

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 chronic 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^[2]. 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^[4]. 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^[4]. It is thus obvious that the more fiber is ingested, the more stools will have to be passed.

Table 1 Classification of fiber

	Material	Chemical structure	Source	Hydrolysis	Fermentation in colon
Soluble fibers	Pectins	Nonstarch polysaccharide	Plant cell wall	Yes	Rapid
	Guar	Nonstarch polysaccharide	Beans	Yes	Rapid
	Ispaghula	Acidic arabinoxylan	Husks of seeds of platago ovata	Yes	Rapid
	Hemicellulose	Nonstarch polysaccharide	Component of plants	Yes	Variable
Insoluble fibers	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	Complete

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 <i>et al</i> 2006 ^[15]	Japan	507/2535	0.65 ($P < 0.05$)	Yes (colon)
Levi <i>et al</i> 2001 ^[16]	Switzerland	286/550	0.55 ($P < 0.05$)	Yes
Ghadirian <i>et al</i> 1997 ^[17]	Canada	402/668	0.50 ($P < 0.01$)	Yes
Slattery <i>et al</i> 1997 ^[18]	United States	1993/2410	0.70 (95% CI 0.5-1.0)	No
Little <i>et al</i> 1993 ^[19]	United Kingdom	147/329	0.60 (not significant after adjustment for energy intake)	No
Steinmetz <i>et al</i> 1993 ^[20]	Australia	220/438	0.77 (not significant)	No

CRC: Colorectal cancer.

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 faeces only. Strange as it sounds, studies have been performed just to show stool weight increases as fiber intake increases^[5]. 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^[5]. 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^[5]. 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^[4]. 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^[3,6].

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*^[11] 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 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 <i>et al</i> 1999 ^[24]	US	88757 (women)	16	0.95 (95% CI 0.73-1.25)	No
Mai <i>et al</i> 2003 ^[25]	US	45491 (women)	8.5	0.94 (95% CI 0.71-1.23)	No
Lin <i>et al</i> 2005 ^[26]	US	36976 (women)	10	0.75 (95% CI 0.48-1.17)	No
Otani <i>et al</i> 2006 ^[27]	Japan	86412	10	Hazard ratio of lowest fiber intake group compared to highest group: 2.3 (95% CI 1.0-5.2)	No
Shin <i>et al</i> 2006 ^[28]	China	73314 (women)	5.7	1.1 (95% CI 0.6-1.8)	No

CRC: Colorectal cancer.

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 <i>et al</i> 2000 ^[30]	Postpolypectomy	2079	Counselling	Recurrent adenoma at 4 yr	0.88 (95% CI 0.70-1.11)	No
Schatzkin <i>et al</i> 2000 ^[31]	Postpolypectomy	1429	Fiber supplement	Recurrent adenoma at 36 mo	1.00 (95% CI 0.90-1.12)	No
Ishikawa <i>et al</i> 2005 ^[33]	Postpolypectomy	398	Fiber supplement	Recurrent adenoma at 4 yr	1.31 (95% CI 0.87-1.98)	No
Jacobs <i>et al</i> 2006 ^[34]	Postpolypectomy	3209	Fiber supplement	Recurrent adenoma	0.91 (95% CI 0.78-1.06)	No
Pooled from 2 studies						

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*^[24] studied 88757 nurses over 16 years and found no effect of dietary fiber on colorectal cancer. Another study of 45491 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 725628 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^[30,31]. Alberts *et al*^[30] 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^[31] 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^[35]. 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^[36]. Several studies that looked at dietary fiber intake by people with chronic constipation did not find any difference in fiber intake compared to controls^[37-40].

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^[35]. 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 bulking stool consistency. The adverse effects were similar. More patients preferred the use of lactulose (61.5%) over fiber (35.9%) to ease constipation^[41]. Most patients who are constipated are already on large amounts of dietary fiber, having been influenced by the media and doctors in promoting high fiber diets indiscriminately.

Voderholzer *et al*^[42] 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^[43]. A meta-analysis of the use of bulking agents in irritable bowel syndrome was performed in Switzerland^[44]. 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^[45]. A more recent randomized-controlled trial in the UK also failed to show benefits of fiber over placebo^[46].

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^[5]. A subsequent population based study of older people also found that dietary fiber did not reduce total transit time^[47]. Another recent publication from France examined the colonic response to food in 323 constipated patients and 60 healthy adults, and found that an abnormal colonic response to food is frequently found in constipated patients^[48]. There have been recent demonstrations that patients with colonic inertia actually have decreased volumes or numbers of interstitial cells of Cajal and enteric neurons^[49,50]. 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^[51]. 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^[52]. 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 anorectal disorders including haemorrhoids and anal fissures. The most important factor in the pathogenesis of haemorrhoids is repeated straining when passing stools^[53]. This results in the disruption of the suspensory ligaments of Park at the anal cushions leading

to prolapse of haemorrhoidal tissue. We believe that the frequent straining and passage of large bulky stools will in fact result in compromise of these suspensory ligaments.

Passage of large bulky stools will also result in direct trauma from stretching of the anal mucosa leading to anal fissures. With the development of chronic fissure, secondary anal sphincter spasm with resultant anodermal ischaemia, it does not make sense to further worsen the spasm by trying to force large bulky stools through the anus.

DIVERTICULOSIS

The fiber deficiency hypothesis in the pathogenesis of colonic diverticula has been widely quoted. The hypothesis is that colons in areas of high fiber intake are large-bore compared to areas of low fiber intake. The pressure required to distend the colon, according to Laplace's law, is greatest where the radius is small and so passage of faeces through a narrow colon requires greater luminal pressure leading to formation of pulsion diverticula^[54]. This hypothesis is actually not universally accepted and controversy still exists. In fact, this hypothesis has never been confirmed with controlled clinical studies comparing healthy subjects with diverticular disease patients^[55]. Actually, the prevalence of diverticular disease in an Asian community was found to be similar to that of American and European studies and thus in variance with the widely held belief that diverticulosis occurs less frequently in oriental communities^[56].

While there are numerous studies which found an increased intake of fiber in Western populations over the last 3 decades^[57,58], the incidence of diverticular disease has not been found to be reduced. Conversely the incidence of diverticulosis and complications of diverticular disease have been increasing in the West despite increase in dietary intake^[9]. We believe this is probably related to the massive gaseous build up associated with a high dietary fiber intake and that in fact fiber has an adverse effect on diverticular disease.

SHOULD INCREASED FIBER INTAKE BE RECOMMENDED?

The only benefits of fiber in the diet are in cases of diabetes mellitus and hyperlipidaemia^[4], due to its anti-nutritive effect. Fiber is non-absorbable and therefore ingestion of large amounts leads to reduction in calorie intake. High natural fiber-diets rich in digestible fiber also produce gas, causing longer lasting satiety.

Soluble fiber supplements should be considered when the desired effect is to delay gastric emptying and small bowel transit. The situations that thus will benefit from soluble fiber supplements are in cases of short gut syndrome or after a total colectomy when the patient suffers intractable frequent diarrhoea, as fiber bulks up stools and normally results in constipation. It can also be used temporarily in patients with diarrhoea secondary to resection of the terminal ileum or cholecystectomy. 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.

COMMENTS

Background

Whilst fruits and vegetables are an essential part of our dietary intake, even the role of fiber in the pathogenesis of diverticulosis has not reached consensus. Although previous correlation and case-control studies have mostly shown fiber to be protective in colorectal cancer, there are quite a number of newer large prospective longitudinal studies that did not reveal a similar association. There were then a few very high profile epic interventional studies that also failed to show that fiber protects against adenoma recurrence. There is also controversy in the use of fiber in patients with constipation and irritable bowel syndrome. Prescription of more fiber has been very popular in these symptoms. However recent studies of patients with chronic constipation have failed to show any beneficial results with the use of fiber. Meta-analyses on irritable bowel syndrome also did not find any clear benefit of using fiber. Even the role fiber in the pathogenesis of diverticulosis has not reached consensus. This paper reviews the physiology and literature on fiber in colorectal diseases.

Research frontiers

Continuing research is required to determine the exact role of fiber in various colorectal diseases. These studies should not only be in the form of large well designed clinical studies but also focus on the physiology of fiber ingestion and its products of fermentation.

Applications

We want to emphasize that what we have all been made to believe about fiber needs a second look. 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.

Peer review

This is a comprehensive review on the use of fiber supplements in many disease conditions. The literature is cited well and the review is overall written well.

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