

# Colorectal disorders: A dietary management perspective

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Dietary lifestyle is relevant for prevention and treatment of various colorectal conditions. Colorectal disorders have significant morbidity and mortality in a western-style community, particularly irritable bowel syndrome (IBS), colorectal cancer, haemorrhoids, constipation and diverticular disease. This review addresses how bowel health can be maintained, what foods and dietary lifestyles are associated with risk for disease and what foods are of real value in management. Bowel health is that state where the individual is satisfied with defaecation, the diet does not create undue risk for disease and lumenal contents maintain an intact and functional mucosa. Bowel health depends on a healthy dietary lifestyle, but in particular on an adequate intake of non-digestible dietary polysaccharide. Diet influences biology in part by altering the lumenal environment. Effects such as high butyrate levels, lowered pH, a predominance of 'healthy' over 'unhealthy' bacteria, rapid intestinal transit, high faecal bulk, a non-leaky epithelial barrier, adsorption of dietary carcinogens by fibre, low bile salt concentrations, reduced generation of toxic bile salts or protein derivatives and provision of certain bioactive substances are seen as beneficial. Diet influences future risk for colorectal cancer (vegetables, animal fats, polysaccharides amongst others) and for diverticular disease (fibre). Adequate fibre and resistant starch can improve constipation and anorectal conditions such as fissure and haemorrhoids. The role of diet in managing patients with IBS is complex. Fibre may worsen symptoms in severe cases of IBS, diverticular disease and inflammatory bowel disease. Certain carbohydrates of limited digestibility/absorbability, such as lactose, fructose and sorbitol, can precipitate IBS symptoms. Low fat, high fibre diets may reduce recurrence of colorectal adenomas. Diet has a significant role to play in colorectal disorders.

**Key words:** colon, rectum, nutrition, cancer.

## Introduction

Dietary lifestyle is highly relevant to the health of the colon and rectum. Not only is the balance of what we consume and our overall nutritional state important to maintenance of bowel health but also to prevention and treatment of colorectal diseases. Components of the diet influence function and biology of the bowel both directly and indirectly, although much of the effect is exerted via the complex lumenal environment.<sup>1</sup> Food consumption varies widely around the world and patterns of colorectal disease can be matched to patterns of food consumption and use of food supplements.<sup>2</sup> A significant proportion of colorectal disorders follow the Western-type dietary lifestyle.

Often, the community's view of bowel health stems from misunderstandings. 'We are what we eat' is a common cliché that is not entirely true because inherited factors, especially for colorectal cancer (CRC), are also important. In some instances, we inherit metabolic characteristics that determine how we interact with the environment. Many in the community are also of the view that 'bowel function' in some way reflects our overall health or indeed influences it. For example, the commonly held misconception that toxins are present in the large bowel, which must be evacuated fully. Food or food-derived health products may also be seen as a safe way to manage disease and maintain health. While this may be true in specific instances as described later, the effect of diet is subtle and slow. Where 'food' components are rapidly active, such as herbal therapies for constipation, the fact that such an agent should really be considered as another

form of pharmaceutical agent is often overlooked. A food or food-derived solution is rarely a rapid solution for an established disease.

Colorectal disorders have significant morbidity and mortality in a Western-style community. Irritable bowel syndrome (IBS) affects more than 15% of adults<sup>3</sup> and CRC affects just over 5% of Australians.<sup>4</sup> Haemorrhoids and constipation are very common although their precise frequency is unclear because of problems created by definitions and the logistic difficulties involved in obtaining data. Diverticular disease gets progressively common with ageing.<sup>5</sup>

In considering the issue of diet and our bowels, we will address several issues: what is bowel health and how is it maintained? What aspects of food consumption and dietary-lifestyle are associated with a risk for developing a given disease? If we change our lifestyle, do we change risk? What is of real value once a given disease has developed? Each of these requires a different approach. Note that in the following discussion, 'diet' will often be loosely used to encompass the overall food balance, the dietary lifestyle (including resultant nutritional state), individual foods, food components, food-derived products, micronutrients and even herbal products.

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### Bowel health

'Bowel health' is becoming a frequently used phrase although it has no clear definition. Definitions will vary depending on whether one is a lay person, nutritionist, physician or biologist. To satisfy all to some degree, the following, obviously complex, definition will be used here: 'Bowel health is that state where the individual is satisfied with their own frequency and ease of defaecation. The balance of food consumed does not place them at undue risk for disease, and there is active luminal fermentation, which maintains an intact and functional mucosa.' This incorporates subjective and objective issues about function, luminal fermentation and biology, and an overall pattern of food consumption and its nutritional consequence.

### Healthy bowel function

What constitutes healthy function clearly means different things to different people but a goal of a regular, soft stool passed at a convenient frequency, under control and without lingering rectal discomfort is adequate for most. Any other requirements would seem to reflect psychological disorders or unrealistic requirements. An adequate intake of 'fibre'; that is, non-digestible polysaccharides (NDP; resistant starch and non-starch polysaccharides) is normally sufficient to achieve this. What constitutes enough fibre varies between individuals and must be determined on an individual basis. Dietary fibre is best derived from multiple sources although in the context of laxation, insoluble non-starch polysaccharide is probably the most effective. Too much fibre, again the exact amount varying between individuals, causes bloating, excess passage of wind, colic and windy rumbles (borborygmi), but this is easily determined by experimentation.

### Active luminal fermentation

Active luminal fermentation is largely ensured by NDP consumption.<sup>6,7</sup> In the colon, carbohydrates are fermented by anaerobic bacteria with a range of consequences including production of substantial quantities of the short chain fatty acids (SCFA) acetate, propionate and butyrate, gases such as H<sub>2</sub>, CO<sub>2</sub> and methane, and a lowered pH.<sup>7</sup> Short chain fatty acids provide energy and maintain the colonic epithelial cell mass,<sup>8</sup> they promote fluid absorption and ensure passage of a convenient amount of faecal water.<sup>7</sup> In severe diarrhoeal states such as cholera, they facilitate colonic salvage of water and minimize dehydration.<sup>9</sup> In particular, butyrate has certain cancer-protecting actions as does a lowered pH.<sup>8,10</sup> Bacteria are essential for fermentation but our ability to manipulate them are greatly restricted compared to the ease of manipulating NDP. Furthermore, our understanding of what constitutes a healthy as opposed to unhealthy microflora, is poor when one insists on proof that links observation to presence of disease or dysfunction.

In summary, bowel health will depend to a large degree on an overall healthy dietary lifestyle and, in particular, on an adequate intake of NDP and its relationship with ease of defaecation.

### Mechanistic effects of diet

In the subsequent discussion related to disease, various perceived mechanisms for beneficial actions of diet will be dis-

cussed. In brief, these include: (i) components of a healthy luminal environment such as high butyrate levels and lowered pH; (ii) a predominance of 'healthy' over 'unhealthy' bacteria; (iii) rapid intestinal transit and high faecal bulk; (iv) a non-leaky epithelial barrier; (v) adsorption of carcinogens by fibre; (vi) low bile salt concentrations and generation of toxic bile salts or protein derivatives; and (vii) provision of certain bioactive substances. Intuitively, one would expect that if we demonstrated that changes such as these could be achieved by a particular dietary manoeuvre, then it would be justified. Unfortunately, the link of changed mechanism to disease outcome is weak in many instances and is not a surrogate for demonstrating benefit in real terms; that is, a clear effect on dysfunction or disease.

### Diet and risk of disease

Certain foods or diet-related items are associated with risk for colorectal disease and so are seen to be important to either avoid or encourage so as to prevent disease. Table 1 lists the colorectal disorders and diseases *thought* to be associated with dietary lifestyle. The evidence relating to these will be discussed in detail.

### Risk of diverticular disease

Concerning diverticular disease, it is well established to be more prevalent in developed countries where fibre intake is low and vice versa.<sup>11</sup> Such studies have also demonstrated an

**Table 1.** Colorectal disorders and diseases thought to be causally related to 'diet'

Constipation and defaecation difficulties
Anorectal disorders such as fissures and haemorrhoids
Irritable bowel syndrome
Certain food-induced diarrhoeal disorders and 'allergies'
Inflammatory bowel disease
Traveller's diarrhoea
Diverticular disease
Colorectal adenomas and cancer

**Table 2.** Dietary factors and colorectal cancer. Strength of evidence and nature of effect (derived with modification from the extensive report from the World Cancer Research Fund<sup>2</sup>)

Food	Impact on risk	Strength of evidence
Vegetables	Decreased	Convincing
NDP/fibre	Decreased	Probable
Starch	Decreased	Possible
Carotenoids	Decreased	Possible
Calcium	Decreased	Possible
Fish	Decreased	Possible
Vitamins	Decreased	Insufficient
Red meat (> 80–140 g/day)	Increased	Probable
Alcohol (> 30 g/day)	Increased	Probable
Obesity	Increased	Probable
Heavily cooked meat	Increased	Probable
Refined sugar	Increased	Possible
Total fat	Increased	Possible
Eggs	Increased	Possible
Iron	Increased	Insufficient

NDP, non-digestible polysaccharides.

increased incidence in immigrants to developed countries indicating that environment rather than inherited factors are important. The responsible mechanism has never been defined but diverticula occur where blood vessels penetrate the muscularis. This is consistent with high luminal pressures causing mucosal pouching at these weak points. The unproven assumption is that low faecal bulk and/or hard stools results in higher than normal intraluminal pressures. Nonetheless, it is reasonable to propose that a diet containing adequate NDP will protect against diverticular disease. In consequence, it should indirectly protect against complications such as inflammation and abscess formation.

### **Risk of colorectal cancer**

Many publications address the many studies relating to risk for CRC.<sup>1,2</sup> Table 2 is derived from the extensive report from the World Cancer Research Fund (WCRF)<sup>2</sup> with some modifications based on other publications.<sup>12</sup> While the WCRF report is controversial in some areas and not necessarily presented with a perspective of relevance to at-risk populations following the Western dietary lifestyle, it addresses the quality of evidence as well as the effect of different foods and food classes. As described in Table 2, some of the dietary conditions related to protection indicate that the adoption of a diet that achieves a healthy bowel as defined earlier is likely to be protective against CRC. A few specific dietary issues need to be addressed in more detail.

### **Vegetables**

Numerous epidemiological surveys link fresh vegetables to protection against CRC.<sup>2</sup> These foods contain a broad range of bioactive substances often called phytochemicals and their benefits go far beyond their antioxidant components. Table 3 lists some of these and their potential modes of action. Some have exciting potential as chemopreventive agents.

### **Meat**

The role of meat has been studied by many and the conclusions reached are controversial and conflicting.<sup>2,13,14</sup> High protein intake is also associated with increased risk of CRC.<sup>15</sup> This not exclusively of red meat origin but it would be the major contributor. It is thought that in the human colon, undigested protein is fermented by resident microflora

to yield toxic and/or mutagenic compounds.<sup>14</sup> Red meat in excess of 80–140 g per day does seem to be associated with increased risk<sup>2,13</sup> but some argue that the evidence is not convincing.<sup>14</sup> Also, it needs to be put into context of the Australian diet. The average daily consumption of red meat by Australian men is now under 100 g and the trend is towards even lower consumption. As red meat is a valuable source of dietary iron, in itself controversial in relation to CRC, encouraging an even lower red meat intake needs care and must be accompanied by meaningful descriptions of what constitutes a daily average of around 100 g. It appears that half of Australian men are already meeting that goal.

Meats, and not just red meats, are a special problem when cooked at high temperature in direct flame. This generates certain procarcinogens (heterocyclic amines) which, when metabolized in susceptible individuals,<sup>16</sup> increase the risk for CRC by up to six-fold.<sup>17</sup> It is interesting that this situation provides a direct model of inherited factors determining host interaction with the environment.

### **Fibre and risk**

The role of fibre is controversial especially in view of the recently published nurses' study in the United States.<sup>18</sup> Furthermore, the WCRF report fails to concede a strong role for fibre despite other bodies being more positive.<sup>19</sup>

Studies *in vitro* and in animal models show unequivocally that fermentative production of butyrate directly influences key aspects of cell biology such as differentiation, proliferation and apoptosis in such a way as to be protective.<sup>8,20</sup> There is an inverse relationship between faecal bulk, which is determined by NDP intake, and cancer risk.<sup>21</sup> Most studies show dietary fibre to be protective with a benefit of about a 40–50% reduction; this is confirmed by the meta-analyses of Trock and co-workers where the odds ratio for CRC was 0.57.<sup>22</sup> Variations between studies almost certainly relate to complex interactions between fibre and the rest of the diet, difficulties with methodologies in epidemiological studies, and the different types and sources of NDP. The Australian Polyp Prevention Project<sup>23</sup> showed a beneficial effect of fibre in combination with a diet low in fat but only a trend when fibre was considered in isolation. Animal studies show that insoluble fibre is more protective than soluble fibre.<sup>24</sup> While cereal fibres are excellent bulking agents in humans, the evidence based on case-control studies is relatively weak.<sup>22</sup> However, the substantial methodological problems present when examining the value of different types of fibre is difficult to overcome.<sup>25</sup> One study using careful methods that are related to fibre type and source has shown a clear benefit for insoluble fibre above the value of other types.<sup>26</sup> Given that the United States nurses study<sup>18</sup> was based on a cohort first studied when methodologies and dietary tools were only in their formative stages and that the range of fibre consumption was only 10–25 g per day when recommended fibre intakes are close to 40 g, this particular study should not be cited as negating the positive value for fibre that is observed in many other studies.

The value of resistant starch (RS) remains relatively unexplored due to major methodological difficulties in epidemiological and case-control studies. Certainly, the luminal environment that is generated seems likely to be protective with increased laxation, lowered pH and increased

**Table 3.** Bioactive compounds in plant-derived foods (phytochemicals) and their potential modes of action

Chemical class	Food source	Biological action
Allium	Garlic	Induces enzymes, detoxification
Dithiolthiones	Cruciferous vegetables	Induce enzymes, detoxification
Isothiocyanates	Spices, vegetables	Induce enzymes that detoxify
D-Limonen	Citrus	Induce glutathione transferase
Phytoestrogens	Cereals, pulses	Alter steroid hormone metabolism
Flavonoids	Tea, fruit, vegetables	Non-toxic antioxidants
Polyphenols	Tea, fruit, vegetables	Detoxification, inhibit N-nitrosation

SCFA concentrations.<sup>10</sup> Total starch intake correlates inversely with risk,<sup>15</sup> hence implying a protective role for RS as it correlates in turn with total starch intake. Starch and RS intakes in Australia are low.<sup>15</sup> It is reasonable to be optimistic about the value of RS but much more work is required.

#### *Fat and energy intake*

Low fat intake has consistently been associated with protection in population studies<sup>2,15</sup> but different fats have varying effects. Animal fats seem the worst. Monounsaturated fats such as olive oil are probably neutral to protective while fish oils<sup>27</sup> seem likely to be protective. There are likely to be multiple mechanistic explanations for these effects, which involve regulation of bile acids and modulation of mucosal eicosanoids. At present it is impossible to know which are most important. However, obesity and inactivity have strong effects on risk of CRC and tend to override the benefits of an otherwise seemingly healthy diet.<sup>2</sup>

#### **Modification of risk and prevention**

Association does not imply causation. Dietary interactions, uncertainty about time frames and complexity in food composition and preparation mean that adherence to or avoidance of a particular lifestyle will not necessarily prevent the disease or be an effective treatment for it. Proof that adoption of a healthy dietary lifestyle will prevent dysfunction or disease is sadly lacking except in the following cases.

Adequate dietary NDP minimize the chance of constipation developing<sup>28</sup> and the future risk of diverticular disease.<sup>29</sup> The latter study showed that the relative risk for diverticular disease in the highest quintile of fibre intake (31 g per day) was 0.55 compared to the lowest quintile (14 g per day).<sup>29</sup>

The symptomatology of certain types of irritable bowel syndrome (IBS) is less likely to recur when patients avoid those particular foods that trigger their symptoms. This is especially so for malabsorbed foods such as lactose.

Despite the huge volume of literature concerning CRC, there are very few interventional studies. One study has shown that a low-fat, high-insoluble fibre diet will prevent the recurrence of larger colorectal adenomas in 2–4 years.<sup>23</sup> It is especially interesting that processed wheat fibre reduces colorectal adenoma formation in the retained rectum of patients with familial adenomatous polyposis within 12 months of starting.<sup>23</sup> While not the typical situation, the fact that a dietary manipulation can exert its effect within 12 months suggests that adoption of a healthy lifestyle is never too late.

A few carefully studied probiotic agents reduce the chance of antibiotic-associated diarrhoea from occurring.<sup>30</sup>

Otherwise, proof is lacking in many areas but it must be acknowledged that these studies can be difficult and absence of proof does not mean lack of value.

#### **Diet as a therapy**

In the context of treating existing disorders, diet has a clear benefit in a few defined settings, but there are many misconceptions and uncertainties.

#### ***Constipation and bowel habit***

Non-digestible polysaccharides clearly improve stool bulk,<sup>28</sup> increase frequency of defaecation, soften stools and accel-

erate transit.<sup>31</sup> These effects are less efficient in women compared to men<sup>32</sup> and women may need to consume more fibre than men to achieve the same effect. It appears that 20–30 g per day of NDP can be adequate<sup>28</sup> but it depends on the food source. Insoluble fibre is more efficient than processed fibre, soluble fibre or RS, perhaps because insoluble fibre is less broken down by fermentation. The laxation effect of RS correlates closely with the amount consumed<sup>10</sup> but it is not as efficient as insoluble fibre. Soluble fibre and RS are quite rapidly fermented in the proximal colon, which might explain why insoluble fibre has more effect. For each gram of fibre consumed, stool mass increases by 2.3–2.9 g per day<sup>33</sup> due to the combined effects of intact fibre and increased bacterial mass and faecal water.

Recommended fibre intake varies according to the recommending authority and method of analysis.<sup>34</sup> It is important to see the need for NDP in the broader context of the balanced diet. A range of fibre sources should be included with the goal of achieving a functional outcome. In other words, the person must experiment for themselves based on their own experience. Limited evidence indicates that fluid intake, physical activity and psychological status limit the benefit of fibre.<sup>35</sup>

Severe constipation often does not respond to fibre alone even when fibre is given in sufficient amounts to cause side-effects such as bloating, wind and colic. The common direction to increase fluid intake and activity is also of limited value in these people.

#### ***Irritable bowel syndrome***

Irritable bowel syndrome is a complex disorder. The symptoms of disordered bowel function and abdominal pain are the final pathway of a number of conditions with a range of management options. Colorectal pathology must be excluded but, in the typical case, this might involve simple investigation only.

Diarrhoea-predominant IBS occurs in a number of settings. Some are clearly due to malabsorbed foodstuffs such as high-fructose fruits and drinks, sorbitol-containing fruits and sweets and lactose in those who are milk intolerant. Others are due to unrecognized stress and other psychopathologies such as depression; in such cases the role of diet is limited. In other situations, none of these can be identified in IBS.

Studies of fibre supplementation in IBS have been complicated by different definitions of IBS, use of different fibres and poor study design. While insoluble fibre such as wheat bran shortens transit time<sup>36</sup> and wheat bran as well as ispaghula and psyllium improve the symptoms of constipation,<sup>37</sup> none of the other symptoms is helped. There is an anecdotal view that certain refined or synthetic fibres cause

**Table 4.** Foods which commonly trigger the symptoms of irritable bowel syndrome (IBS) in those with diarrhoea-predominant IBS

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Diary products
Fatty and oily foods (all types)
Wheat products with or without fibre and independent of coeliac disease
Sorbitol-containing foods, including sweets and gum
Fructose-rich fruits, sweets and drinks

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less wind but confirming data are lacking. A significant proportion of those with diarrhoea-predominant IBS get worse with increased fibre<sup>38</sup> and many physicians find reducing fibre is beneficial.

Nonetheless, diet can play a useful role if only to minimize triggering of the more bothersome symptoms. Many patients notice that any food, even a glass of water, triggers their symptoms; these patients can end up on the simplest and plainest of diets, sometimes quite deficient in energy or nutrients. The search for true food allergies as a cause of IBS has been unsuccessful.<sup>39</sup> This does not mean that specific foods do not cause symptoms but that many people misunderstand allergy and that the cause is food intolerance that does not involve the immune mechanisms that are typical of allergy. Some patients find that certain foods can reproducibly trigger symptoms and one investigator<sup>40</sup> found that almost half of patients suffering IBS can identify a food that triggers symptoms; when removed from the diet or taken in small amounts there is a worthwhile reduction in symptoms. Such foods, which are summarized in Table 4, are sometimes referred to as 'trigger foods' and include dairy products, certain fatty and oily foods, wheat products with or without fibre and independent of coeliac disease, sorbitol-containing foods, and fructose-rich fruits and drinks.<sup>41</sup> One study found that 91 of 189 patients improved with dietary exclusions.<sup>40</sup> Dairy products were responsible in 41% of responders and grain products in 39%. Some have interpreted this as being due to salicylates in foods but this observation has not been rigorously explored.

The possibility of specific food intolerances is real and worth exploring. Most exclusion diets that work exclude these triggering foods, whether by design or accident. Unfortunately, some people go to extremes in the control of their symptoms and maintain a poorly balanced diet that can be deficient in energy sources. The mechanisms for specific food intolerances remain largely unknown with the exception of the malabsorbed carbohydrates (lactose, fructose and sorbitol).

The idea that yeast causes IBS is difficult to comprehend. No yeast survives cooking. The confusion between food yeast and candida (which only causes diarrhoea in the immunosuppressed) is pseudo-science.

To summarize, fibre benefits the constipation phase in IBS and exclusion of food triggers is of some benefit in those with clearly defined food intolerance. Food allergy as a cause of IBS is rare. Exclusion diets can be dangerous and must be used with great care and planning.

### **Anorectal disorders**

Anecdotally, patients often indicate that symptoms of anal fissure or haemorrhoids, including bleeding, settle when they take more fibre and soften their stools. While confirmatory studies are few,<sup>42,43</sup> there seems little reason to doubt the therapeutic benefit. A high fibre diet certainly reduces recurrence rate after haemorrhoidectomy.<sup>44</sup>

### **Diverticular disease**

Short of complications such as inflammation and abscess, the symptoms of diverticular disease are the same as those of IBS. The treatment is also that of IBS, but these people can be particularly sensitive to fibre and often have to reduce

fibre intake to the point of being constipated to minimize the other symptoms such as colic and wind. Nonetheless, a few good studies have shown fibre to be of benefit.<sup>34,45</sup> In these patients, a trial of insoluble fibre that is first followed by soluble fibre perhaps with RS is worthwhile; those who benefit seem to prefer the soluble fibres.

### **Inflammatory bowel disease and colitis**

Nutritional deficit can complicate Crohn's disease and obviously needs to be corrected. Nutritional therapy alone will not significantly improve the inflammation of Crohn's disease or colitis, unless used in combination with appropriate medical therapy. Dietary fibre itself has no obvious benefit in Crohn's disease or ulcerative colitis.<sup>46,47</sup> The studies found that those patients with colitis tended to be quite sensitive to the side-effects of fibre, especially urgency and frequent stools. Direct instillation of SCFA may improve diversion colitis but there is no proven way to generate these in a practical way as fibre is an irritant. Perhaps a trial of RS to generate SCFA would be worthwhile as there are no irritating particles. Oils that contain omega-3 may have a worthwhile anti-inflammatory action in inflammatory bowel disease. A study by Belluzzi *et al.* showed that fish oil enhanced the remission rates in Crohn's disease,<sup>48</sup> possibly via its effects on eicosanoid production.

### **Colorectal adenomas and cancer**

The huge body of evidence linking dietary lifestyle with CRC relates to protection against the risk of developing CRC. There is no place for the dietary management of patients with cancer, although the fact that they have at least a 10% chance of developing a second cancer ought to motivate the adoption of a healthier lifestyle.

### **Probiotics and diarrhoea**

Limited studies with just a few probiotics indicate that those which are capable of colonizing the human colon and changing the balance of bacterial species do have the capacity to be protective against antibiotic-associated diarrhoea.<sup>30</sup> Ideally, probiotics should be started at the same time as the antibiotics with the aim of preventing diarrhoea. Evidence for the general positive health benefits of *Lactobacilli* applies to only a few strains used for commercial applications. *Lactobacillus GG*, a variant of *L. casei* ssp rhamnosus, has been studied extensively in adults and children. *Lactobacillus reuteri* and *Saccharomyces boulardii*, also seem to be promising agents for improving the course of acute diarrhoea in children when used therapeutically.

### **Traveller's diarrhoea**

Foods are often responsible for this condition but it is either due to an exacerbation of IBS or pathogens in the food.

### **Secretory diarrhoea**

A recent publication has shown that administration of RS as Hi-maize<sup>®</sup> (Starch Australasia Ltd, Sydney, NSW, Australia) to patients with severe diarrhoea due to cholera results in a shortened duration of diarrhoea.<sup>9</sup> The mechanism is thought to be increased generation of SCFA in the colon with subsequent stimulation of colonic reabsorption of fluid and correction of dehydration.

## Conclusion

Bowel health for most people equates to ease of defaecation, but it goes beyond this to encompass a well-balanced diet that contains nutrients seen to be preventive against diseases such as CRC and diverticulosis, and avoids nutrients that carry a risk in the context of an unbalanced diet. While the healthy luminal environment can be defined in biological terms it equates with mechanistic issues seen to be important in reducing one's risk for colorectal disease. It also requires a whole change in dietary lifestyle. Whether we will ever be able to accurately define a diet that is proven beyond doubt to be protective against CRC remains to be seen. In the meantime, a balanced approach to diet with some extra emphasis on insoluble fibre intake and avoidance of heavily burnt meat is prudent for preventing colorectal disease.

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