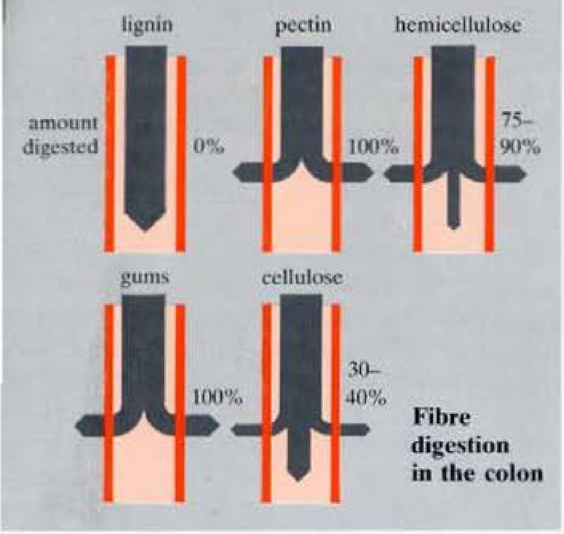
As the weeks passed the use of laxatives greatly declined, and the number of bowel movements increased. Individuals varied widely in the quantity of fibre necessary to replace laxatives.

The lowest value was 3·5 g of supplementary fibre while the highest was 24·5 g — and this highly constipated individual still required 26 weeks before being able to manage without laxatives. Over the course of the study the average fibre intake of all the participants increased from 13·9 g to 25·4 g per day. (The average Australian intake is 18–19 g.)

A further interesting, and somewhat unexpected, finding was an increase in the patients' intake of several important nutrients, such as vitamins C, D, B6, and folic acid, iron, and magnesium. Most fibre-containing foods are also nutritious and, as many old people may not always receive adequate intakes of nutrients like these, this finding represents an important plus.

Obviously the best approach to take in nursing home care is to change the whole diet, rather than give supplementary fibre-containing foodstuffs as happened in this experiment. Many institutions, however, may have to rely on outside catering companies; if so, they should persuade them to make whole-grain cereals, fruits, and vegetables the basis of their food. Even if it costs a little more, it may well be worth it in terms of both improved health care and savings on the consumption of laxatives.

Dietary intake in a group of institutionalised elderly and the effect of a fibre supplementation program on nutrient intake and weight gain. K.I. Baghurst, A.K. Hope, and E.C. Down. *Community Health Studies*, 1985, **9** (2), 99–108.



Different types of fibre are digested to varying degrees as they pass through the large intestine (or colon).

wood, and is not digested even by gut bacteria), remains good for providing bulk and speeding up the rate of passage through the gut (transit time), but is perhaps not as beneficial as the many other plant foodstuffs that can undergo full fermentation and may also lower our levels of cholesterol. Nutritionists advocate a change of diet to include more fresh fruit and vegetables, pulses, and grains, rather than simply taking fibre in an artificial form, just as, years ago, children held their noses and swallowed their cod-liver oil.

But the attentive reader may have one final question: if our intestinal bugs can digest cellulose, why can't we live off grass, like our fermenting friends the cows and sheep?

A possible answer is provided by the work of an American researcher, Dr Peter van Soest, who fed groups of human volunteers a range of different fibre supplements, one group receiving pure cellulose. (The volunteers ate a nutritionally adequate diet with the supplements.) At the beginning of the study the subjects could ferment at least 50% of the cellulose, but gradually the degree of digestion declined, until after 6 weeks of receiving the cellulose supplement they could ferment only a small fraction.

Analysis of faeces revealed that the cellulose-digesting bacteria were lost from the bowel after about 40 days. In one individual, tests did not recover this strain of bacteria again for 2 years. Why did cellulose cause the demise of the very bacteria that digested it?

Nobody knows for sure, but one likely explanation is as follows: the cellulose, by increasing faecal bulk, reduces the transit time; as a result bacteria are excreted from the bowel in large quantities and at a rapid rate, causing a gradual decline in their population. In the ruminant animals the design of the gut — with four stomachs — allows a much longer time for fermentation and so a continuously multiplying culture of cellulose-digesting bacteria can remain.

Dr Topping and his colleagues are hoping to do more research on the digestion of fibre in humans, but for the moment the message from their work is that fibre is not just a supplement to oil the wheels of digestion, but a complex of many different substances that, when taken in a balanced form, have more health benefits than we first thought.

Roger Beckmann

Fibre and the sugar in blood

Fibre has acquired a good reputation in some unexpected areas of human physiology — for example, in helping to deal with the problem of controlling the level of glucose in the blood, the central issue in diabetes.

The correct name for this well-known condition is diabetes mellitus, and mellitus means sweet. That name derives from the most obvious symptom of the untreated disease — the fact that sugar, specifically glucose, is excreted in the urine.

Diabetes is an extremely complex condition, but the central problem — a failure to control glucose levels in the blood — is simple enough. This may come about because the pancreas fails to produce adequate quantities of the hormone insulin, which, travelling in the blood-stream, allows cells to take up glucose (which they use as a fuel), and also encourages the liver to store some glucose in the form of glycogen. With insufficient insulin, levels of glucose in the blood rise so high that the sugar appears in the urine, and starts to affect brain function.

In another form of diabetes — one that often develops in middle-aged, overweight people — the pancreas may even produce too much insulin, but it fails to have the necessary effect, and levels of glucose in the blood still rise.

In normal individuals the concentration of glucose in the blood varies, being highest shortly after a meal, and lowest after fasting, for instance before breakfast. (A meal doesn't have to contain sugar to increase glucose levels; starch, widely found in so many foodstuffs, is a polymer of glucose and is broken down into its glucose sub-units during digestion and absorbed into the blood-stream in that form.) But the variations in glucose levels in normal individuals are well controlled by insulin, the amount of this produced being dependent on the concentration of glucose.

Data from epidemiology (the study of disease patterns in whole populations) have suggested a link between an increase in diabetes known to be occurring in Western societies and the reduced consumption of fibre-rich foods. Several scientists have reported that high-fibre diets can often help in keeping glucose levels more stable in both forms of diabetes. (Of course, this does not apply to all cases of the disease, and it is worth bearing in mind that plenty of other factors have been linked to diabetes, ranging from genetic predisposition to certain viral infections.)

It seems that the amount of fibre with the starch, and the physical form in which the starch is taken, will affect its exposure to intestinal enzymes, speeding up or slowing down its digestion into glucose. In low-fibre diets the starch is more rapidly broken down, and a large quantity of glucose enters the blood-stream quickly. It's possible that over long periods these frequent large glucose loads can desensitise the correct insulin-controlled response. If the starch is surrounded by fibre, particularly viscous gums, the glucose release takes place more slowly, and the body does not have to mount such a strong response, which could involve producing more insulin than would be possible in an incipient diabetic.

More about the topic

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