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Physiological effects of dietary carbohydrates in the large bowel: Is there a need to recognize dietary fibre equivalents?

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Dietary carbohydrates entering the large bowel can affect its physiology either through their physical presence or by fermentation by the microflora. Major fermentative end-products include short chain fatty acids (SCFA). In adults the principal acids are acetate, propionate and butyrate which act to promote normal colonic function and may mediate many effects ascribed formerly to dietary fibre. Butyrate is thought to be especially important for the maintenance of a normal colonocyte population. Short chain fatty acid levels appear to be high in the human proximal colon and fall towards the distal region through their uptake on passage of the faecal stream. This distribution is important as most large bowel pathology occurs in the distal colon. While non-starch polysaccharides (NSP), major components of dietary fibre, are important fermentative substrates, resistant starch (RS) may be more significant quantitatively. Thus, large bowel SCFA are raised by the consumption of fibre but the magnitude of the change is variable. Certain processed foods appear to contain substantial amounts of RS which can produce major elevations in SCFA. However, the changes in total and individual SCFA are not uniform between foods so that an RS source which can be used as a manufacturing ingredient has considerable potential benefit. High amylose starches are one such option and consumption of foods containing them increases faecal SCFA and butyrate. Acylated starches represent a further improvement on such starches as they should survive processing conditions of high temperature and moisture which would gelatinize high amylose starches. Acylated starches have been shown to raise large bowel SCFA in rats and are being examined for their potential to deliver specific acids. These starches and other novel products which can improve large bowel health are not assayed in current fibre analyses. It may be time to think in terms of 'fibre equivalents' so as to include carbohydrates which are not from traditional fibre sources but have physiological actions similar to fibre.

Key words: carbohydrates, large bowel, dietary fibre, short chain fatty acids, resistant starch.

Introduction

Consumption of carbohydrate-rich foods is believed to confer a number of benefits in terms of the lessened risk of non-infectious degenerative diseases. In the case of the large bowel, this translates to an expectation of protection against conditions such as constipation, diverticular disease and colorectal cancer. For dietary carbohydrates to influence the large bowel directly they must bypass digestion in the small intestine. This is known to be the case for a wide range of naturally occurring plant non-starch polysaccharides (NSP) which are major components of dietary fibre and for which the human small intestine lacks the intrinsic hydrolases.[1]

Other natural or synthetic carbohydrates such as fructo- and galacto-oligosaccharides, lactose, sorbitol and lactulose also may escape digestion either through a similar enzyme deficit or absorption through the lack of a suitable transport mechanism. Additionally, a significant fraction of ingested starch (resistant starch (RS)) escapes small intestinal amylolysis for a number of reasons. These include physical inaccessibility, granular structure or lack of gelatinization and/or retrogradation and chemical modification (Table 1).[2]

In the caecum and colon these carbohydrates can affect large bowel function in two ways. Firstly, they can increase digesta mass and, hence, faecal bulk. This has been shown for wheat bran which raises stool mass in a dose-dependent manner[3] through passage of undigested fibre (mainly as NSP with a small amount of lignin).[4] Wheat bran and similar food products are effective in the rapid promotion of laxation[5] and in reducing the risk of diverticular disease in the long term.[6] While some fibre-rich foods affect the large bowel through their physical presence, it appears that the actions of many are mediated through short chain fatty acids (SCFA) produced by the fermentation of dietary carbohydrates by the resident microflora. This appears to be the case for RS, which may be a very important fuel for the colonic bacteria.

Short chain fatty acid production and actions of dietary carbohydrates in the large bowel

Short chain fatty acids are produced through the anaerobic fermentation of carbohydrates by a complex colonic bacterial ecosystem.[1] In essence, the reactions are carried out by the bacteria in order to provide metabolizable energy for their growth and cellular maintenance. These organisms derive nitrogen either from protein or as NH_3 from urea via the urease reaction. Acetate, propionate and butyrate are the

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Table 1. Nutritional classification of resistant starches

Types of resistant starch	Examples of occurrence
RS1 – Physically inaccessible	Partly milled grains and seeds
RS2 – Resistant granules	Raw potato, green banana, pulses, high amylose starches
RS3 – Retrograded	Cooked and cooled potato, bread, cornflakes
RS4 – Chemically modified	Etherised, esterified, cross-bonded starches (used in processed foods)

major SCFA produced by this fermentation in normal adult humans and generally they are present in faecal samples in the following order: acetate > propionate ≥ butyrate. Lesser amounts of other acids, including lactate, succinate and formate, can occur and branched chain acids may be produced through the degradation of amino acids. In addition to SCFA, other end-products of fermentation include heat, water and gases: CO_2, CH_4 and H_2.

While this paper is directed towards a review of large bowel metabolism in humans consuming adult foods, it should be noted that the fermentative products in milk-fed infants are rather different. In faecal samples from these children, acetate is a major acid (as in adults) but the contribution of butyrate seems to be very low.[7] Studies with faecal inocula suggest that substantial quantities of other metabolites, including formate and ethanol, may be formed in infants.[8] The significance of this particular product profile to the colonic physiology of infants needs to be established.

Short chain fatty acids are the major anions in the large bowel of humans and other omnivores such as the dog and pig.[9] These acids exert a number of actions on large bowel metabolism which appear to be critical to the maintenance of normal colonic function (for detailed reviews of SCFA action, see Cummings *et al.*).[10] One effect which appears to be common (at least for the major acids) is a lowering of luminal pH. This is thought to be of benefit as a more acid colonic environment leads to inhibition of the growth of pH-sensitive pathogenic organisms. Moreover, under acid conditions basic compounds with toxic or carcinogenic potential become ionized and so are not absorbed by the mucosa, while acidic potential carcinogens such as secondary bile acids or free fatty acids may become insoluble through loss of ionization.

All of the major SCFA appear to stimulate colonic blood flow and muscular activity and they also appear to promote the absorption of cations, especially Na^+, K^+ and Ca^{2+}. There is also some evidence that SCFA (or at least fermentation) slows upper intestinal transit through activation of the ileocolonic brake.[11] In the large bowel, acetate, propionate and butyrate seem to promote colonocyte proliferation but butyrate alone seems to have specific benefits. In addition to repairing diversion and ulcerative colitis, butyrate appears to be a major metabolic fuel for normal colonocytes. As is discussed elsewhere in this symposium, abundant *in vitro* data show that this acid acts to promote a normal phenotype in colonocytes and to oppose the growth of malignant cells, although confirmation of this effect *in vivo* remains to be obtained.

The distribution of colonic short chain fatty acids in humans: Implications for large bowel health

Consideration of the anatomy of the human large bowel suggests that fermentation should be localized in the caecum and proximal colon as these are the regions closest to the small intestine. Consequently, they are the first to encounter the digesta stream after it has passed though the ileocaecal valve and are the regions of the large bowel where the concentration of potential fermentative substrates should be highest. It follows that fermentation should be most rapid in the proximal hind gut and that SCFA levels should also be highest in that area, reflecting their greater production. Additionally, it would be expected that SCFA availability should fall towards the distal colon through their absorption and metabolism coupled with diminished formation through substrate depletion. Early comparative studies in animals suggested that this was the case with high concentrations of SCFA solutions in the proximal hind gut, declining towards the distal colon.[12]

This profile has been confirmed in subsequent studies in pigs fed diets containing various sources of fibre.[13–15] Comparable studies in humans are difficult for a number of obvious ethical and logistic reasons, although Cummings *et al.* have made determinations of SCFA in trauma victims.[16] Manifestly, this approach is a very limited one from the standpoint of systematic experimentation, as are studies in which the colon is intubated. One means of accessing digesta contents directly in humans is to examine SCFA in the stomal effluent from surgical patients. Such an investigation has been done in individuals with ileostomy and transverse or sigmoid colostomy when it was reported that SCFA levels were very low in ileal effluent, high in collections from the transverse colon and low in those from the sigmoid colon.[17] This profile is very similar to that recorded in pigs and accords with the concept that human SCFA production predominates in the proximal colon and that the availability of these acids may be limited in the distal colon. This is an important consideration as the risk of important colonic pathologies such as colorectal cancer is much greater in the distal colon.[18] If, as appears likely, the limited availability of SCFA (especially butyrate) in the distal colon is a factor in such disease states, then means of delivering them to that region may be of great benefit in improving disease risk.

Studying bowel short chain fatty acids: The need for animal models

An understanding of the factors which influence human colonic SCFA is critical in designing strategies to effect their delivery to the organ as a whole as well as to the sites of maximum need. Surgical patients are one means of achieving this goal and a limited study in eight individuals with transverse colostomy has shown that it is possible to modify large bowel SCFA.[19] This was achieved by feeding either a wheat bran or an oat plus barley breakfast cereal. Butyrate excretion was unchanged over the 5 days of the experimental trial in the former group but declined on day 5 when the subjects consumed the oat plus barley product.

While these data are generally similar to those obtained from animals, they show the limitations of studies in surgical patients. It proved difficult to recruit individuals with stoma in the proximal colon and the time for which they were available to participate in the study was short. There are also two intrinsic problems, that is, that such patients have had a serious organic disease and that they have undergone surgery for this problem; both are factors which could compromise the data. Clearly, the limited avenues for human experimentation necessitate the use of suitable animal models. While the rat is used widely, it has a number of deficiencies including the fact that it is a caecal fermenter and has a complex musculature which retains fermentable material in the caecum.[9] Consequently, the distribution of SCFA in the large bowel[20] is quite dissimilar to that in humans. Further, rats practice faecal refection, the abolition of which has effects on large bowel SCFA which are dependent on the type of dietary fibre which is fed.[21] Finally, rats do not possess a gallbladder which means that they are recycling bile acids semicontinuously. This is an important difference between this species and humans and may influence SCFA as animal studies show that greater bile acid excretion lowers large bowel butyrate levels.[22]

Of the animal species which are available, the pig appears to be the most suitable as it has a gastrointestinal system close to humans with a gallbladder and a comparable caecum and colon. However, the porcine large bowel is rather large in comparison to humans, which necessitates the feeding of large quantities of fibre.[23] Pigs eat a very wide range of common human foods in quantities reasonably similar to those eaten by people. When they consume diets enriched in NSP by the addition of fibre concentrates such as rice bran[14] or wheat bran,[15] total large bowel digesta mass and SCFA pools are elevated, consistent with human faecal data.

Foods and large bowel short chain fatty acids: Analytical versus physiological resistant starch

Although the data from pigs show that increased NSP consumption raises large bowel SCFA pools and digesta masses, they show also that these variables are disproportionately higher when certain foods are fed at levels of NSP equivalent to fibre concentrates. For example, when brown rice or canned navy (baked) beans are fed, both SCFA and digesta are higher by 150% or more in comparison with rice or wheat bran, respectively.[14,15] The difference has been ascribed to starch which has escaped small intestinal digestion even though the RS content of the former foods is quite low when measured by standard techniques. The latter measure of RS has been termed 'analytical RS' while that determined *in vivo* as increases in SCFA or digesta has been described as 'physiological RS'.[24] Physiological RS can add considerably to the effective fibre content of a food in its influence on large bowel fermentation and thus can have a greater impact on bowel health and disease risk greater than might be suggested by the analytical data.

Studies with pigs offer further insights into the relationships between diet and the distribution of SCFA in the large bowel. From studies *in vitro* with human faecal inocula[25] it has been assumed that RS fermentation favoured butyrate production; an effect which could help to account for the relationship between greater starch consumption and diminished risk of colorectal cancer obtained from a meta-analysis of human population studies.[26] However, the animal data show that not all sources of RS are equivalent in their effects on SCFA. The fermentation of baked beans appeared to favour propionate production while that of brown rice enhanced butyrate. One reason for this difference lies in individual variation in the large bowel bacteria. This may occur in people as the faecal flora in some individuals appears to be unable to ferment some forms of RS.[27] These differences need not be an effect of starch alone given the suggestion in a recent report that in rats, the type of dietary protein may influence large bowel RS fermentation both in terms of rate and the SCFA which are formed.[28]

A further point which emerges from studies in pigs is that not only does diet affect the molar ratios of the SCFA which are formed, but also it influences their distribution along the colon. Thus, with baked beans, total SCFA levels were relatively high in the proximal colon compared with brown rice where the acids were disproportionately higher in the distal colon. These data are of some relevance to the risk of human large bowel disease which is greatest in the region where other risk factors may be high and SCFA are lowest.[29] Equally importantly, it appears that SCFA levels in the distal colon cannot be predicted from those in the proximal region. The most likely explanation for this variation between foods is the rate of fermentation in relation to their carriage along the colon in the digesta stream. This would account for the lack of change in faecal SCFA following consumption of some forms of RS despite evidence of significant colonic fermentation as evidenced by increased breath H_2 evolution.[30]

These animal data have revealed an additional, potentially important, factor which is not always addressed in human feeding trials, that is, adaptive changes in the microflora in response to dietary change. That this could be an influence has been shown in a further comparative study in which pigs were fed brown rice or white rice plus rice bran (controls) for 3 weeks.[31] Faecal samples taken at weekly intervals showed that starch excretion was higher in the brown rice group during week 1 but declined to the levels found in animals fed rice bran in week 2. Excretion of SCFA (including butyrate) showed a similar pattern although values remained significantly higher than in controls at week 2. One explanation for this profile is the time taken for the colonic microflora to adapt to new foods. If this is the case, then studies on RS and SCFA production should be designed to ensure that any acute adaptive effects are maintained in the longer term.

High amylose and chemically modified starches as a means of increasing the resistant starch content of foods

The sum of the animal and (relatively limited) human data indicate that increased bioavailability of SCFA, especially butyrate, in the distal colon is important for the promotion of large bowel health and reducing the risk of serious illnesses including cancer. It appears that some starchy processed foods are particularly effective in achieving this aim through their intrinsic RS content. However, the consumption of starches in Australia is relatively low by international standards so that efforts to increase the consumption of such foods could well require substantial promotional and educational efforts.[26] An alternative is to raise the RS content of other processed foods by fortification, that is, through the addition of particular types of RS. This has been achieved through the use of high amylose starches which are more resistant to gelatinization and retrograde more rapidly on cooling than do starches high in amylopectin.[2] Studies by van

Munster *et al.* in human volunteers showed that a consumption of high amylose starch promoted SCFA excretion and changed the profile of other faecal variables (such as pH and secondary bile acids) to favour large bowel health.[32] In Australia a high amylose starch has been incorporated into a range of food products including bread and breakfast cereals and consumption of such products has been shown to enhance SCFA and butyrate excretion.[33] Clearly, if these data translate to the general population who consume such products, they promise to improve large bowel health considerably.

While high amylose starches can raise the RS content of certain foods, their application is inherently limited as they may become gelatinized (and hence susceptible to small intestinal amylolysis) under the conditions of heat and moisture used in food processing. There is also the limitation that the products of RS fermentation depend on the mix of bacterial species in an individual's colon. There appears to be scope for a cheap and effective means of delivering specific SCFA to the distal large bowel which is less susceptible to these influences. This may be achieved by using carbohydrates, specifically starches, acylated with the desired SCFA. Such starches are resistant to small intestinal amylolysis and so pass into the large bowel. This attribute has been explored recently in a study where it was shown that starches modified through either acetylation or attachment of β-cyclodextrin gave lower glycaemic indices than did unmodified starches.[34]

The potential of acylated starches to deliver SCFA to the large bowel appears to be largely unexplored. We have examined this possibility in rats fed either a control starch or a starch acylated to a level of 5% with either acetate, propionate or butyrate.[35] Short chain fatty acid concentrations and pools were low in the caecum and colon of rats fed the unmodified (control) starch. In animals fed the acylated starches total SCFA were raised in all regions of the large bowel although digesta mass was raised only in the caecum. These higher SCFA values presumably reflect the fermentation of the RS present in those diets. However, the greatest increase in each case was in the particular SCFA acylated to the starch. Thus, the highest levels of butyrate were found in rats fed the butyrylated starch. These data are consistent with the passage of RS4 into the caecum followed by release of the acyl units (due to bacterial lipase and/or esterase activity), and hydrolysis of the residual starch with fermentation of the released glucose. Acylated starches have the advantage that, as RS4, they are free of some of the problems associated with other types of RS, especially in food processing. Thus, acylated starches should be able to survive the conditions of heat and moisture which lead to gelatinization of other starches. Products containing these starches are currently under development.

Expanding the definition of dietary fibre to include new carbohydrates: The concept of 'fibre equivalents'

When Burkitt, Trowell and coworkers carried out the pioneering work which led to what has become known as the 'fibre hypothesis', they were comparing disease risks in a population of native Africans consuming an unrefined diet with Europeans eating highly processed foods.[36] Only subsequently did attention shift from the diet as a whole to the fibre component. Largely because of the importance of fibre in the nutrition of ruminant farm animals (for which plant cell wall polysaccharides are very important as substrates for the rumen microflora), the analytical technology was relatively restricted in the range of carbohydrates which were measured. Effectively, adequate analysis was limited to foods (such as wheat bran) which were high in what has become known as 'insoluble fibre' or 'roughage'. Only latterly has the methodology become available to measure the full range of carbohydrates which meet the definition of dietary fibre as the structural and exudative polysaccharides of plants (plus lignin) which are not digested in the human small intestine but pass into the large bowel. However, this definition still hinges on the indigestibility of structural and exudative polysaccharides and overlooks storage and other carbohydrates which escape small intestinal digestion. In essence, it is a coupling of origin with chemical composition and analysis.

This approach tends to minimize the potential for foods containing dietary fibre to improve health, which would seem to be the reason for interest in them in the first place. Thus, wheat bran might be high in insoluble NSP (which would be reflected in analytical values for fibre) but one could argue that its capacity to promote laxation was much more pertinent to health scientists and consumers. Such an argument would accord with the recent granting of a health claim by the United States Food and Drug Administration for oat bran to be recognized as a dietary aid for the lowering of plasma cholesterol.[37] The balance of the evidence favours the soluble fibre component of such foods as the active agent. Should that be the case, other compounds could be regarded as equivalent to fibre in this regard and hydroxypropyl methylcellulose (HPMC) is a ready example. Based on animal studies, this synthetic polysaccharide, not degraded by human small intestinal enzymes, appears to be a very effective cholesterol lowering agent.[38] Further, the lowering of plasma cholesterol by HPMC appears to be through either greater faecal steroid excretion or diminished small intestinal steroid absorption, both of which are candidate mechanisms for the hypocholesterolaemic action of 'natural' NSP such as β-glucan from oats.

It is to be expected that other instances of the fibre-like actions of novel foods and food ingredients which are not recognized as fibre currently will emerge. Thus, it may be appropriate to consider such carbohydrates and other food components which have those actions as 'fibre equivalents'.[36] This concept of equivalence is established for vitamin A and, more recently, has been proposed for folate.[39] Fibre equivalents could be defined as carbohydrates from any source having actions similar to those described for fibre; for example, faecal bulking, cholesterol reduction, laxation, fermentation (with SCFA production) and modification of the large bowel flora. This list is not exhaustive and recognizes major effects only. Nevertheless, it does open up the possibility of measuring fibre in physiological terms although analyses are required in order to meet the needs of nutrient labelling and consumer education. Such a change would enable all non-digested carbohydrates (and other food components) to be ranked in terms of their effectiveness in particular areas of fibre action.

References

1. Cummings JH, Macfarlane GT. The control and consequences of bacterial fermentation in the human colon. J Appl Bacteriol 1991; 70: 443–459.
2. Brown IL, McNaught KJ, Moloney E. Hi-maize™: new directions in starch technology and nutrition. Food Aust 1995; 47: 272–275.
3. Jenkins DJA, Peterson RD, Thorne MJ, Ferguson PW. Wheat fiber and laxation: dose response and equilibration time. Am J Gastroenterol 1987; 82: 1259–1263.
4. Stephen AM, Cummings JH. Mechanism of action of dietary fibre in the human colon. Nature 1980; 284: 283–284.
5. Baghurst KI, Hope AK, Down EC. Dietary intake in a group of institutionalised elderly and the effect of a fibre supplementation programme on nutrient intake and weight gain. Comm Health Stud 1985; 9: 99–108.
6. Aldoori W, Giovannucci EL, Rockett HR, Sampson L, Rimm EB, Willett WC. A prospective study of dietary fiber types and symptomatic diverticular disease in men. J Nutr 1998; 128: 714–719.
7. Edwards CA, Parrett AM, Balmer SE, Wharton BA. Faecal short chain fatty acids in breast-fed and formula-fed babies. Acta Paediatr 1994; 83: 459–462.
8. Wolin MJ, Yerry S, Miller TL, Zhang Y, Bank S. Changes in production of ethanol, acids and H_2 from glucose by the fecal flora of a 16- to 150-d-old breast-fed infant. J Nutr 1998; 128: 85–90.
9. Stevens CE, Hume ID. Contributions of microbes in vertebrate gastrointestinal to production and conservation of nutrients. Physiol Rev 1998; 78: 393–427.
10. Cummings JH, Rombeau JL, Sakata T, eds. Physiological and clinical aspects of short-chain fatty acids. Cambridge: Cambridge University Press, 1995.
11. Ropert A, Cherbut C, Roze C, Le Quellec A, Holst JJ, Fu-Cheng X, Bruley des Varannes S, Galmiche JP. Colonic fermentation and proximal gastric tone in humans. Gastroenterol 1996; 111: 289–296.
12. Elsden SR, Hitchcock MWS, Marshall RA, Phillipson AT. Volatile acid in the digesta of ruminants and other animals. J Exp Biol 1946; 22: 191–202.
13. Bach-Knudsen KE, Jensen BB, Andersen JO, Hansen I. Gastrointestinal implications in pigs of wheat and oat fractions. 2. Microbial activity in the gastrointestinal tract. Br J Nutr 1991; 65: 223–248.
14. Marsono Y, Illman RJ, Clarke JM, Trimble RP, Topping DL. Plasma lipids and large bowel volatile fatty acids in pigs fed white rice, brown rice and rice bran. Br J Nutr 1993; 70: 503–513.
15. Topping DL, Illman RJ, Clarke JM, Trimble RP, Jackson KA, Marsono Y. Dietary fat and fiber alter large bowel and portal venous volatile fatty acids and plasma cholesterol but not biliary steroids in pigs. J Nutr 1993; 123: 133–143.
16. Cummings JH, Pomare EW, Branch WJ, Naylor CPE, Macfarlane GT. Short chain fatty acids in the human large intestine, portal, hepatic and venous blood. Gut 1987; 28: 1221–1226.
17. Mitchell BL, Lawson MJ, Davies M, Kerr-Grant A, Roediger WEW, Illman RJ, Topping DL. Volatile fatty acids in the human intestine: studies in surgical patients. Nut Res 1985; 5: 1089–1092.
18. Correa P, Haenszel W. The epidemiology of large-bowel cancer. Adv Cancer Res 1978; 26: 1–141.
19. Pant I, Topping DL, Wong SH, Shearman DJ, Farmakalidis E. Fermentation of soluble and insoluble fibre in the human colon. In: Wahlqvist ML, Truswell AS, Smith R, Nestel P, eds. Nutrition in a Sustainable Environment. London: Smith-Gordon, 1994; 895.
20. Illman RJ, Topping DL. Effects of food restriction and starvation-refeeding on volatile fatty acid concentrations in the rat. J Nutr 1986; 116: 1694–1700.
21. Jackson KA, Topping DL. Prevention of coprophagy does not alter the hypocholesterolaemic effects of oat bran in the rat. Br J Nutr 1993; 70: 211–219.
22. Illman RJ, Storer GB, Topping DL. White wheat flour lowers plasma cholesterol and increases cecal steroids relative to whole wheat flour, wheat bran and wheat pollard in rats. J Nutr 1993; 123: 1094–1100.
23. Van Soest PJ. Comparative aspects of animal models. In: Kritchevsky D, Bonfield C, eds. Dietary fiber in health and disease. St Paul: Eagan Press, 1995; 321–339.
24. Annison G, Topping DL. Resistant starch: Chemical structure vs physiological function. Ann Rev Nutr 1994; 14: 297–320.
25. Weaver GA, Krause JA, Miller TL, Wolin MJ. Cornstarch fermentation by the colonic microbial community yields more butyrate than does cabbage fermentation; cornstarch fermentation rates correlate negatively with methanogenesis. Am J Clin Nutr 1992; 47: 61–66.
26. Cassidy A, Bingham SA, Cummings JH. Starch intake and colorectal cancer risk: an international comparison. Br J Cancer 1994; 69: 937–942.
27. Cummings JH, Beatty ER, Kingman SM, Bingham SA, Englyst HE. Digestion and physical properties of resistant starch in the human large bowel. Br J Nutr 1996; 75: 733–747.
28. Morita T, Kasaoka S, Oh-hashi A, Ikai M, Numasaki Y, Kiriyama S. Resistant proteins alter caecal short-chain fatty acid profiles in rats fed high amylose cornstarch. J Nutr 1998; 128: 1156–1164.
29. Cats A, DeVries EGE, Mulder NH, Kleibeuker JH. Regional diffrences of physiological functions and cancer susceptibility in the human large intestine. Int J Oncol 1996; 9: 1055–1069.
30. Tomlin J, Read NW. The effect of resistant starch on colon function in humans. Br J Nutr 1990; 64: 589–595.
31. Bird AR, Illman RJ, Hyakawa T, Topping DL. Brown rice increases faecal and large bowel short-chain fatty acids in pigs. Proc Nutr Soc Aust 1998; 21: 135.
32. van Munster IP, Tangerman A, Nagengast FM. Effect of resistant starch on colonic fermentation, bile acid metabolism, and mucosal proliferation. Digestive Dis Sci 1994; 39: 834–842.
33. Noakes M, Clifton P, Nestel PJ, Le Leu R, McIntosh GH. Effect of high amylose starch and oat bran on metabolic variables and bowel function in subjects with hypertriglyceridemia. Am J Clin Nutr 1996; 64: 944–951.
34. Raben A, Andersen K, Karberg MA, Holst JJ, Astrup A. Acetylation of or β-cyclodextrin addition to potato starch – beneficial effects on glucose metabolism and appetite sensations. Am J Clin Nutr 1997; 66: 304–314.
35. Annison G, Topping DL, Illman RJ, Trimble RP, McGrath L. Novel food ingredients for delivering specific short chain fatty acids to the large bowel. Proc Nutr Soc Aust 1998; 22: 91.
36. Cobiac L, Topping DL. Dietary Fibre: Potential role for in the aetiology of disease. In: Sadler M, Caballero B, Strain S, eds. Encyclopaedia of Human Nutrition. London: Academic Press, 1999: 546–552.
37. Food and Drug Administration. Food labelling: health claim; oats and coronary heart disease: final rule (21 CFR 101). Federal Register, January 23, 1997; 62: 3584–3601.
38. Gallaher DD, Hassel CA, Lee KJ. Relationships between viscosity of hydroxypropyl methylcellulose and plasma cholesterol in hamsters. J Nutr 1993; 123: 1732–1738.
39. Baily LB. Dietary reference intakes for folate: the debut of dietary folate equivalents. Nutr Rev 1998; 56: 294–299.