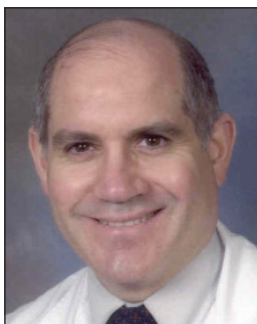


Carol Rees Parrish, R.D., M.S., Series Editor

Nutrients and Constipation: Cause or Cure?



Lawrence R. Schiller

For over 100 years diet has been postulated as the major cause for constipation and improvement of diet has been the major lifestyle recommendation for the management of constipation. The basic evidence supporting low fiber intake as a cause for constipation and other bowel problems goes back to epidemiological observations in the 1960's. More recent evidence suggests that low calorie intake and perhaps food sensitivities may play a role in the pathogenesis of constipation. Modification of diet has been touted as a cure-all for constipation, but the evidence base in support of this is weak. In fact, some suggested modifications, such as increased fiber intake, may be counterproductive in some patients, and others, such as the use of probiotics or prebiotics, lack much scientific evidence of efficacy. Nevertheless, patients with constipation want to know what they ought to do about their diets and certain guidelines can be recommended.

INTRODUCTION

Constipation is a common affliction said to be present in 15% of the population (1). It is most commonly defined as difficulty with defecation characterized by infrequency or dyschezia (painful, hard or incomplete evacuations) (1). The Rome Committee, a group of experts in functional gastrointestinal disorders, promulgated a specific set of criteria for

functional constipation which can be used for research and clinical purposes (Table 1) (2).

While constipation is most likely related to several inciting factors, the concept of inadequate dietary fiber intake has assumed a place of prominence as *the* etiology of constipation among both the lay population and physicians. This dates back to the early 1970's when epidemiological studies suggested that bowel movement frequency and weight varied with fiber intake from population to population, and to even earlier observations that stool output could be increased by increasing dietary fiber of a variety of types (3). Obser-

Lawrence R. Schiller, M.D., Digestive Health Associates of Texas, Baylor University Medical Center, Dallas, TX.

vations that the Western diet had progressively less fiber as it became more “refined” over the preceding century were coupled with observations from developing nations that constipation and a bevy of other conditions ranging from hemorrhoids to coronary artery disease were less common when fiber intake was much higher than in the West. Once the association had been made, educated Westerners increased their fiber intake as part of a “healthy diet.” Far from disappearing though, constipation continues to be a common problem and patients still ask what they should eat to fix the problem.

More recently, other elements of the diet such as milk proteins have been suggested to cause constipation due to food sensitivities (4). More exotic dietary supplements, such as prebiotics and probiotic bacteria, have been suggested as treatments for constipation (5). This paper will briefly review the evidence for nutrients as a cause or cure for constipation.

NUTRIENTS AS A CAUSE OF CONSTIPATION

Fiber

Insufficient fiber intake has been postulated as a cause for constipation and supplementation of fiber as a cure for over 120 years (3). Fiber is defined as plant wall constituents that resist digestion in the human small intestine. Cellulose, hemicellulose, pectins, fructooligosaccharides, and resistant starches are the major carbohydrate components. Lignin is the main non-carbohydrate constituent of fiber. Chemical analysis of foodstuffs introduced the concept of “crude fiber;” residue left after extraction with boiling sulfuric acid, sodium hydroxide, water, alcohol and ether. Since this processing is significantly more capable of degrading foodstuffs than the secretions of the gastrointestinal tract, the grams of crude fiber substantially underestimates total dietary fiber and leads to confusion when fiber intake is quantitated. Fiber experts also distinguish “soluble” and “insoluble” fiber, which have different abilities to form gels and be fermented, but which have not been distinguished in most epidemiological studies (6). Each food contains a different spectrum of substances categorized as fiber in addition to any differences in the total amount of fiber present. Thus, fiber sources are not necessarily interchangeable in terms of potential biological effects.

Table 1
Rome Criteria for Diagnosis of Functional Constipation*

1. Must include 2 or more of the following:
 - a. Straining during at least 25% of defecations
 - b. Lumpy or hard stools in at least 25% of defecations
 - c. Sensation of incomplete evacuation for at least 25% of defecations
 - d. Sensation of anorectal obstruction/blockade for at least 25% of defecations
 - e. Manual maneuvers to facilitate at least 25% of defecations (e.g., digital evacuation, support of the pelvic floor)
 - f. Fewer than 3 defecations per week
2. Loose stools are rarely present without the use of laxatives
3. There are insufficient criteria for IBS (i.e., pain is not the predominant symptom)

*Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis (2).

Fiber passes into the colon where substantial bacterial fermentation occurs, producing short chain fatty acids and gases (3). Thus, although our endogenous metabolism can not digest these substances, our “captive” bacterial flora can. It has been estimated that the endogenous flora of an average adult can metabolize up to 80 g of carbohydrate delivered to the colon each day (7). The net amount of energy recovered by bacterial metabolism of dietary fiber is relatively small (a typical fiber intake of 25 g per day would theoretically yield only 100 kcal if fully available to the host). Even if the dietary fiber was not metabolized at all, the direct impact of this amount of solids on stool weight would be small (e.g., only an additional 25 g out of 100 g of stool). However, the polymers that constitute dietary fiber in stool solids complex about 4 to 5 times their weight of water and thus are a major determinant of stool weight and consistency (8). In addition, the fermentation of fiber in the colon produces many smaller molecules that can exert osmotic activity, enhancing water retention intraluminally and perhaps stimulating motility in the colon (3). Thus, there is physiological plausibility for a lack of dietary fiber as a cause for constipation.

Epidemiological associations from 35 years ago have provided additional support for this concept. Burkitt popularized the observation that rural Africans consuming a high fiber diet produced more stool daily than urban Africans on a mixed diet or English subjects consuming a low fiber diet (470 g/day versus 185 g/day versus 108 g/day) (9). Although intake was not measured in this study, and the numbers of subjects were small, this information tied in with contemporaneous studies of intestinal transit and short-term diet modification studies to influence physicians and ultimately the public that a high fiber intake was desirable.

Despite the widespread increase in the availability and ingestion of fiber-rich foods, there has been no documented decrease in the prevalence of constipation in the United States over the last three decades. This suggests that deficient fiber intake is not the main cause of constipation (although increased fiber intake may be of use in the treatment of some patients, see below). Recent studies from Japan highlight the lack of an inverse association of constipation with fiber intake and instead stress the relationship to the types of food consumed (consumers of a "traditional Japanese" diet doing better than those on a "Western" diet) (10,11).

Low Total Calorie Intake

Low total calorie intake also has been postulated as a cause for constipation. Studies in children from Greece showed that constipated children had a lower caloric intake than non-constipated children (12). Studies in the elderly suggest that constipated individuals consume fewer meals per day and tend to take in fewer calories; there was no correlation with fiber intake (13). Since eating stimulates colon motor function by the gastrocolic reflex, eating less food less often would stimulate colon motility less. In addition, constipated patients may have altered colon motor responses to eating. A large study using radio-opaque markers to measure segmental colon transit has shown that constipated individuals have abnormal colon motor responses to eating: 41% of patients with normal transit constipation had no response to eating (as compared to only 13% of controls); those with colonic inertia had attenuated responses (14).

Food Sensitivities

Another intriguing line of research into the nutritional pathogenesis of chronic constipation has to do with food intolerances (4). Food allergy is usually linked with diarrhea, but studies in children imply that cow's milk protein intolerance may produce chronic constipation: 28% of children with chronic constipation in one small study improved on an exclusion diet and relapsed with rechallenge (15). Other studies have linked this to a proctitis with prominent intraepithelial lymphocytic infiltration and lamina propria eosinophilic infiltration (16). Studies in adults with laxative-refractory constipation have shown similar histological changes in the rectal mucosa and improvement with a hypoallergenic elimination diet (17,18). Food intolerances and allergies are unlikely to play major roles in most cases of chronic constipation, but this concept bears further investigation.

Iron and Calcium

Some dietary constituents routinely may produce constipation in individuals consuming them. Prominent among these are calcium and iron supplements. The prevalence of constipation due to ingestion of these supplements as well as the mechanism of the constipating effect is unknown. Calcium may complex with short chain fatty acids to produce soaps and reduce the effect of short chain fatty acids on epithelial transport and colon motility.

NUTRIENTS AS A TREATMENT FOR CONSTIPATION

Fiber supplementation has been proposed as a treatment for constipation for more than 120 years (3). Subsequent studies in normal subjects showed that short term increases in fiber intake were associated with increased stool output. One of the best designed studies of the effects of dietary fiber intake was conducted at a U.S. Department of Agriculture facility in North Dakota (19). Normal volunteers were isolated at the facility, were fed only standard or modified diets for one month at a time and stool output was recorded scrupulously by observers who were with the subjects constantly. Stool frequency and weight could be altered by a factor of two by variations in diet. Other studies suggest that wheat bran is most effective at increasing stool weight, followed by fruits and vegetables, oats,

Table 2
Commercial Fiber Supplements and Cost (per 3 g of fiber)

<i>Name</i>	<i>Dose</i>	<i>Total Fiber (g)</i>	<i>Cost (3 g of Fiber)</i>	<i>Gluten Free</i>
Fiber Containing Cereals				
Fiber One	1/2 cup	14	0.05	No
All Bran Original	1/2 cup	10	0.07	No
All Bran Wheat Flakes	3/4 cup	5	0.14	No
Raisin Bran	1 cup	8	0.11	No
Oatmeal	1/2 cup dry	4	0.10	No
Fiber Laxatives				
Benefiber	2 Tbsp	3	0.18	Yes
Citrucel	1Tbsp	6	0.27	Yes
Fiber Choice SF	2 Tablets	4	0.22	No
Fiber Sure	3 Tsp	15	0.16	No
FiberCon	2 Tablets	1	0.30	No
Generic Orange Smooth Text	3 Tsp	9	0.27	No
Generic Regular	3 Tsp	9	0.10	No
Generic Orange SF	1 Tbsp	9	0.27	No
Generic Fiberlax	2 Tablets	1	0.20	No
Konsyl	1 Tsp	6	0.15	Yes
Metamucil Fiber Wafers	2 Wafers	6	0.25	No
Metamucil Orange Smooth Texture	3 Tbsp	9	0.14	No
Metamucil Orange Smooth Texture SF	3 Tsp	9	0.11	No
Metamucil Course Milled	3 Tsp	9	0.11	No
Metamucil Smooth Text	3 Tbsp	9	0.14	No

Table developed by Claire Morris, University of Virginia Health System Dietetic Intern March 2007

corn, and soya (see Table 2 for a comparison of commercial fiber supplements) (20). Mucilages, cellulose and pectin were less effective than wheat bran. Studies like this clearly show that fiber intake can drive stool output in normal individuals.

There is less evidence that this occurs in patients with constipation. While some studies have suggested that dietary fiber is beneficial (21,22), more recent work has emphasized the ineffectiveness of dietary fiber in most patients with constipation (23). This should not be surprising since the definition of constipation has been expanded to include more than just infrequency; and inadequate fiber intake is no longer thought to be the major factor in the pathogenesis of constipation. Nevertheless, some patients may respond to fiber supplementation and most patients with constipation should be tried on fiber before pursuing

advanced diagnostic testing or other therapies (see Table 3 for some exclusions to this rule) (24).

Fiber is most likely to be helpful in patients with normal transit constipation and those who might benefit from modification of stool consistency (i.e., production of softer stools). It is unlikely to help those who have serious dysmotility problems whether due to drugs (e.g., opiates) or to enteric neuromuscular disorders causing colonic inertia (Table 3). Patients who have evacuation problems due to pelvic floor disorders, such as pelvic floor descent due to denervation or dyssynergia (contraction of the pelvic floor or anal canal when attempting to defecate), also may not benefit from fiber supplementation; it may only result in more stool to evacuate with straining. Similarly, patients with secondary constipation

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Table 3
Patients with Constipation Who Will Not Benefit from Increased Fiber

Dysmotility disorders

- Chronic intestinal pseudoobstruction
- Hypothyroidism
- Colonic inertia
- Gastroparesis

Neuromuscular disorders especially in immobile or wheelchair bound patients

- Amyotrophic lateral sclerosis
- Multiple sclerosis
- Muscular dystrophy
- Friedreich ataxia
- Scleroderma involving the gut
- Cerebral palsy
- Para- or quadriplegia

Chronic use of opiates

- Oncology patients, especially when doses of pain medication are increased
- Chronic pain patients
- Narcotic bowel syndrome

Pelvic floor disorders

- Pelvic descent due to denervation
- Dyssynergic defecation

due to some other disorder such as hypothyroidism will do better with treatment of the underlying disorder.

The key to using fiber successfully is to assess dietary fiber intake before making recommendations for fiber supplementation. If a given patient is already taking 25–30 g of dietary fiber daily, further supplementation is unlikely to be helpful. If less than this amount is being consumed, fiber should be added slowly in graduated doses to eventually reach 25–30 g per day. If the entire supplemental amount is added at once, it is likely that the patient will develop intolerable side effects, such as bloating or excessive flatus. These side-effects are due to fermentation by the colonic flora; every 10 g of carbohydrate reaching the colon may yield as much as 1000 mL of gas from fermentation (7).

There is no clear consensus as to the ideal dietary fiber supplement for constipated patients. While studies

Table 4
Recommendations for the Use of Dietary Fiber in Constipation

1. First, ask if the patient routinely uses laxatives in order to have a bowel movement—if they do, they may not respond to fiber therapy
2. Also ask what their normal bowel habit has been—if they have had life-long infrequency (<3 BM per week) but do not have other symptoms of constipation such as straining to have a bowel movement—they do not need therapy
3. Empiric therapy with dietary fiber is an appropriate initial therapeutic trial in patients who meet criteria for functional constipation and have no alarm signs or symptoms (e.g., evidence of bleeding, weight loss, and bowel obstruction)
4. Take a careful dietary history to try to estimate current fiber intake; use dietary tables to quantitate fiber intake in grams per day
5. If the patient is already consuming 25 g–30 g of fiber per day, supplementation is less likely to be helpful; expectations should be tempered
6. Start fiber supplementation with 5 g daily; titrate the amount up to a target daily intake of 25 g in 5 g increments
7. Monitor the patient for side-effects: gas, bloating, and pain; if side-effects develop, either reduce the dose temporarily and then retitrate up at a slower rate, or try a different fiber preparation
8. Assess the response to fiber therapy after a few weeks at the target dose; abandon fiber therapy and try something else if there is no demonstrable improvement

in normal individuals would favor wheat bran, there appears to be little clinical difference when different products are used in patients—even those that performed poorly in studies in normal subjects seem to be effective in some patients (1,25). It is probably best to be familiar with several different preparations and to see which one is most tolerable and most effective for an individual patient. Suggestions for the use of dietary fiber in patients with constipation are listed in Table 4.

The use of other dietary treatments for constipation is even less well studied. Much attention has been paid to the potential role of the enteric flora in the genesis of constipation and the opportunity to manipulate that

flora by the use of prebiotics or probiotics (5,25,26). Probiotics are “good” bacteria that can be ingested with positive effects on health. Bifidobacteria and Lactobacilli are the groups most often placed in this category, but classification of a given strain as a probiotic is empiric at present (25). Prebiotics are food chemicals that promote the growth of probiotic bacteria in the gut by acting as substrates for those bacteria (26). The most commonly identified prebiotics are oligosaccharides, but in a larger sense, lactulose and dietary fiber fulfill a similar role. A series of small clinical trials suggest that prebiotics can ameliorate constipation, but how much of this is due to a prebiotic effect as opposed to an osmotic laxative effect is unknown (5).

Other studies suggest that the fecal flora is different in patients with constipation than in nonconstipated subjects, with reduction in the numbers of Bifidobacteria (5). While it is not clear whether this is cause or effect, several (but not all) attempts to “correct” the bacterial flora by ingestion of probiotic strains have shown at least transient improvement in constipation. Since there is no a priori scheme for what constitutes a probiotic bacterium, each individual probiotic preparation needs to be tested for effectiveness; generalizations are not possible. The preponderance of limited evidence suggests that there may be a treatment effect with probiotic bacteria, but clearly a more vigorous scientific basis for their effect would be welcome.

CONCLUSIONS

Chronic constipation is a common symptom complex that may be fostered by inadequate fiber intake, but this is unlikely to be a common cause for constipation. Nevertheless, addition of fiber to the diet improves symptoms in some patients. In some individuals, reduced calorie intake, intolerance for specific foods in the diet, or disturbed enteric bacterial flora may produce constipation; these etiologies need to be explored further. Likewise, the potential role of prebiotics and probiotics in the management of chronic constipation requires further evaluation. ■

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