Fiber and colorectal diseases: Separating fact from fiction

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Abstract
Whilst fruits and vegetables are an essential part of our dietary intake, the role of fiber in the prevention of colorectal diseases remains controversial. The main feature of a high-fiber diet is its poor digestibility. Soluble fiber like pectins, guar and ispaghula produce viscous solutions in the gastrointestinal tract delaying small bowel absorption and transit. Insoluble fiber, on the other hand, pass largely unaltered through the gut. The more fiber is ingested, the more stools will have to be passed. Fermentation in the intestines results in build up of large amounts of gases in the colon. This article reviews the physiology of ingestion of fiber and defecation. It also looks into the impact of dietary fiber on various colorectal diseases. A strong case cannot be made for a protective effect of dietary fiber against colorectal polyp or cancer. Neither has fiber been found to be useful in the prevention of acute constipation and irritable bowel syndrome. It is also not useful in the treatment of perianal conditions. The fiber deficit - diverticulosis theory should also be challenged. The authors urge clinicians to keep an open mind about fiber. One must be aware of the truths and myths about fiber before recommending it.

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Key words: Fiber; Physiology; Colorectal cancer; Constipation; Irritable bowel syndrome; Diverticulosis; Hemorrhoids

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INTRODUCTION
Most patients exclaim that they have not been taking adequate fiber when diagnosed with a colorectal disease. Over the last 40 years, the benefits of high fiber diets have been drummed into the minds of doctors and patients alike. Whilst fruits and vegetables are an essential part of our dietary intake, the role of fiber in the prevention of colorectal diseases remains controversial. Burkitt[1] first postulated the protective effects of fiber from his observation of the rarity of haemorrhoids, diverticulosis of the colon and colorectal cancer in an African diet rich in fiber. The next three decades marked the tremendous marketing of high fiber products and the development of fiber supplements. There is hardly any modern patient with constipation who is not on additional fiber supplementation. Furthermore, it is rare indeed to find a patient newly diagnosed with colorectal cancer who will not blame himself for not having taken more fiber. Recent studies have, however, not supported these benefits of fiber. Excessive fiber intake may in fact be harmful[3]. This paper reviews the current knowledge about fiber and colorectal diseases.

PHYSIOLOGY OF DIETARY FIBER
INGESTION
The term “dietary fiber” in current usage encompasses a broad range of substances. The word is not precise but its public appeal has made it the darling buzzword of health promotion. The main feature of a high-fiber diet is its poor digestibility[3]. A distinction is often made between water soluble substances such as pectins, guar, ispaghula and some hemicelluloses, and insoluble fibers such as cellulose, hemicelluloses and lignins (non polysaccharide plant cell wall components). These substances differ in physico-chemical properties (Table 1). Non-starch polysaccharides quantitatively make up most of the fiber in our diet.

Soluble fiber like pectins, guar and ispaghula produce viscous solutions in the gastrointestinal tract delaying small bowel absorption. While this may reduce cholesterol absorption, they may also inhibit pancreatic enzyme activity and protein digestion leading to an antinutritive effect[8]. These substances not only delay delivery of chyme to the site of absorption but also impair the process of hydrolysis by inhibiting pancreatic enzyme activity. The production of sticky lumps in the intestine leads to bloating and flatulence, which actually delays colonic transit.

Insoluble fiber, on the other hand, pass largely unaltered through the gut. Insoluble fiber is variably fermented in the colon. However, up to 80% are excreted in normal subjects[9]. It is thus obvious that the more fiber is ingested, the more stools will have to be passed.
Pure insoluble fiber is the ultimate junk food. It is neither digestible nor absorbable and therefore devoid of nutrition. People who ingest fiber are ingesting them to make feces only. Strange as it sounds, studies have been performed just to show stool weight increases as fiber intake increases[3]. It is already obvious by definition that this must be the case. In normal individuals, mean colon transit time is reduced when the stool weight is increased[6]. This is a normal physiological response necessary to clear a colon packed with faeces. In constipated individuals, the mean colonic time has not been found to be reduced as much as in normal individuals[7]. This reflects an attenuated response to a colon packed with faeces in individuals constipated for whatever reasons.

Resistant starch is found in oats and cornflakes. They are resistant to α-amylase digestion. Modest (10 g/d) increases in resistant starch intake do not increase stool output suggesting that it may be completely fermented by colonic bacteria[9]. However, fermentation results in an increased production of colonic gas, leading to bloatedness and a distended abdomen.

One common but erroneous belief is that the moisture content of stool is increased when fiber intake is increased. The moisture content actually remains at 70% to 75% and does not change when more fiber is consumed. For most fiber substances, increase in quantity does not result in a more effective holding of water in the gut lumen[3].

A high fiber diet has also been shown to be associated with excessively long colons and a higher incidence of megacolon and volvulus[7], suggesting a negative effect of excessive fiber on colonic transit.

Fiber is fermented rapidly and may lead to a massive surge in microbial activity in the colon. Hydrogen, methane and carbon dioxide are then produced, causing cramps, bloatedness and distension[8].

Indeed, the incidence of diverticulosis and complications of diverticular disease have been increasing in the West despite increase in dietary fiber intake[9]. This is probably related to the massive gaseous build up associated with a high dietary fiber intake.

**FIBER AND COLORECTAL CANCER**

Since the time that Burkitt suggested an inverse relationship between fiber and colorectal cancer based on epidemiological data[1], a number of international correlation studies have been published. Initial studies in the seventies and eighties seemed to support this theory[10-13]. However, with adjustments for the confounding factors of associated meat and fat intake, the correlation was weakened[10,12-14]. Liu et al[12] studied data from 20 industrialised countries. While they found that after controlling for fiber, there was a highly significant correlation between cholesterol intake and colon cancer, the converse was not true. When cholesterol was controlled, there was no correlation between fiber and colon cancer. McKeown-Eyssen et al[14] also had similar findings in a study of 38 countries after adjusting for animal fat, that increased fiber did not correlate with colon cancer.

There are also numerous case-control studies that have been published on this topic[15-20]. The results are presented in Table 2. Two systematic reviews of older case-control studies published only managed to suggest a protective effect of fiber. Trock et al[21] analysed 23 case-control studies. Of these 23 studies, 15 demonstrated lower

### Table 1 Classification of fiber

| Material   | Chemical structure       | Source                               | Hydrolysis | Fermentation in colon |
|------------|--------------------------|--------------------------------------|------------|-----------------------|
| Soluble    |                          |                                      |            |                       |
| Pectins    | Nonstarch polysaccharide | Plant cell wall                      | Yes        | Rapid                 |
| Guar       | Nonstarch polysaccharide | Beans                                | Yes        | Rapid                 |
| Ispaghula  | Acidic arabinoxyan       | Husks of seeds of platago ovata      | Yes        | Rapid                 |
| Hemicellulose | Nonstarch polysaccharide | Component of plants                 | Yes        | Variable              |
| Insoluble  |                          |                                      |            |                       |
| Cellulose  | Nonstarch polysaccharide | Component of plants                  | Minimal    | Variable              |
| Hemicellulose | Nonstarch polysaccharide | Component of plants                  | Partial    | Variable              |
| Lignin     | Nonpolysaccharide cell wall component | Component of plants                  | Nearly none | Minimal              |
| Resistant starch | Starch not digested in small intestine | Polysaccharide                   | Legumes/ Grains | Minimal              |

### Table 2 Case-control studies correlating dietary fiber with colorectal neoplasia

| Reference                  | Country         | Number of cases/controls | Odds ratio of CRC comparing highest to lowest fiber intake groups | Dietary fiber protective |
|----------------------------|-----------------|--------------------------|---------------------------------------------------------------|--------------------------|
| Wakai et al 2006[16]       | Japan           | 507/2535                 | 0.65 (P < 0.05)                                               | Yes (colon)              |
| Levi et al 2001[19]        | Switzerland     | 286/350                  | 0.55 (P < 0.05)                                               | Yes                      |
| Chedarian et al 1997[20]   | Canada          | 402/668                  | 0.50 (P < 0.01)                                               | Yes                      |
| Slattery et al 1997[21]    | United States   | 1903/2410                | 0.70 (95% CI 0.5-1.0)                                         | No                       |
| Little et al 1993[22]      | United Kingdom  | 147/329                  | 0.60 (not significant after adjustment for energy intake)     | No                       |
| Steinmetz et al 1993[23]   | Australia       | 220/438                  | 0.77 (not significant)                                        | No                       |

CRC: Colorectal cancer.
Table 3  Longitudinal studies correlating dietary fiber with colorectal neoplasia

| Reference               | Location | Cohort                | Follow-up period (yr) | Odds ratio of CRC comparing highest to lowest fiber intake groups | Dietary fiber protective |
|-------------------------|----------|-----------------------|-----------------------|------------------------------------------------------------------|-------------------------|
| Fuchs et al 1999[26]    | US       | 88 757 (women)        | 16                    | 0.95 (95% CI 0.73-1.25)                                           | No                      |
| Mai et al 2005[27]      | US       | 45 491 (women)        | 8.5                   | 0.94 (95% CI 0.71-1.23)                                           | No                      |
| Lin et al 2009[28]      | US       | 36 976 (women)        | 10                    | 0.79 (95% CI 0.48-1.17)                                           | No                      |
| Otani et al 2006[29]    | Japan    | 86 412                | 10                    | Hazard ratio of lowest fiber intake group compared to highest group: 2.3 (95% CI 1.0-5.2) | No                      |
| Shin et al 2006[30]     | China    | 73 314 (women)        | 5.7                   | 1.1 (95% CI 0.6-1.8)                                              | No                      |

Table 4  Intervention studies correlating dietary fiber with colorectal neoplasia

| Reference               | Setting              | n          | Intervention       | End point                       | Odds ratio of recurrence in intervention group | Dietary fiber protective |
|-------------------------|----------------------|------------|--------------------|---------------------------------|-----------------------------------------------|-------------------------|
| Alberts et al 2000[31]  | Postpolypectomy      | 2079       | Counselling        | Recurrent adenoma at 4 yr       | 0.88 (95% CI 0.70-1.11)                         | No                      |
| Schatzkin et al 2000[32] | Postpolypectomy      | 1429       | Fiber supplement   | Recurrent adenoma at 36 mo      | 1.00 (95% CI 0.90-1.2)                          | No                      |
| Ishikawa et al 2005[33] | Postpolypectomy      | 398        | Fiber supplement   | Recurrent adenoma at 4 yr       | 1.31 (95% CI 0.87-1.98)                         | No                      |
| Jacobs et al 2006[34]   | Postpolypectomy      | 3209       | Fiber supplement   | Recurrent adenoma               | 0.91 (95% CI 0.78-1.06)                         | No                      |

Pooled from 2 studies

The presence of colorectal cancer with higher fiber intake, 6 showed arguable results after adjustment while only 2 did not show a protective effect of fiber. They calculated a combined odds ratio of 0.57 (95% CI 0.50-0.64) of colorectal cancer for the subgroup with the highest fiber or vegetable intake compared with the subgroup with the lowest intake. Howe et al[22] subsequently pooled data from 13 case-control studies and arrived at a relative risk of 0.53 (95% CI 0.47-0.61) for development of colon cancer in patients with the highest fiber intake. This study was later criticised for methodological flaw[23], not taking into account the quality of the studies included in the analysis. A re-analysis of the higher quality studies revealed no protective effect of fiber. Hence, one has to be careful of case-control studies looking at fiber and colorectal cancer. The problem of recall bias and confounding factors often weaken these studies and evidence offered by these studies is likely to be inconclusive.

Interestingly, more recently, better designed longitudinal studies have not supported a correlation between fiber and prevention of colorectal cancer. Fuchs et al[35] studied 88 757 nurses over 16 years and found no effect of dietary fiber on colorectal cancer. Another study of 45 491 women in the Breast Cancer Detection Demonstration Project[25] over 8.5 years also found little evidence that dietary fiber intake lowers the risk of colorectal cancer. Other studies in China, Japan and the United States also failed to show a protective effect of fiber from colorectal cancer[26-28]. These studies are shown in Table 3. Park et al[29] pooled data from 13 prospective cohort studies and analysed data of 725 628 men and women and concluded, after accounting for other dietary risk factors that high dietary fiber intake was not associated with a reduced risk of colorectal cancer.

Several large interventional studies have also not been able to demonstrate any effect of fiber supplements on decreasing colorectal adenoma recurrence[36,31]. Alberts et al[31] analysed 1303 postpolypectomy patients aged 40 to 80 years. They used dietary supplementation in the form of wheat bran. They estimated a mean total fiber intake of 27.5 g/d in the intervention group compared to 18.1 g/d in the control group. They found no significant difference in the recurrent adenoma rate between the 2 groups at 36 mo. The other study by Schatzkin[32] analysed 1905 patients over the age of 35 years. Intervention took the form of dietary counselling. The recurrence rate of adenomas at 4 years was similar in both the intervention and control groups. A Cochrane review also concluded that there was no evidence based on randomized trials that increased fiber will reduce the incidence or recurrence of adenomatous polyps over a 2 to 4 year period[32]. Two more studies subsequently also failed to demonstrate a protective effect of intervening with fiber supplement on adenoma recurrence[33,34]. These studies are shown in Table 4.

In summary, a strong recommendation cannot be made for a protective effect of dietary fiber against colorectal polyp or cancer. Despite a lack of evidence however, current recommendations are still to increase dietary fiber. In the latest position statement of the American Dietetic Association[3], increasing dietary fiber is still promoted to protect against colon cancer despite stating that there is no proof of efficacy in this regard.

FIBER AND THE PHYSIOLOGY OF DEFECATION

The first question that needs to be asked must be whether one stool movement per day is the desired frequency for everyone? There is no evidence to support the theory that a long but normal residence of stools in the colon will lead to physical diseases[19]. Secondly, if an individual has a single bowel movement in a week but is able to evacuate all the faecal material easily, does this constitute a pathological bowel habit?

Infants on breast feeding are known to be able to go...
for long periods of time without any bowel movement. This is because the breast milk is thoroughly absorbed with minimal residue. Therefore, if an individual has a low residue diet and therefore less frequent bowel movements simply because there is less faecal material to evacuate, this is not pathological.

On the other hand, if an individual ingests a large amount of high fiber material, a large proportion of that dietary intake will be unabsorbed and subsequent faecal material will be very bulky. Fiber makes faeces bulkier and heavier. In other words, the more fiber one ingests, the more faeces one will have to evacuate. By the mass effect of the formation of more faecal material, there will be a resultant increase in frequency of evacuation. By increasing fiber intake, stool frequency and faecal weight will be correspondingly increased. However, this is a classic case of rubbish in, rubbish out only.

The formation of large amounts of faecal material can actually have a detrimental effect on the patient. Faeces that is bulky and hard is more difficult to evacuate in a patient with a pre-existing evacuatory problem. Increasing faecal loading by increasing the fiber intake to increase stool frequency cannot be logical if one is trying to decrease colonic load as a motive.

**Fiber in Constipation and Irritable Bowel Syndrome**

Most physicians, including gastroenterologists and colorectal surgeons are quick to prescribe fiber supplements for constipation, citing inadequate fiber as the cause for constipation. Most patients complaining of constipation are likely to receive additional fiber from their doctors. However, is there evidence that fiber supplementation actually improves constipation?

A recent study from Brazil found that a low dietary fiber dosage was not associated with constipation[35]. Several studies that looked at dietary fiber intake by people with chronic constipation did not find any difference in fiber intake compared to controls[36-40]. Muller-Lissner emphasized that a diet poor in fiber should not be assumed to be the cause of chronic constipation. In contrast, they found that many patients with severe constipation deteriorated when dietary fiber intake was increased[41]. This is in line with our own experience. A recent prospective randomized crossover trial comparing ispaghula husk with lactulose in the treatment of idiopathic chronic constipation performed in Singapore found that compared to fiber, lactulose resulted in a significantly higher mean bowel frequency and less bulky stool consistency. The adverse effects were similar. More patients preferred the use of lactulose (61.5%) over fiber (35.9%) to ease constipation[42]. Most patients who are constipated are already on large amounts of dietary fiber, being influenced by the media and doctors in promoting high fiber diets indiscriminately.

Voderholzer et al[43] studied 149 patients with chronic constipation in Germany. The patients were treated with Plantago ovata seeds, 15-30 g/d for a period of 6 wk. They found that 80% of patients with slow transit and 63% of patients with a disorder of defaecation did not improve with additional dietary fiber.

There are also a number of reviews on the role of fiber in the treatment of irritable bowel syndrome. A review of 17 studies mostly using either ispaghula or wheat bran, found that fiber only conveyed marginal benefits on global irritable bowel syndrome symptoms and constipation, emphasizing that insoluble fiber may even worsen the clinical outcome[44]. A meta-analysis of the use of bulking agents in irritable bowel syndrome was performed in Switzerland[45]. After exclusion of low-quality trials, the odds ratio of symptomatic improvement with bulking agents did not reach statistical significance. A Cochrane review also found that there was no clear evidence of benefit for bulking agents in irritable bowel syndrome[46]. A more recent randomized-controlled trial in the UK also failed to show benefits of fiber over placebo[47].

Another noteworthy point is that although stool frequency may be increased by the mass effect of fiber packing in the colon in normal individuals, this is not so in individuals who are chronically constipated. A meta analysis found that bran did not reduce transit time as expected in patients who are constipated[48]. A subsequent population based study of older people also found that dietary fiber did not reduce total transit time[49]. There have been recent demonstrations that patients with colonic inertia actually have decreased volumes or numbers of interstitial cells of Cajal and enteric neurons[50-52]. In these patients, it will not make sense to increase their faecal load. An increase in the fiber intake in these patients will not result in a decrease in stool transit time. There is, in fact, a deleterious effect of increasing faecal load without effectively increasing evacuation!

Fiber is not helpful in patients who have defaecation disorders. A recent study in France suggests that this condition is more frequent than previously thought[53]. In patients with pelvic floor dyssynergia, the main problem is the paradoxical contraction or failure to relax the pelvic floor during attempts to defaecate. Having large amounts of bulky stool in the rectum is unlikely to improve defaecation in these patients. Biofeedback is superior in this situation[54]. Similarly, it will not serve any benefit for patients with large rectoceles to have large bulky faeces, which tend to aggravate the situation. In summary, there is little physiological basis for increasing fiber intake and thus bulkiness of the stool in constipating and defaecatory disorders.

**Hemorrhoids and Fissures**

The need to evacuate large bulky stools frequently may also give rise to various ano-rectal disorders including haemorrhoids and anal fissures. The most important factor in the pathogenesis of haemorrhoids is repeated straining when passing stools[55]. This results in the disruption of the suspensory ligaments of Park at the anal cushions leading...
Soluble fiber supplements should also be considered in the treatment of anal incontinence where there is leakage of liquid faeces, while symptoms are improved by making the stools harder and more solid, and therefore more constipated.

When one recommends a high fiber diet or prescribes a fiber supplement, one must thus be very certain of what the treatment goals are.

## CONCLUSION

Whilst it is not the intention of the authors to totally discourage fiber in the diet and the use of fiber supplements, there does not seem to be much use for fiber in colorectal diseases. We, however, want to emphasize that what we have all been made to believe about fiber needs a second look. We often choose to believe a lie, as a lie repeated often enough by enough people becomes accepted as the truth. We urge clinicians to keep an open mind. While there are some benefits of a diet high in natural fiber, one must know the exact indications before recommending such a diet. Myths about fiber must be debunked and truth installed.

## REFERENCES

1. Burkitt DP. Epidemiology of cancer of the colon and rectum. Cancer 1971; 28: 3-13
2. Chuwa EW, Seow-Choen F. Dietary fibre. Br J Surg 2006; 93: 3-4
3. Marlett JA, McBurney MI, Slavin JL. Position of the American
Dietetic Association: health implications of dietary fiber. *J Am Diet Assoc* 2002; 102: 993-1000

4 Spiller RC. Pharmacology of dietary fibre. *Pharmaceut Ther* 1994; 62: 407-427

5 Muller-Lissner SA. Effect of wheat bran on weight of stool and gastrointestinal transit time: a meta-analysis. *Br Med J (Clin Res Ed)* 1988; 296: 615-617

6 Eastwood MA, Robertson JA, Brydon WG, MacDonald D. Measurement of water-holding properties of fibre and their faecal bulking ability in man. *Br J Nutr* 1983; 50: 539-547

7 Madiba TE, Thomson SR. The management of sigmoid volvulus. *J Coll Surg Edinb* 2000; 45: 74-80

8 Goodlad RA. Dietary fibre and the risk of colorectal cancer. *Gut* 2001; 48: 587-589

9 Kang JY, Hoare J, Tinto A, Subramanian S, Ellis C, Majeed A, Melville D, Maxwell JD. Diverticular disease of the colon—on the rise: a study of hospital admissions in England between 1989/1990 and 1999/2000. *Aliment Pharmacol Ther* 2003; 17: 1189-1195

10 Sengupta S, Tjandra JJ, Gibson PR. Dietary fiber and colorectal neoplasia. *Dis Col Rectum* 2001; 44: 1016-1033

11 McKeown-Eyssen GE. Bright-See E. Dietary factors in colon cancer: international relationships. *Nutr Cancer* 1984; 6: 160-170

12 Liu K, Stamler J, Moss D, Garside D, Persky V, Soltero I. Dietary intake of calcium, fiber, and other micronutrients in relation to colorectal cancer risk: Results from the Shanghai Women's Health Study. *J Int Cancer* 2006; 119: 2938-2942

13 Park Y, Hunter DJ, Spiegelman D, Bergkvist L, Berrino F, van den Brandt PA, Buring JE, Colditz GA, Freudenberg JL, Fuchs CS, Giovannucci E, Goldbohm RA, Graham S, Harnack L, Hartman AM, Jacobs DR Jr, Kato I, Krogh V, Leitzmann MF, McCullough ML, Miller AB, Pietenpol P, Rohan TE, Schatzkin A, Willett WC, Wolk A, Zeleniuch-Jacquotte A, Zhang SM, Smith-Warner SA. Dietary fiber intake and risk of colorectal cancer: a pooled analysis of prospective cohort studies. *JAMA* 2005; 294: 2849-2857

14 Albers DS, Martinez ME, Roe DJ, Guillen-Rodriguez JM, Marshall JR, van Leeuwen JB, Reid ME, Ritenbaugh C, Vargas PA, Bhattacharyya BA, Earnest DL, Sampiriner RE. Lack of effect of a high-fiber cereal supplement on the recurrence of colorectal adenomas. *Phoenix Colon Cancer Prevention Physicians’ Network. N Engl J Med* 2000; 342: 1156-1162

15 Schatzkin A, Lanza E, Corle D, Lance P, Iber F, Caan B, Shike M, Weisfled J, Burt R, Cooper MR, Kikendall JW, Cahill J. Lack of effect of a low-fat, high-fiber diet on the recurrence of colorectal adenomas. *Polyp Prevention Trial Study Group. N Engl J Med* 2000; 342: 1149-1155

16 Asano T, McLeod RS. Dietary fibre for the prevention of colorectal adenomas and carcinoma. *Cochrane Database Syst Rev* 2002: CD003430

17 Ishikawa H, Akedo I, Otani T, Suzuki T, Nakamura T, Takeyama I, Ishiguro S, Miyaoka E, Sobue T, Kakuize T. Randomized trial of dietary fiber and Lactobacillus casei administration for prevention of colorectal tumors. *Int J Cancer* 2005; 116: 762-767

18 Jacobs ET, Lanza E, Alberts DS, Hsu CH, Jiang R, Schatzkin A, Thompson PA, Martinez ME. Fiber, sex, and colorectal adenoma: results of a pooled analysis. *Am J Clin Nutr* 2006; 83: 343-349

19 Muller-Lissner SA, Kamm MA, Scarpignato C, Wald A. Myths and misconceptions about chronic constipation. *Am J Gastroenterol* 2005; 100: 232-242

20 de Carvalho EB, Vitolo MR, Gama CM, Lopez FA, Taddei JA, de Morais MB. Fiber intake, constipation, and overweight among adolescents living in Sao Paulo City. *Nutrition* 2006; 22: 744-749

21 Preston DM, Lennard-Jones JE. Severe chronic constipation of young women: ‘idiopathic slow transit constipation’. *Gut* 1986; 27: 41-48

22 Anderson AS. Dietary factors in the aetiology and treatment of constipation during pregnancy. *Br J Obstet Gynaecol* 1986; 93: 245-249

23 Campbell AJ. Busby WJ, Horwath CC. Factors associated with constipation in a community based sample of people aged 70 years and over. *J Epidemiol Community Health* 1993; 47: 23-26

24 Towers AL, Burgio KL, Locker JL, Merkel IS, Safaean M, Wald A. Constipation in the elderly: influence of dietary, psychological, and physiological factors. *J Am Geriatr Soc* 1994; 42: 203-207

25 Quah HM, Ooi BS, Seow-Choen F, Sng KK, Ho KS. Prospective randomized crossover trial comparing fibre with lactulose in the treatment of idiopathic chronic constipation. *Tech Coloproctol* 2006; 10: 111-114

26 Vorderholzer WA, Schatte W, Muhl idorfer BE, Klauser AG, Birnkr B, Muller-Lissner SA. Clinical response to dietary fiber treatment of chronic constipation. *Am J Gastroenterol* 1997; 92: 95-98

27 Bijkerk CJ, Muris JW, Knotterus JA, Hoes AW, de Wit NJ. Systematic review: the role of different types of fibre in the treatment of irritable bowel syndrome. *Aliment Pharmacol Ther*
Lesbros-Pantoflickova D, Michetti P, Fried M, Beglinger C, Blum AL. Meta-analysis: The treatment of irritable bowel syndrome. *Aliment Pharmacol Ther* 2004; 20: 1253-1269

Quartero AO, Meineche-Schmidt V, Muris J, Rubin G, de Wit N. Bulking agents, antispasmodic and antidepressant medication for the treatment of irritable bowel syndrome. *Cochrane Database Syst Rev* 2005: CD003460

Rees G, Davies J, Thompson R, Parker M, Liepins P. Randomised-controlled trial of a fibre supplement on the symptoms of irritable bowel syndrome. *J R Soc Health* 2005; 125: 30-34

Evans JM, Fleming KC, Talley NJ, Schleck CD, Zinsmeister AR, Melton LJ 3rd. Relation of colonic transit to functional bowel disease in older people: a population-based study. *J Am Geriatr Soc* 1998; 46: 83-87

Bouchoucha M, Devroede G, Faye A, Le Toumelin P, Arhan P, Arsac M. Colonic response to food in constipation. *Int J Colorectal Dis* 2006; 21: 826-833

He CL, Burgart L, Wang L, Pemberton J, Young-Fadok T, Szurszewski J, Farrugia G. Decreased interstitial cell of Cajal volume in patients with slow-transit constipation. *Gastroenterology* 2000; 118: 14-21

Wedel T, Spiegler J, Soeliner S, Roblick UJ, Schiedeck TH, Bruch HP, Krammer HJ. Enteric nerves and interstitial cells of Cajal are altered in patients with slow-transit constipation and megacolon. *Gastroenterology* 2002; 123: 1459-1467

Siproudhis L, Pigot F, Godeberge P, Damon H, Soudan D, Bigard MA. Defecation disorders: a French population survey. *Dis Colon Rectum* 2006; 49: 219-227

Chiaroni G, Whitehead WE, Pezza V, Morelli A, Bassotti G. Biofeedback is superior to laxatives for normal transit constipation due to pelvic floor dyssynergia. *Gastroenterology* 2006; 130: 657-664

Seow-Choen F. Surgery for haemorrhoids: ablation or correction. *Asian J Surg* 2002; 25: 265-266

Simpson J, Scholefield JH, Spiller RC. Pathogenesis of colonic diverticula. *Br J Surg* 2002; 89: 546-554

Colecchia A, Sandri L, Capodicasa S, Vestito A, Mazzella G, Staniscia T, Roda E, Festi D. Diverticular disease of the colon: new perspectives in symptom development and treatment. *World J Gastroenterol* 2003; 9: 1385-1389

Chia JG, Wilde CC, Ngoi SS, Goh PM, Ong CL. Trends of diverticular disease of the large bowel in a newly developed country. *Dis Colon Rectum* 1991; 34: 498-501

Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. *Arch Dis Child* 2000; 83: 18-24

Reid DJ, Conrad SA, Hendricks SM. Tracking nutrition trends, 1989-1994: an update on Canadians' attitudes, knowledge and reported actions. *Can J Public Health* 1996; 87: 113-118

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