Preventive potential of antioxidative vitamins and carotinoids against cancer

In a prospective study performed in Basel in 1971 with 2974 male participants, cancer mortality and vitamin status were found to be correlated [16]. This as yet uncompleted study demonstrates a significant inverse correlation between plasma β-carotin and all types of cancer, as well as cancer of the lung and of the stomach. Retinol was related only to gastric cancer, whereas low vitamin C and low vitamin E were related to all types of cancer, including gastric cancer. These results point to the fact that the antioxidative vitamins may be more important than has been thought so far. At present, several intervention studies throughout the world are going on to investigate this point. A possible implication of these studies that must be considered is that the RDAs of the antioxidative vitamins may have to be increased.

Final remark

In the present paper some of the 'hot topics' in vitamin application have not been reviewed, in particular the controversial evidence on the application of megadosages of vitamin C and E. Further research has to be carried out, especially to distinguish between the physiological and pharmacological effects of vitamins.

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The physiological and nutritional importance of dietary fibre

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Summary. Fibrous material is an integral part of the daily diet, and it exerts direct physiological effects throughout the gastrointestinal tract, in addition to affecting metabolic activities more indirectly.
The interplay of these effects is responsible for the presumed desirable influence of fibre on weight regulation, carbohydrate and lipid metabolism, and on colon function. Numerous mechanisms of action have been identified which are related to the type and the physicochemical nature of the fibre. This review concentrates mainly on the serum cholesterol-lowering effect of dietary fibre, its colonic fermentation, and finally on some possible adverse effects that one should be aware of when consuming high amounts of dietary fibre.
Key words. Dietary fibre; nutrition; review; health; cholesterol; minerals; fermentation; gastrointestinal tract; cancer.

Introduction and background

More than twenty years ago, Cleave and Campbell suggested that the steadily increasing proportion of refined carbohydrates in western diets might be etiologically related to a range of diseases of civilization [7]. The concept attracted the interest of Burkitt and Trowell who, during long years of medical service in Africa, had noticed very low incidence of colonic and cardiovascular diseases, and of diabetes. They postulated a role for dietary fibre in the prevention of these and other diseases and formulated the so-called fibre hypothesis [6].

This hypothesis has stimulated much experimental and epidemiological research. Initially, this research was hampered by the lack of a definition of dietary fibre, and of suitable methods for its measurement, because it is a mixture of many constituents, naturally present in a complex botanical structure. Dietary fibres from different sources exhibit different physical and chemical properties. In addition, these are modified when dietary fibre is isolated from its original environment, and also during the passage through the intestine. Accordingly, different fibres have different effects in the gastrointestinal tract, with various metabolic consequences.

It is therefore quite understandable that many aspects of the fibre hypothesis remain controversial, but the dietary fibre concept has gained enough credibility for health authorities to recommend an increase in fibre intake to a level substantially above the present consumption [10, 28].

More recently, much interest has been focused on the therapeutic potential of dietary fibre, especially for the management of conditions like constipation, hyperlipidemias, diabetes, obesity and diverticular disease. Concentrated fibre preparations are now being marketed with both preventive and therapeutic health claims, in a borderline area between food and drugs.

Against this background, the present review deals with a few selected unsolved scientific problems involving the concept and definition of dietary fibres, their physiological effects, and the biological mechanisms involved.

Definition and measurement of dietary fibre

Trowell used the term 'dietary fibre' as an umbrella-description for a physiological concept, and defined it as 'the skeletal remains of plant cells that are resistant to hydrolysis by the enzymes of man'[38]. Subsequently, it became evident that this broad description needed a complementary operational definition, especially in the context of dietary fibre analysis, and the proposition to define dietary fibre as the sum of lignin and the polysaccharides that are not hydrolyzed by the endogenous secretions of the human digestive tract [39] has become widely accepted. A more recent proposition [14], namely to define dietary fibre simply as 'Non-starch polysaccharides' appears to be less acceptable and has definite drawbacks [4, 31].

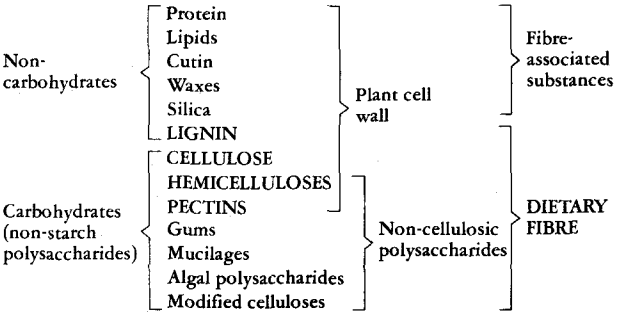

Figure 1. The terminology of the dietary fibre complex (reprinted from Schweizer and Würsch [29] with permission).

Figure 1 summarizes the terminology of the dietary fibre complex with its botanical and chemical aspects, and its main components. The chemistry and biochemistry of these plant-cell wall and dietary fibre constituents is well known [3, 32]. However, the more subtle aspects of cell wall architecture, and their physiological consequences in the gastrointestinal tract, remain to be discovered [36].

The measurement of dietary fibre has been considerably improved in recent years, and the advantages and limitations of the various analytical methods are quite well known [30]. The major problem, which has led to continuous and lively analytical discussions, is that dietary fibre cannot be precisely delimitated, be it physiologically or chemically, so as to fit precisely into an analytical concept. Conversely, no analytical method can entirely cope with the various aspects of the fibre concept.

These delimitation problems can be well illustrated with a few borderline components, and they are of more than theoretical importance [5, 31]. For example, resistant starch, i.e. starch which is not digested in the small intestine, acts as dietary fibre but is not a non-starch polysaccharide. Polydextrose, which is an undigestible non-starch polysaccharide, is not measured as dietary fibre by present methods, because it is too soluble in aqueous ethanol.

Physiological effects

In principle, dietary fibres can exert physiological effects throughout the gastrointestinal tract. Many of these direct effects are interrelated and are also the origin of more indirect metabolic consequences, as outlined in figure 2. In the upper intestine the effects of dietary fibres are predominantly due to their physical action, whereas in the lower intestine the fact that fibres are fermented by colonic bacteria is also important. The interplay of these effects is responsible for the presumed desirable influence of fibre on weight regulation, carbohydrate and lipid metabolism and on colon function, but also for potential negative effects on the digestion, absorption and utilization of essential nutrients.

Because the physiological effects of dietary fibre have been recently dealt with at length in a number of

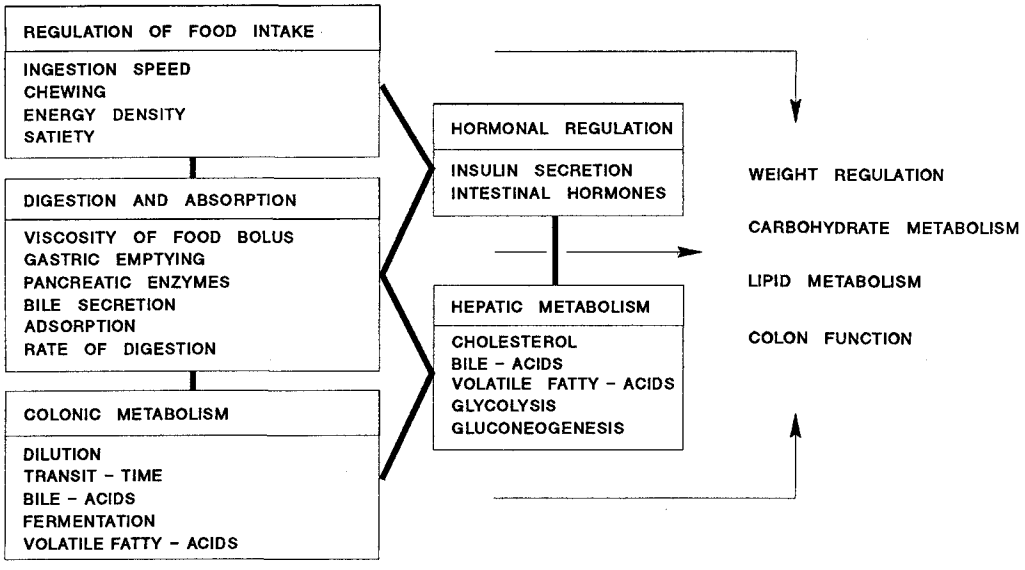

Figure 2. Physiological effects of dietary fibre.

books[1,12,35,40], only three particular aspects will be developed here.

Serum cholesterol-lowering effects of dietary fibre

There is no doubt that dietary fibre is one of the food components that influence blood cholesterol levels in man. The evidence for this stems from three different types of studies: epidemiological studies involving populations with particular life-styles, experimental studies with high-fibre diets, and studies in which diets are supplemented with specific fibre concentrates or fibrous foods.

Studies related to life-style and diet have shown that vegetarians ingesting high fibre diets have lower plasma total cholesterol and LDL cholesterol and higher HDL cholesterol than matched controls[20]. Men on high fibre diets were less at risk for atherosclerosis and other degenerative diseases after 10 years of observation than those ingesting less fibre[21].

Experimental high-fibre diets have been shown to lower cholesterol in healthy volunteers, in diabetics and in hyperlipidemics[23]. However, these diets were usually also restricted in total and saturated fat as well as in their cholesterol content. In all these cases, a major beneficial effect of dietary fibre is likely to be an indirect one, namely through substitution of high-fibre food items for those which favour high blood lipids[25]. It is therefore interesting to consider also fibre supplements or single fibrous foods added to controlled basal diets. These fibre sources can conveniently be divided into three categories: particulate fibres, viscous fibres and intermediate type fibres.

The particulate fibres are characterizied by their mostly insoluble nature. They are usually cereal brans or seed hulls such as wheat bran, corn bran and soy hulls. Pure cellulose also belongs to this category. In general, particulate fibres have no cholesterol-lowering effect, whereas many viscous polysaccharides, traditionally known as food-thickening agents, have been demonstrated to lower cholesterol, albeit in much higher doses than usually consumed. Pectin consistently lowers cholesterol by 5–19% in both normal and hypercholesterolemic individuals at doses ranging from 10 to 50 g per day[18]. Most of the decrease has been attributed to LDL cholesterol. No clear dose-response relationship emerges from these studies, but 10 g of pectin per day produced a significant effect, whereas 6 g per day was still ineffective in both normal and hypercholesterolemic volunteers. Similarly to pectin, guar gum has a definite and consistent effect on plasma cholesterol with reductions of total cholesterol between 6 and 32% after doses of 11–20 g per day in hyperlipidemics[17]. In most studies the above effects were reached in addition to those of the patients' usual lipid-lowering diet, and in some cases in addition to medication by cholestyramine or clofibrate.

The long-term ingestion of guar gum has been shown to be without adverse nutritional effects, but in one long-term study with 30 g guar gum per day, no hypocholesterolemic effect was found either[24]. Psyllium gum and locust bean gum are other polysaccharide hydrocolloids with a hypocholesterolemic potential.

Among the intermediate-type fibres which have physical and chemical properties in between those of particulate and viscous fibres, oat bran received enormous attention after quite spectacular cholesterol-lowering effects were described in both healthy and hypercholesterolemic men[2]. These effects can probably be attributed to a soluble and viscous β-glucan contained at a level of 6–10% in oat bran. However, the large amounts of oat bran necessary to achieve effects (40–100 g per day) make it

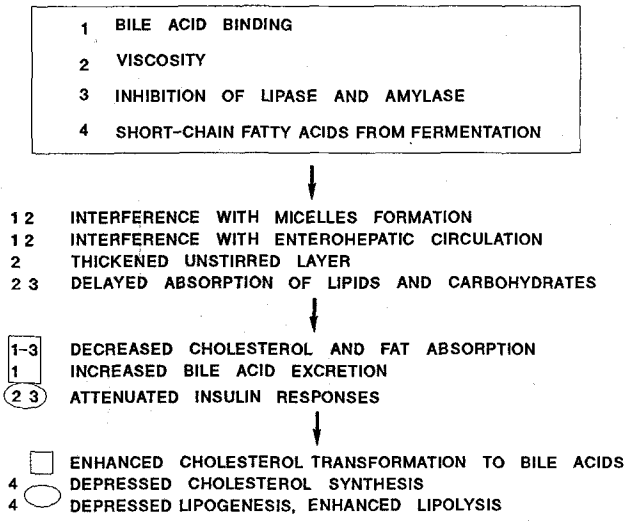

Figure 3. Possible mechanisms for fibre-mediated cholesterol-lowering effects.

likely that indirect displacement or substitution effects play also an important role[37]. Therefore, health claims based on such findings remain debatable.

The mechanisms by which certain types of dietary fibre lower blood cholesterol and particularly the low-density lipoprotein (LDL) fraction are not yet well understood. Figure 3 is an attempt to represent the main suggestions for the mechanism, which have found variable amounts of experimental support[18,40].

Binding of bile-acids by dietary fibre in the intestinal lumen can decrease the reabsorption of bile acids and increase their faecal excretion. This would enhance bile acid synthesis from cholesterol in the liver. Such a draining mechanism was seen initially as being mainly responsible for decreased plasma cholesterol. However, a number of dietary fibres which increase bile acid excretion do not decrease serum cholesterol and, conversely, not all fibres that lower serum cholesterol increase faecal bile acids.

Dietary fibre can interfere with the micellar solubility of lipids (again through bile-acid binding) and decrease absorption of dietary cholesterol and lipids, or possibly alter the rate and site of absorption. Decreased absorption, however, does not seem to play a major role since the faecal excretion of neutral steroids and total faecal fat are generally not much increased.

Through hindering diffusion of sugars of the absorptive site, viscous fibres can delay the absorption of carbohydrates and attenuate postprandial insulin secretion. Since insulin stimulates cholesterol synthesis, the delayed carbohydrate absorption could be at the origin of depressed cholesterol synthesis. Finally, the short-chain fatty acids produced by colonic fermentation of fibre could partially inhibit cholesterol synthesis in the liver and perhaps in peripheral tissues. However, the inhibition of cholesterol synthesis has only been demonstrated in isolated rat hepatocytes.

In conclusion, it is clear that the relative importance of all these mechanisms, and of further factors, in the cholesterol-lowering effect of certain dietary fibres, remains to be established.

Colonic fermentation of unabsorbed carbohydrates and its implications

Fibre polysaccharides are not digested and absorbed in the small intestine and therefore pass into the colon, where they are partly metabolized by the bacterial microflora, mainly to short-chain or volatile fatty acids (VFA), hydrogen, methane and carbon dioxide[8,15]. The VFA are absorbed and metabolized, and thus dietary fibre provides some energy for the human body. Although this energy contribution can be considered as negligible within the total daily diet, it is important in the case of individual fibre-rich foods for which calorie reduction claims are made.

The fermentability of dietary fibres is very dependent on the fibre source. Both the extent of fermentation and the pattern of the fermentation products formed vary with particle size, degree of cell wall lignification, polysaccharide structure and solubility. In addition, the course of fermentation depends on the microflora composition which varies from one individual to another[15].

It is therefore not surprising that the metabolic consequences of colonic fermentation are still poorly understood, although for many years they have been a central element of the hypothesis that fibre could prevent colon cancer (fig. 4). Much effort has been devoted recently to studying the effects of VFA on mucosal cell differentiation and proliferation[22,26], and butyrate could indeed be a potential anticarcinogen[11]. Whether VFA effects observed in vitro in cell cultures are applicable to normal cells in vivo is presently unknown, but the importance of fibre fermentation in the regulation of colonic mucosal turnover is quite apparent.

A major obstacle to further progress is the difficulty of knowing the rate and site of fermentation and, therefore, the composition and fluxes of the fermentation products. For example, the production of hydrogen seems to occur predominantly in the caecum and right colon, at a relatively low pH, whereas methane production occurs at more distal sites and a higher pH[15]. Thus, the nature of the substrate and the individual's transit time can influence methane production, which in turn determines in part the VFA production.

Negative effects of dietary fibre

The most spectacular negative effects of dietary fibre consumption have been a few isolated cases of intestinal obstruction after extreme intakes of wheat bran and gums without sufficient liquid. Apart from such anecdotes, a brief examination of adverse effects of dietary fibre on the digestion, absorption and utilization of essential nutrients is indicated.

High dietary fibre intake is often associated with increased faecal nitrogen loss[33,34]. Indigestible cell wall

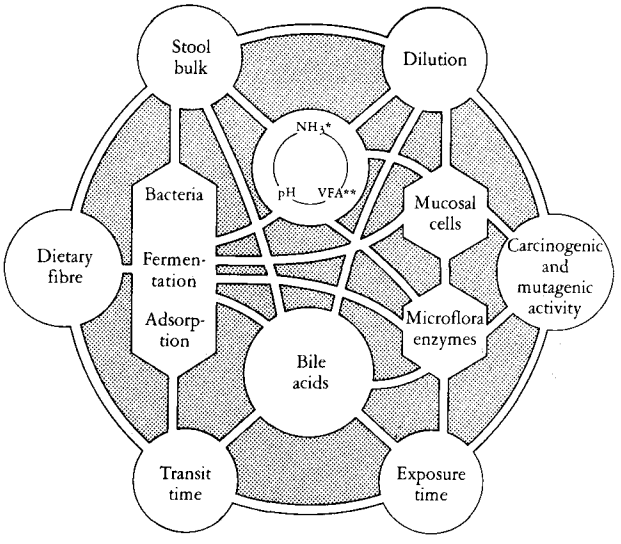

Figure 4. Possible roles of dietary fibre in colon cancer prevention (reprinted from Schweizer and Würsch [29] with permission).

protein partially explains the reduction in apparent protein digestibility, but increased faecal nitrogen loss can also be due to enhanced excretion of unabsorbed intestinal secretions, sloughing of mucosal cells, microbial protein, and possibly the reduced digestion and absorption of dietary protein. Some fibres can inhibit pancreatic enzymes in vitro [13] and, in ileostomy patients, in whom colonic events are virtually excluded, pectin increased nitrogen excretion whereas wheat bran had no effect [27]. However, the increases observed in faecal nitrogen losses are typically below 700 mg nitrogen per day for fibre intakes of up to 35 g in humans. Therefore, where protein intake is adequate there seems to be little reason for concern.

In many ways, the situation with lipids is analogous to the one with proteins. Increased intakes of certain dietary fibres, be it as fibre-rich foods [34] or as pure fibre polysaccharides [33], can lead to an increase in the lipid content of the faeces. As with protein, the precise origin of the faecal fat is unknown, but there is evidence that dietary fibre can interfere with the digestion and absorption of fat. The increase in faecal fat may reach 3 g in healthy volunteers [34] but can be somewhat higher in ileostomy patients consuming 15 g of pectin per day with a high-fat diet [27].

Numerous experiments in vitro, in experimental animals and in humans have shown that certain types of dietary fibre can bind minerals and affect their absorption [16]. In addition, whole grain cereals and leguminous seeds contain substantial amounts of phytic acid which is known to form insoluble complexes with divalent cations. Phytic acid, fibre or both have accordingly been seen as being responsible for negative mineral balances resulting from high-fibre diets or diets enriched with fibre concentrates [9, 16]. The results of such studies have, however, been highly variable and partially conflicting due to a number of inherent limitations. Thus, in vitro binding studies are unable to simulate the complex cation exchanges occurring in vivo during digestion and absorption. Purified fibre fractions may not show the same activity as the intact food, and pure sodium phytate often has more pronounced effects than endogenous food phytate. Also, a low percentage absorption of minerals contained in fibre sources can be offset by a higher supply resulting from the higher absolute amounts provided by the fibres. Finally, short-term metabolic balance experiments could overestimate fibre effects, since adaptation has been observed in longer-term studies [16]. Adaptation may also explain why vegetarians with fibre intakes of typically 30–40 g per day have a similar trace metal status to non-vegetarians consuming only 15–25 g fiber per day, although the latter may respond with initially negative balances when switched to vegetarian diets [19]. It is possible that colonic absorption of minerals can compensate for reduced small-intestinal absorption.

It is nevertheless likely that dietary fibre and especially phytate have contributed to deficiency diseases in a few particular populations consuming monotonous high-fibre diets which are at the same time marginal in specific micronutrients. Consequently, some care seems to be indicated when advising high fibre intakes for population segments known to be at risk for mineral deficiencies, such as the elderly or children.

Nutritional importance

Even if there are still many open questions, unsolved and controversial, there is agreement that a moderate increase in dietary fibre intake from the present average of 15–20 g to about 30 g per day would be beneficial for the general population in our highly industrialized countries. Provided that the higher fibre intake is achieved through selection of fibre-rich foods rather than through fibre supplements, these foods will displace e.g. energy-dense and high-fat foods. Direct fibre effects and indirect displacement effects are likely to act in a complementary way. Furthermore, the message 'more' (fibre) seems to be more attractive for nutrition education programmes than the usual 'less' (fat, cholesterol, salt etc.).

Because of the special nature of dietary fibre there is little risk of overconsumption, even with concentrated fibre sources such as wheat bran, provided that an adequate liquid intake is ensured. Indeed, fibre-rich diets are quite bulky and satiating; both the lack of palatability, and the fact that large amounts of fibre tend to cause abdominal discomfort and flatulence, usually prevent exaggeration.

The above considerations are for the adult population and should not be extrapolated to small children or even infants, who have different nutritional needs and for whom the desirable fibre intake is not known at present. Some reservations may also be indicated concerning a number of unconventional, so-called 'novel' fibre sources. These often stem from unusual botanical

sources and/or are physically or chemically modified. Accordingly, they should be carefully evaluated before approval and use.

Conclusions

Although many aspects of dietary fibres, their physiological effects and their nutritional importance remain controversial, the dietary fibre concept has found its place in nutrition. The initial enthusiastic reports have stimulated a lot of research work. As a consequence, untoward generalizations and exaggerated health claims will gradually disappear and be replaced by sound knowledge put into the right nutritional context. Future research should aim to going beyond the static stage and investigate more thoroughly the dynamics of gastrointestinal events and how various fibres influence them.

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