Sugars in Nutrition, edited by M. Gracey,
N. Kretchmer, and E. Rossi,
Nestlé Nutrition Workshop Series, Vol. 25.
Nestec Ltd., Vevey/Raven Press, Ltd., New York © 1991.

Dietary Fiber and Unabsorbed Carbohydrates

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Carbohydrate is the main source of energy in the diet, provided essentially by starch, sucrose, starch derivatives, and lactose. Food, however, also contains other natural or added carbohydrates that are not digested by the small intestine or escape absorption and thus reach the large intestine to be metabolized in part or completely by the microflora. These carbohydrates (Table 1) and, in particular, dietary fiber, have important physiological and metabolic effects at all levels of the intestinal tract. These effects were somewhat underestimated until renewed interest in dietary fiber began some 20 years ago (1), and, more recently, interest in the sugar alcohols increased because of their reduced digestibility and noncariogenic properties.

In dealing with this topic, I concentrate on the metabolism of the nonabsorbed carbohydrates in the intestine and the consequent effects on the host through alteration of the pH, the microflora, and generation of short-chain fatty acids.

DIETARY FIBER

Trowell originally defined dietary fiber as "the remnants of the plant cell walls that are not hydrolysed by the alimentary enzymes of man" (2). This was modified subsequently to include all "plant polysaccharides and lignin which are resistant to hydrolysis by the digestive enzymes of man" (3). This physiological definition is now most widely accepted, although some still prefer a more botanical definition or even a purely chemical one.

The plant cell wall is predominantly a mixture of polysaccharides organized in a complex supramolecular arrangement with minor amounts of protein and minerals. Lignin is deposited in mature plants to serve as the linkage between the supramolecular structures.

The physiological properties of dietary fibers are intimately linked to their physical and chemical properties, which are dependent on their composition and structure. These properties vary greatly from one source of fiber to another and can be altered when the dietary fiber is isolated by fractional separation from the plant matrix or

TABLE 1. *Dietary carbohydrates that can be partially or totally nondigested and unabsorbed.*

Simple sugars	Complex carbohydrates
Lactose	Dietary fiber
(fructose)	Starch
Sugar alcohols	Mucopolysaccharides
Raffinose, stachyose	Glycoproteins

by food processing, such as milling, cooking, or freezing. Table 2 shows some typical chemical differences between dietary fibers from two important groups of foods.

Considerable effort has been spent in measuring the physical properties of dietary fiber, such as water-holding capacity, ion exchange, and bile acid adsorption capacities. Such *in vitro* measurements have, however, not been of much predictive value for the biological activity of dietary fiber along the gastrointestinal tract because the physical properties of fibers are profoundly altered during their intestinal passage, especially through colonic fermentation (5). Nevertheless, their physical properties are said to be beneficial against constipation, diverticular disease, and colonic cancer, as well as to modify starch digestion, glucose absorption, and hyperlipidemia. These roles have been discussed extensively by others (6,7), and this review is confined to the metabolism of dietary fiber and the consequences to the host. The fermentation of fiber by the large intestinal microflora is not limited to its soluble fraction, as it is sometimes believed. In fact, some soluble fibers, such as psyllium gum, carboxymethylcellulose (CMC), and xanthan gum, are only partially fermented. On the other hand, fibers in peas and carrots are both fermented by 50%, although pea fiber is only 17% soluble and carrot fiber is 49% soluble. The extent of fermentation undergone by a particular fiber source depends on a number of factors, among which the degree of lignification of the cell wall polysaccharides may

TABLE 2. *Source of fibers and properties*

	Cereals	Vegetables/fruits
Ratio insoluble/soluble fiber	>10:1	5:1/1:1
Cellulose in insoluble fibers	<50%	>50%
Lignification	Strong	Weak
Major hemicelluloses	Pentosans	Hexosans
Pectins in soluble fibers	<5%	30–70%
Degree of esterification (%)	—	40–65/60–95
Water-holding capacity	Low	High
Fermentability	Low	High

From Schweizer TF, Würsch P. (4).

TABLE 3. *Resistant starch content in various foods*

	% Dry matter	Based on starch (%)
Cereals	0.4–2.0	0.5–2.6
Bread	0.9–1.7	1.2–2.4
Pasta	1.3–1.7	1.6–2.4
Rice	0.5–1.7	0.7–2.4
Potato	1.0–3.5	1.1–4.0
Legumes	3.5–5.7	7.0–10.0

Adapted from Ring SG, *et al.* (8), and Würsch P (16).

help explain why the more lignified cereal brans are fermented to a lesser extent than are vegetable or fruit cell walls.

STARCH

Starch mostly is eaten cooked. During the heating process, starch granules gelatinize, thus increasing their susceptibility to α-amylase digestion. A fraction of the starch, however, is found to be undigestible after prolonged incubation of the cooked starch with the enzyme. This fraction has been called resistant starch (RS) (Table 3). Paradoxically, the digestibility of starch seems to decrease after heat processing and storage. This occurs from a reassociation of amylose and amylopectin chains, followed by a slow crystallization of the macromolecules (8,9). Retrogradation of amylose is rapid, and the resulting material is highly resistant to amylolysis. In contrast, amylopectin undergoes a slow retrogradation—staling in bread—and is degraded completely by amylase after 24 hours incubation (8). Generation of RS requires a profound disruption of the native granule structure and favorable conditions of temperature and starch/water ratio, which facilitate molecular reassociation. Freshly cooked potato has been reported to contain less than 2% RS, but it increases rapidly to about 3% on cooling. Reheating produces a temporary decrease, but cooling the potato again increases further the level of resistant starch (10). RS has been shown to resist digestion *in vivo* in rats (11) as well as in humans (10,12), but it is more or less readily metabolized by the intestinal microflora. RS is structured in a homogeneous crystalline matrix, but it seems to differ in size and density and, hence, in susceptibility to bacterial degradation depending on the type of starch. It has been observed that RS from pea starch was fermented *in vitro* by human organisms almost half as fast as RS from maize starch, and its degradation was even incomplete after prolonged incubation (13). It has to be shown, however, if this lower degradation of RS from pea starch is a characteristic limited to the leguminous starches and high amylose starches, or if it is broadly distributed among the starchy foods. Furthermore, the process that produces RS might also affect the susceptibility of RS to bacterial degradation. Studies on the digestibility of starch involving ileostomized

patients have shown that some starches other than RS may escape digestion and absorption in the small intestine. This level can vary from 0.5% starch in rice (14) to 10% for beans (15). Therefore, from data available so far, one can estimate that RS and unabsorbed starch represent 2–5% of the total starch consumed in the average Western diet, that is, less than 10 g carbohydrate/day (16).

SUGARS

In normal infants, lactose, which is the main dietary sugar in milk, is hydrolyzed in the small intestine by lactase (β-galactosidase), and the resulting glucose and galactose are absorbed. Intestinal lactase activity in normal humans may be low at birth, but there is a rapid increase thereafter. This is followed by a loss of lactase activity in a large number of populations, with the exception of North and Central Europeans, some Mediterranean groups, and those elsewhere primarily of European descent (17). Lactose maldigestion can be detected by measuring the concentration or rate of excretion of breath hydrogen or the blood glucose response after lactose is consumed (18,19). The majority of lactose maldigesters can consume the lactose equivalent of 240 ml of milk (12 g lactose) without symptoms, though not all the ingested lactose may be digested (17). There is also evidence that such subjects can tolerate greater amounts of lactose, especially if ingested in divided doses, without developing symptoms.

The α-galactosides (raffinose, stachyose, and verbascose) are found mostly in leguminous seeds at concentrations of 2.5–3.5%. The small intestine does not possess the α-galactosidase that is able to digest these oligosaccharides, and they, therefore, are completely unabsorbed. Due to the generally low level of consumption of these foods, the daily intake of these sugars should generally be far below 5 g.

Sugar alcohols are categorized into the bulk sweeteners whose purpose is to replace sucrose in foods. Their functional properties should be as close as possible to those of sucrose by providing bulk and a clean sweetness. They, however, differ from sucrose or the other common dietary sugars with regard to several physiological properties: (a) not metabolized by the bacteria of the dental plaque, (b) low affinity to the disaccharidases of the small intestine (disaccharide alcohols), (c) passive absorption (pentitol, hexitols), and (d) no induction of insulin secretion.

The slow digestion and absorption rates of sugar alcohols often result in their partial or total malabsorption. The unabsorbed fraction, in turn, is metabolized by the bacteria of the large intestine. The degree of malabsorption is still a subject of controversy because of the difficulty in quantitating the malabsorption and because of possible variability in the degree of malabsorption caused by the food form (20). Recently, malabsorption of several sugar alcohols has been compared by using the breath hydrogen test (21). Quantification of the exhaled hydrogen produced by metabolism of the carbohydrates by the colonic flora can be calculated from the area under the incremental breath hydrogen response curves (Fig. 1). Several studies have

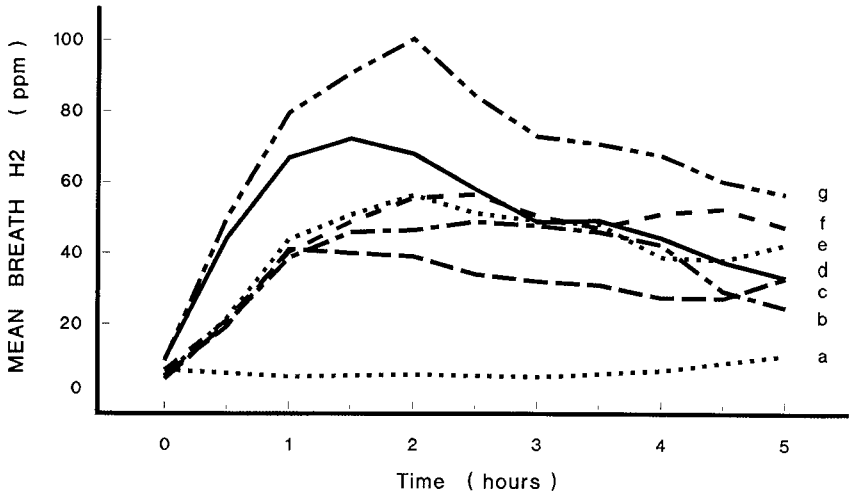

FIG. 1. Mean profiles of breath hydrogen excretion following ingestion of 10 g hexitols or 19 g disaccharides (mean of 7 subjects). Zero hour = initial increase; a, basal; b, sorbitol; c, mannitol; d, lactulose; e, palatinit; f, maltitol; g, lactitol. From Würsch P, et al. (21).

shown that there is a linear relationship between the amount of hydrogen produced and the carbohydrate fermented. It can hence be deduced that sorbitol, mannitol, maltitol, and palatinit are only partially absorbed and that the remainder reaches the large intestine and is completely fermented. The amount of polyol usually ingested is nevertheless very small, apart from the consumers of confectioneries made with these carbohydrates. This amount can in some extreme cases reach 30 g/day, with the consequent risk of intestinal disorders.

METABOLISM OF CARBOHYDRATE IN THE LARGE INTESTINE

The carbohydrates that are not digested and absorbed in the small intestine pass into the large intestine and are subsequently metabolized by the bacterial flora. Microbial counts range between 10^{11} and 10^{12} organisms/g of stool, with well over 95% anaerobes (22). The microflora produce a wide range of glycosidases that are able to hydrolyze virtually all the glycosidic linkages of the carbohydrates. Cellulose and starch are broken down to glucose, which is taken up by the microorganisms of the large intestine and metabolized to pyruvate and subsequently to short-chain fatty acids (SCFA), hydrogen, methane, and carbon dioxide (Fig. 2). Hemicellulose and other complex polysaccharides are hydrolyzed to the constituent pentoses and hexoses, which are further metabolized (23).

Because the breakdown of carbohydrate occurs in anaerobic conditions, the microflora is able to use only a fraction of the potential energy available for maintenance

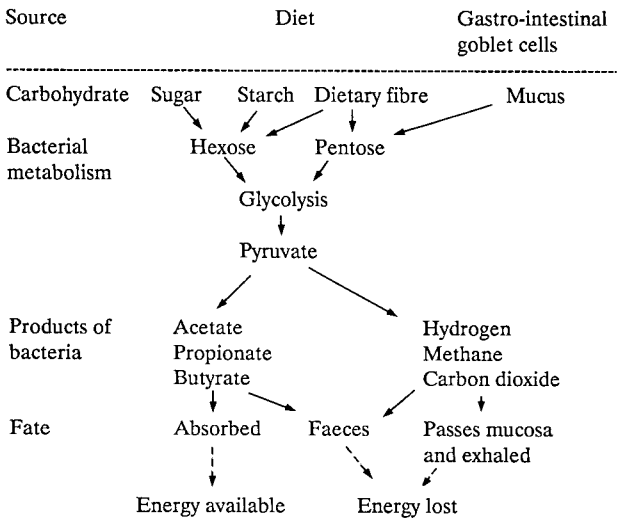

FIG. 2. Carbohydrate metabolism by the bacteria of the large intestine. From McNeil NI (23).

and growth. Various equations of the metabolism of hexoses have been proposed similar to that from Miller and Wolin (24).

$$34.5 \text{ mol hexose} \rightarrow 48 \text{ mol acetate} + 11 \text{ mol propionate} + 5 \text{ mol butyrate} + 23.7 \text{ mol } CH_4 + 34.25 \text{ mol } CO_2$$

One mole of hexose will yield 4 to 5 moles of ATP and a large quantity of SCFA, which are mostly absorbed in the large intestine and used by the host (25,26).

However, studies of the fermentation of monosaccharides and polysaccharides *in vitro* with fecal incubation systems show that the pattern of SCFA varies with the type of substrate, increased butyrate occurring from starch or sorbitol (27). Increasing malabsorption of starch by providing an α-glucosidase inhibitor to volunteers produced an increase of the stool weight and doubled the daily excretion of SCFA (28). The increase was particularly high for butyrate (+182%).

SHORT-CHAIN FATTY ACIDS

The concentration of SCFA in the large intestine of various mammals, including humans, was found to be in the range of 60 to 170 mmol/kg (29,30), which corresponds to the concentration found in the feces. Because the dry matter of feces is 2 to 3 times higher, this implies that part of the SCFA is absorbed by the colon. Unfortunately, we have no information yet on the changes in SCFA concentration in the colon when a low-fiber diet is replaced by a high-fiber diet. SCFA production occurs even when no dietary carbohydrate or fiber is ingested, as intestinal mucus and other endogenous materials enter the large intestine to be fermented (31).

TABLE 4. *Concentration of short-chain fatty acids in the human intestine and blood*

	Acetate	Propionate	Butyrate
Intestine (mmol/kg)			
Cecum	69	25	2
Transverse colon	58	23	3
Rectum	50	20	2
Blood (μmol/liter)			
Portal	258	88	29
Hepatic	115	21	12
Peripheral	70	5	4

From Cummings JH, *et al.* (29).

The absorption and metabolism of SCFA have been reviewed extensively by Bugaut (32). They are metabolized rapidly in the digestive epithelium, and their complete oxidation to CO_2 is often a preponderant catabolic pathway. Bergman and Wolff (33) showed that approximately 30%, 50%, and 90% of acetate, propionate, and butyrate, respectively, do not reach the portal blood of ruminants, but in the case of humans, no precise quantitative data exist. These SCFA, in particular acetate produced by cellulose, pectin, or lactulose fermentation, have been detected in the peripheral blood (29,34). Cummings et al. (29) measured the concentration of these SCFA in different parts of the large intestine and in portal, hepatic, and peripheral venous blood (Table 4). The molar ratios of the three principal SCFA changed on going from the colonic contents to portal blood to hepatic vein, indicating greater uptake of butyrate by the colonic epithelium and propionate by the liver. The ratio of acetate/butyrate was 10 in the portal blood and only 2 to 3 in the colon, as was also found in *in vitro* fecal fermentations (24,27). The concentration of the SCFA is very low in the small intestine, under 0.4 mmol/kg in the jejunum, and rises to 3.7 to 7.7 mmol/kg in the ileum (23).

The amount of energy yielded by these processes has been studied on a few occasions in humans, but the values are quite controversial (25,35). This assessment is important, however, especially in the case of the dietary fibers, which at present are supposed to provide no energy, and some sugar alcohols, which in various countries are labeled "low calorie."

In animals, the fermentation process can provide an important part of their energy requirement. In ruminants, it reaches 70–80% (36). Western and Japanese diets provide about 20 to 25 g dietary fiber, of which about 5 g are lost in the feces. Malabsorption of starch is estimated at 5 to 10 g, as explained previously, which is totally fermented. Therefore, 20 to 30 g of exogenous carbohydrate are estimated to be fermented in the large intestine. From the equation presented and taking into account a small loss of SCFA in the feces, one can estimate that daily 50 to 80 kcal are provided by these intestinal processes without accounting for increased cell mass, protein, and lipid losses.

ROLE OF SCFA ON MUCOSAL CELL PROLIFERATION

There are few data concerning metabolism and biological contribution of SFCA in the epithelium of the large intestine of most mammals, particularly humans. Colonic epithelial cells, like all other mammalian cells, must grow, differentiate, and be renewed if they are to remain healthy and serve their intended functions. Lupton et al. (37) showed that supplementing a basal fiber-free diet of rats with either 10% pectin or guar significantly increased the mucosal mass, cell number, and their proliferation, with the greatest differences occurring in the cecum, compared with a supplementation of 20% oat bran or cellulose. Both pectin and guar gum were completely fermented, unlike the bran. Stimulation of colonic cell division was observed with the fermentable sugars lactulose and sorbitol (38). Johnson and Gee (39) found that the consumption of guar gum and CMC led to a uniform increase in mitosis as compared to cellulose supplementation, but in the CMC-fed group, the rate was progressively higher in the more distal position. Both cellulose and CMC are minimally fermentable fibers, but the first is essentially insoluble. Enlargement of the cecum and colon was observed with CMC and guar gum, which suggests that this hypertrophy occurs also in response to bulk rather than only to SCFA formation (40). Goodlad et al. (41) confirmed that the trophic effect of dietary fiber on intestinal epithelial cell production was mediated mostly by fermentation of fiber, since they observed a lack of any proliferative response in germ-free rats.

Different dietary fibers have specific effects on villous crypt structure, with pectin causing the most consistent changes. In an examination of the role of dietary fibers in small intestinal growth, Jacobs (42) concluded that oat bran reduced the crypt cell population size by decreasing cell replication, pectin increased villous cell exfoliation, causing a faster cell migration along the villus, and guar gum gave a marked increase in mucosal growth without alteration in crypt villous morphometry, but with increased crypt cell production. Nevertheless, we do not know if SCFA produced from undigested dietary carbohydrates contribute significantly to the maintenance and growth of mucosal cells in humans. Interestingly, in this context, in humans receiving fiber-free parenteral nutrition, no morphological change in the mucosa has been observed, although a decrease in enzymatic activity in the upper duodenum occurred (43).

In animals, stimulation of mucosal development and epithelial cell division has been shown repeatedly, but the mechanism seems to be quite complex. *In vitro*, the SCFA stimulate cell proliferation below 1 mM concentration but are strong inhibitors above that concentration (44). However, *in vivo*, the stimulation occurs even at the high physiological concentration, which is in the range of 50 to 170 mM (32,45). Furthermore, the trophic effect was observed also in the jejunum when the SCFA were instilled at the lower ileum level in rats (45). The effect was dose dependent and in the order butyric > propionic > acetic acid. It is suggested that a systemic mediatory mechanism transmits the stimuli of SCFA to the epithelial cells, and this mechanism would dominate the direct inhibitory action of SCFA.

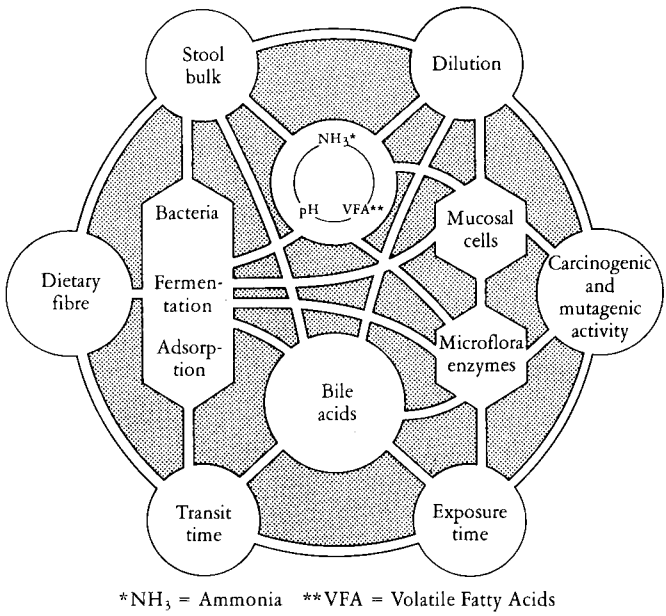

FIG. 3. Dietary fiber actions in the large bowel. From Schweizer TF, Würsch P (4).

UNDIGESTIBLE CARBOHYDRATE AND CANCER DEVELOPMENT

The undigested polysaccharides are known to be the only protective material to enter the large bowel from dietary residues, but the protective mechanisms are still not evidenced. Long-term high-fiber diet generally results in a decreased incidence of colon cancer. The effect has been attributed to the bulk effect, which can cause a decrease in transit time, reduced adsorption of chemicals by fiber, or dilution of the carcinogens present in the intestinal lumen. The data available so far are, however, mainly epidemiological and at best suggestive (4).

Dietary fibers reduced the incidence of chemically induced colon tumors in animal studies by adsorbing and diluting the possible mutagens and tumor promoters and by decreasing the transit time. Some bile acids are toxic to colonocytes, and they induce a compensatory hyperplasia, which places more dividing cells at risk to mutagenesis by fecal mutagens. For several years, it has been shown that some fiber or fiber fractions adsorb bile acids and influence their metabolism. The fiber action is essentially of a physical type (Fig. 3). However, based on animal experiments, it is established that fermentation of fibers influences the intestinal mucosa, as shown previously.

The SCFA stimulate mucosal growth and/or turnover (45,46), and butyrate induced an alteration of growth of human tumor cells (47). SCFA inhibit the growth of a number of different cell lines by blocking the cell cycle immediately following mitosis. Recent data indicate that butyrate may be toxic to lymphoid cells, but it does

not affect the viability of normal cells. The influence of butyrate on cell growth is a general property of SCFA, which are metabolized by normal mucosa as well. The SCFA would seem to be effective in stabilizing colonic mucosa, especially at high concentration, which is generally the case (Table 4). However, in order to demonstrate a positive or negative influence of SCFA on the development of colonic cancer, SCFA should induce a clear differential effect on normal versus malignant or premalignant cells. Fermentation also lowers the luminal pH, which modifies colonic microbial metabolic acidity and is associated with increased epithelial cell proliferation and colon carcinogenesis. Nevertheless, it is believed that the presence of SCFA in the colon decreases the toxicity of bile acids by decreasing their ionization potential. Thus, a decrease of deoxycholate synthesis from cholic acid and its reduced absorption has been obtained in humans with oral lactulose, which acidifies the colonic content (48).

Andrieux et al. (49) found a high correlation between minimal cecal pH *in vivo* and the bacterial transformation of cholic acid in rats fed various partially digestible carbohydrates. Measurement of the amount of fermented carbohydrate showed most interestingly that the transformation of cholic acid was less affected by the fermentation of amylomaize and potato starch than by lactose and lactulose. After carbohydrate ingestion was stopped, the transformation remained unchanged in rats fed on the starches, whereas it was primarily restored in those fed on the β-galactosides. These results indicate that apart from the reduction of pH, there is another factor that changes this transformation, and most probably the bacterial population itself was modified.

DIETARY FIBER AND CHOLESTEROL METABOLISM

It has been suggested repeatedly that the hypocholesterolemic actions of certain fiber-rich foods, such as oat bran, bean (50), or pectin, could be related to the absorption from the colon of the SCFA that are produced by extensive bacterial degradation of these fibers. Thus, propionate (51) or acetate (52) lowers cholesterol when fed in the diet of experimental animals and also alters cholesterol synthesis in isolated hepatocytes by competitive inhibition of the 3-OH-3-methylglutaryl-CoA synthetase, which is the first enzyme involved in the synthesis of the sterols, as well as the enzyme in the second step (53). Propionate does not mediate the cholesterol-lowering effects of fermentable carbohydrate, and its dietary actions seem to arise from a marginal toxicity due to its rapid absorption (54). Fiber fractions, such as wheat bran aleurone, that yield propionate as a major fermentative product actually raise plasma cholesterol in animal studies (55). Measurement of the specific effects of the fermentative products often is hidden behind other mechanisms that occur, particularly in the small intestine, as reducing cholesterol absorption, adsorbing bile salts and increasing fecal sterols losses as observed with pectin. This polysaccharide has been shown to increase hepatic lipogenesis *in vitro*, but no change was found *in vivo*. When pectin was infused directly into the cecum of rats, however, stimulation of

hepatic lipogenesis occurred, but the effect of pectin was stopped by concomitant administration of antibiotics (56).

The evolution of cholesterol has been followed during prolonged consumption by rats and humans of nondigestible carbohydrates in conditions that do not disturb the digestion process at the small intestinal level. The daily consumption of 8 g fructo-oligosaccharides (Neosugar) by type II diabetic patients resulted in a significant reduction of basal glucose level (−8%) of cholesterol, in particular LDL (−8%). No change occurred in normal individuals (57). Lactulose (18–25 g/day), given to healthy individuals for 2 weeks, produced a significant rise in fasting serum total cholesterol and LDL cholesterol as well as triglyceride and apolipoprotein B (28). The cholesterol-lowering effect of some dietary fiber might be the result of mechanisms that could be more related to their physical actions in the small intestine and that modify, among others, the bile acid excretion or the rate of glucose absorption in the small intestine. In fact, a serum cholesterol-lowering effect has been demonstrated for a number of gel-forming soluble fibers but only exceptionally with wheat bran (58). Wheat and oat brans contain less than 5% and 10% soluble polysaccharides, respectively (59), but only the latter and its viscous soluble β-glucan fraction have been shown to reduce plasma cholesterol in rats, chicks, and humans (50,60). A 20–25% long-term reduction of plasma cholesterol was obtained with a daily consumption of 60 g oat bran (50), which provided approximately 6 g soluble viscous β-glucan. A significant reduction of bile acid excretion occurred but not neutral sterols. Comparatively, more than 15 g of highly viscous pectin or guar consumed daily was necessary to produce a more than 10% decrease in serum cholesterol (6). Total plasma cholesterol also was effectively reduced by consuming 100 g dry weight of legumes, such as beans or lentils. The total fiber contribution would be around 20 g, of which 7 g are soluble. To that should be added 5 to 10 g of unabsorbed carbohydrate and resistant starch (15). It has been postulated that gastrointestinal hormones may mediate the effects of these soluble viscous polysaccharides, in particular, glucagon, which is an important endogenous regulator of plasma cholesterol (61). Furthermore, it was found that the lipid fraction of oat or barley bran has itself a hypocholesterolemic effect. This fraction contains phytosterols and tocols, in particular, α-tocotrienol, which is a potent inhibitor of cholesterol biosynthesis *in vitro* and *in vivo* in chicks at very low concentration (62). The hypocholesterolemic property of these brans may well be the result of the additive actions at the intestinal and hepatic levels of the polysaccharide, the lipid, and the protein fractions. It must be stressed, however, that these dietary modifications also very often lead to changes in energy intake, in lipid profile, or in the glycemic responses to meals.

REFERENCES

1. Burkitt DP. Related diseases—related causes? *Lancet* 1969;2:1229–31.
2. Trowell H. Ischemic heart disease and dietary fiber. *Am J Clin Nutr* 1972;25:926–32.
3. Trowell H, Southgate DAT, Wolever TMS, Leeds AR, Gassull MA, Jenkins DJA. Dietary fiber redefined. *Lancet* 1976;1:967.

4. Schweizer TF, Würsch P. Dietary fiber and the prevention of cancer. *Nestlé Res News* 1986;43–52.
5. Eastwood MA, Brydon WG, Tadesse K. Effect of fiber on colon function. In: Spiller GA, McPherson-Kay R, eds. *Medical aspects of dietary fiber*. New York: Plenum Press, 1980:1–26.
6. Spiller GA, ed. *Handbook of dietary fiber in human nutrition*. Boca Raton, FL: CRC Press, 1986.
7. Vahouny GV, Kritchevsky D. *Dietary fiber. Basic and clinical aspects*. New York: Plenum Press, 1982.
8. Ring SG, Gee JF, Whittam M, Orford P, Johnson IT. Resistant starch: its chemical form in foodstuffs and effect on digestibility in vitro. *Food Chem* 1988;28:97–109.
9. Würsch P. Structure of starch in food: interaction of starch and sugars with other food components. In: Dobbing J, ed. *Dietary starch and sugars in man: a comparison*. Berlin: Springer Verlag 1989:35–48.
10. Englyst HN, Cumming JH. Digestion of polysaccharides of potato in the small intestine of man. *Am J Clin Nutr* 1987;45:423–31.
11. Björk I, Nyman M, Pedersen B, Siljeström M, Asp N-G, Eggum BO. On the digestibility of starch in wheat bread: studies *in vitro* and *in vivo*. *J Cereal Sci* 1986;4:1–11.
12. Englyst HN, Cumming JH. Digestion of the polysaccharides of some cereal foods in the human intestine. *Am J Clin Nutr* 1985;42:778–87.
13. Wyatt G, Horn N. Fermentation of resistant food starches by human and rat intestinal bacteria. *J Sci Food Agric* 1988;48:281–8.
14. Jenkins DJA, Cuff D, Wolever TMS, et al. Digestibility of carbohydrate foods in an ileostomate: relationship to dietary fiber, *in vitro* digestibility and glycemic response. *Am J Gastroenterol* 1987; 82:98–106.
15. Schweizer TF, Andersson H, Langkilde AM, Reimann S, Torsdottir I. Nutrients excreted in ileostomy effluents after consumption of mixed diets with beans or potatoes. II. Starch, dietary fiber and sugars. *Eur J Clin Nutr* 1990;44:567–75.
16. Würsch P. Starch in human nutrition. *World Rev Nutr Diet* 1989;60:199–256.
17. Scrimshaw NS, Murray EB. The acceptability of milk and milk products in populations with high prevalence of lactose intolerance. *Am J Clin Nutr* 1988;48:1083–159.
18. Calloway DH, Murphy EL, Bauer D. Determination of lactose intolerance by breath analysis. *Am J Dig Dis* 1969;14:811–5.
19. Barr RG. Limitations of the hydrogen breath test and other techniques for predicting incomplete lactose absorption. In: Paige DM, Bayless TM, eds. *Lactose digestion: clinical and nutritional implications*. Baltimore, MD: Johns Hopkins University Press, 1981:110–4.
20. Würsch P, Anantharaman G. Aspects of the energy value assessment of the polyols. In: Grenby TH, ed. *Progress in sweeteners*. Barking, UK: Elsevier, 1989:241–66.
21. Würsch P, Koellreutter B, Schweizer TF. Hydrogen excretion after ingestion of five different sugar alcohols and lactulose. *Eur J Clin Nutr* 1989;43:819–25.
22. Hill MJ, Drasar BS. The normal colonic bacterial flora. *Gut* 1979;16:318–23.
23. McNeil NI. Nutritional implications of human and mammalian large intestinal function. *World Rev Nutr Diet* 1988;56:1–42.
24. Miller TL, Wolin JM. Fermentation by saccharolytic intestinal bacteria. *Am J Clin Nutr* 1979;32:164–72.
25. Wolin MJ. Fermentation in the rumen and human large intestine. *Science* 1981;213:1463–8.
26. Cummings JH, Branch WJ. Fermentation and the production of short-chain fatty acids in the human large intestine. In: Vahouny GV, Kritchevsky D, eds. *Dietary fiber, basic and clinical aspects*. New York: Plenum Press, 1968;131–50.
27. Mortensen PB, Holtug K, Rasmussen HS. Short-chain fatty acid production from mono- and disaccharides in a fecal incubation system: implications for colonic fermentation of dietary fiber in humans. *J Nutr* 1988;118:321–5.
28. Brighenti F, Testolin G, Jenkins DJA. Laboratory assessment and metabolic effects of increased colonic fermentation induced by unabsorbed carbohydrates. Fibre 90, Norwich, 17–20 April, 1990.
29. Cummings JH, Pomare EW, Branch WJ, et al. Short chain fatty acids in human large intestine, portal hepatic and venous blood. *Gut* 1987;28:1221–7.
30. Clémens ET, Stevens CE, Southworth M. Sites of organic acid production and pattern of digesta movement in the gastrointestinal tract of swine. *J Nutr* 1975;105:759–768.
31. Fleming SE, Rodriguez MA. Influence of dietary fibers on fecal excretion of volatile fatty acids by human adults. *J Nutr* 1983;113:1613–25.
32. Bugaut M. Occurrence, absorption and metabolism of short chain fatty acids in the digestive tract of mammals. *Comp Biochem Physiol* 1987;868:439–72.

33. Bergman EN, Wolff JE. Metabolism of volatile fatty acids by liver and portal-drained viscera in sheep. *Am J Physiol* 1972;221:586–92.
34. Pomare EW, Branch WJ, Cummings JH. Carbohydrate fermentation in the human colon and its relation to acetate concentration in venous blood. *J Clin Invest* 1985;75:1448–54.
35. McNeil NI. The contribution of the large intestine to energy supplies in man. *Am J Clin Nutr* 1984; 39:338–42.
36. Hayssen V, Lacy RC. Basal metabolic rates in mammals: taxonomic differences in the allometry of BMR and body mass. *Comp Biochem Physiol* 1965;81A:741–54.
37. Lupton JR, Coder DM, Jacobs LR. Long-time effects of fermentable fibers on rat colonic pH and epithelial cell cycle. *J Nutr* 1988;118:840–5.
38. Lupton JR, Coder DM, Jacobs LR. Influence of luminal pH on rat large bowel epithelial cell cycle. *Am J Physiol* 1985;249:G382–8.
39. Johnson IT, Gee JM. Gastrointestinal adaptation to soluble nonavailable polysaccharides in the rat. *Br J Nutr* 1986;55:497–505.
40. Wyatt GM, Horn N, Gee JM, Johnson IT. Intestinal microflora and gastrointestinal adaptation in the rat in response to non-digestible dietary polysaccharides. *Br J Nutr* 1988;60:197–207.
41. Goodlad RA, Ratcliffe B, Fordham JP, Wright NA. Does dietary fiber stimulate intestinal epithelial cell proliferation in germ free rats? *Gut* 1989;30:820–5.
42. Jacobs LR. Effect of dietary fiber on mucosal growth and cell proliferation in the small intestine of the rat: a comparison of oat bran, pectin and guar with total fiber deprivation. *Am J Clin Nutr* 1983; 37:954.
43. Guedon C, Schmitz J, et al. Decreased brush border hydrolase activities without gross morphologic changes in human intestinal mucosa after prolonged TPN in adults. *Gastroenterology* 1986;90:373.
44. Friedman E, Lightdale C, Winawer S. Effects of psyllium fiber and short chain organic acids derived from fiber breakdown on colonic epithelium cells from high-risk patients. *Cancer Lett* 1988;43:49–52.
45. Sakata T. Stimulatory effect of short-chain fatty acids on epithelial cell proliferation in the rat intestine: a possible explanation for trophic effects of fermentable fiber, gut microbes and luminal trophic factors. *Br J Nutr* 1987;58:95–103.
46. Sakata T. Effects of indigestible dietary bulk and short chain fatty acids on the tissue weight and epithelial cell proliferation rate of the digestive tract in rats. *J Nutr Sci Vitaminol* 1986;32:355–62.
47. Dexter DL, Lev R, McKendall GR, Mitchell P, Calabres P. Sodium butyrate-induced alteration of growth properties and glycogen level in cultured human colon carcinoma cells. *Histochem J* 1984; 16:137–49.
48. Van Berge Henegouwen GP, Van der Werf SDJ, RubenTh A. Effect of long-term lactulose ingestion on secondary bile salt metabolism in man: potential protective effect of lactulose in colonic carcinogenesis. *Gut* 1987;28:675–80.
49. Andrieux C, Gadelle D, Leprince C, Sacquet E. Effects of some poorly digestible carbohydrates on bile acid bacterial transformations in the rat. *Br J Nutr* 1989;62:103–19.
50. Anderson JW, Gustafson NJ. Hypocholesterolemic effects of oat and bran products. *Am J Clin Nutr* 1988;48:749–53.
51. Chen W-JL, Anderson JW, Jennings D. Propionate may mediate the hypocholesterolemic effects of certain soluble plant fibers in cholesterol-fed rats. *Proc Soc Exp Biol Med* 1984;175:215–8.
52. Beynen AC, Lemmens AG. Dietary acetate and cholesterol metabolism in rats. *Z Ernahrungswiss* 1987;26:79–82.
53. Ide T, Okamatsu H, Sugano M. Regulation of dietary fats of 3-hydroxy-3-methylglutaryl-CoA reductase in rat liver. *J Nutr* 1978;108:601–12.
54. Illmann RJ, Topping DL, McIntosh GH, et al. The hypocholesterolemic effects of dietary propionate: studies in whole animals and perfused rat liver. *Ann Nutr Metab* 1988;32:97–107.
55. Cheng B-Q, Trimble RP, et al. Comparative effects of dietary wheat bran and its morphological components on volatile fatty acid concentrations in the rat. *Br J Nutr* 1987;57:69–76.
56. Rolandelli RH, Koruda MJ, Settle RG, Leskiw MJ, Stein TP, Rombeau JL. The effect of pectin on hepatic lipogenesis in the enterally fed rat. *J Nutr* 1989;119:89–93.
57. Yamashito K, Kawai K, Itakura M. Effects of fructo-oligosaccharides on blood glucose and serum lipids in diabetic subjects. *Nutr Res* 1984;4:961–6.
58. Vahouny GV, Cassidy MM. Effect of dietary fiber on intestinal absorption of lipids. In: Spiller GA, ed. *Handbook of dietary fiber in human nutrition*. Boca Raton, FL: CRC Press, 1986:121–8.
59. Henry RJ. Pentosan and (1-3), (1-4)-β-glucan concentrations in endosperm and wholegrain of wheat, barley, oats and rye. *J Cereal Sci* 1987;6:253–8.

60. Welch R, Peterson DM, Schramka B. Hypocholesterolemic and gastrointestinal effects of oat bran fractions in chicks. *Nutr Rep Int* 1988;38:551–61.
61. Friedman M, Byers SO, Rosenman RH. Effect of glucagon on blood cholesterol levels in rats. *Lancet* 1971;2:464.
62. Quireshi AA, Burger WC, Peterson DM, Elson CE. The structure of an inhibitor of cholesterol biosynthesis isolated from barley. *J Biol Chem* 1986;261:10544–50.

DISCUSSION

Dr. Guesry: It is very fashionable today to recommend increased consumption of fiber. There is some advantage to increase the consumption of fiber, but higher consumption of fiber also increase consumption of phytic acid, which itself reduces calcium, iron, and zinc absorption. What are the differences between oat bran and other cereal brans, and to what extent does the intestinal flora adapt to changes in energy salvage from fibers?

Dr. Würsch: Wheat bran contains roughly 3% soluble fiber, whereas oat bran has up to 10%, beside 50% of raw starch. The soluble fiber is a β-glucan polymer, very viscous in solution. Oat bran, therefore, increases the viscosity of the food in the intestine in the same way as pectin or guar. However, what is surprising is that with a daily intake of 60 g oat bran (6 g β-glucan), a 20% decrease in blood cholesterol occurred (1), whereas more than 15 g guar was needed to obtain a comparable effect (2). The large intestinal microflora adapts very rapidly to any new carbohydrate substrate, generally within a few days. An increased dietary fiber consumption will increase the bacterial mass. Once it is stabilized, the energy saving for the host *via* volatile fatty acid absorption and metabolism should be unchanged in proportion.

Dr. Rossi: Do you consider that the intestinal flora is always constant? What is the situation in breast-fed infants?

Dr. Würsch: The case of breast-fed infants is very special. The amount of unabsorbed carbohydrates that reaches the large intestine is considerable, and their chemical structure is unique, favoring the growth of specific strains. In a diversified diet, a reasonable increase of unabsorbable carbohydrate from dietary origin, mostly dietary fiber, should not change the population profile dramatically.

Dr. O'Dea: I would like to comment on bioavailability of nutrients in high-fiber diets. Many of the normal processing methods, such as leavening of bread and cooking of legumes, will reduce the phytic acid content. Oat bran has unexpectedly high or strong effects in lowering cholesterol, given the relatively low level of β-glucan. A related point is that one of the major components of oat bran (and brans in general) is starch. It is uncooked and may be operated as a *de facto* fiber.

Dr. Würsch: Results of systematic studies of the effects of the different fractions of oat bran are starting to appear. Lund *et al.* (3) have compared the viscosity of the terminal ileum content in rats that received guar or β-glucan. The viscosity with β-glucan was higher than with guar, but they did not specify which guar they used. Absorption of minerals can be somewhat reduced by consumption of bean. However, in adults this effect is very low. Langkilde et al. (4) studied the excretion of minerals from diets including 174 g bean (dry weight) per day consumed by ileostomized subjects. They consumed 2 g phytate/day. Absorption of calcium, zinc, and iron only tended to be lower, but significantly so for magnesium. In other studies, Frölich and Sandström (5) found that the amount of zinc absorbed from whole flour bread (high-phytate, high-zinc) was still greater than that absorbed from refined,

unfortified wheat bran. Inhibition of mineral absorption was found to be greatest when phytic acid was added rather than when phytate was already present in the food.

Dr. Lifschitz: Breast-fed infants, rather than those who are formula fed, do not have propionate in their feces. Second, from a metabolic standpoint, the flora of breast-fed infants undergoes a process of maturation over the first few months of life, whereas that of formula-fed infants is very rapidly established soon after birth. From the metabolic standpoint, there are differences in the way the flora responds to acid pH. When feces of breast-fed infants are incubated anaerobically in the presence of excess lactose at acid pH, the hydrolysis of lactose is arrested, resulting in an accumulation of the sugar in the incubate. In the fecal incubates of formula-fed infants, lactose was hydrolyzed, but there was no further fermentation of glucose and galactose to volatile fatty acids. Others have shown that when normal, formula-fed infants were given rice cereal, both stool weight and fecal nitrogen increased, a finding similar to that observed when adults are given the unabsorbable carbohydrate, lactulose (6).

Dr. Würsch: Is it due to the more rapid turnover of the enterocytes?

Dr. Lifschitz: There is an increased bacterial mass, but indirect evidence from some animal studies suggests that animals given a high-fiber diet had an increase in not only fecal flora mass but also DNA, which probably originated from the enterocytes. Therefore, nonabsorbable or not completely absorbable nutrients might affect not only the bacterial flora but also small bowel cell turnover.

Dr. Wahlqvist: Methodological shortcomings have contributed to the kind of confusion that you have addressed in respect to oat bran versus other sorts of dietary fibers from foods. It is not surprising that we have what was referred to a moment ago as "crazy food" and "crazy nutrition." It is interesting how often we, from the scientific community, apportion the responsibility for that on the consumer when, in reality, it is a by-product of the scientific process in which we ourselves are engaged. That requires us to develop new and complementary approaches and methodologies. We are bound to engage in much more mathematical modeling of food and food intake and also much better factorial or multilogistic analysis of nutrients, such as dietary fiber, and, more than that, the food from which it comes.

Dr. Truswell: There are three special features about oat bran. One is that oatmeal contains a lot of polyunsaturated lipid, some of which is in the bran. Feeding a simulated oat lipid with 120 g/day of rolled oats caused an 8% fall in the serum cholesterol (7). Part of the fall with oat products may be a polyunsaturated fatty acid effect. Second, you should look not only at the Lexington data. There are other studies, most of which do not show such an effect. I suspect that a major effect of these soluble fibers on cholesterol metabolism is through an effect on viscosity in the terminal ileum (8), which is where the bile acids are reabsorbed. In many studies where there has been a reduction in the plasma cholesterol, there has been an increase in the fecal bile acid excretion.

Dr. Gracey: I want to introduce a word of caution about the intestinal microflora. When you spoke about that Japanese study and the tenfold change in a particular intestinal bacterial species, that is a change of only one log of that bacterial population, which in microbiological terms is quite a small change in magnitude.

Dr. Guesry: I would like to come back to phytic acid. Kerin O'Dea minimized the importance of this, probably because she is working with adults. For a 6-month-old baby, the 1% phytic acid that you mention, in soya formula, is able to reduce the absorption of iron and zinc by 50%. As a pediatrician I am worried that this trend or fashion of all-bran types of food for adults would reach infant foods and would be deleterious in babies.

Dr. Holdsworth: We have been studying the effect of a completely fiber-free diet in the

form of enterally fed elemental diets in Crohn's disease. Over a 1-week and 1-month period, we found no change in aerobic or anaerobic viable counts, qualitative or quantitative, apart from a fall in lactobacilli in some subjects. The diet happens to be free of lactose.

REFERENCES

1. Anderson JW, Gustafson NJ. Hypocholesterolemic effects of oat and bran products. *Am J Clin Nutr* 1988;48:749–53.
2. Jenkins DJA, Reynolds D, Slavin B, et al. Dietary fiber and blood lipids: treatment of hypercholesterolemia with guar crispbread. *Am J Clin Nutr* 1980;33:575–80.
3. Lund EK, Gee JM, Brown JC, Wood PJ, Johnson IT. Effect of oat guar on the physical properties of the gastrointestinal contents and on the uptake of D-galactose and cholesterol by rat small intestine in vitro. *Br J Nutr* 1989;62:91–101.
4. Langkilde AM, Andersson H, Schweizer TF, Torsdottir I. Nutrients excreted in ileostomy effluents after consumption of mixed diets with beans or potatoes. I. Minerals, protein, fat and energy. *Eur J Clin Nutr* 1990;44:559–66.
5. Frölich W, Sandström B. Zinc absorption from composite meals. In: Inglett GE, ed. *Nutritional bioavailability of zinc*. Washington, DC: American Chemical Society, 1983;211–21.
6. Shulman RJ, Boutton TW, Klein PD. Impact of dietary cereal on nutrient absorption and fecal nitrogen loss in formula fed infants. *Pediatr Res* 1989;25:730A.
7. Judd PA, Truswell AS. The effect of rolled oats on blood lipids and fecal steroid excretion in man. *Am J Clin Nutr* 1982;34:2061–7.
8. Judd PA, Truswell AS. The hypocholesterolaemic effects of pectins in rats. *Br J Nutr* 1985;53:409–25.