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# Diverticular Disease — A Reexamination of the Fiber Hypothesis

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Ask a roomful of dietitians about a dietary factor that has solid evidence for helping to prevent a disease, and chances are many would pick fiber and the prevention of diverticulosis. That's why a recent study from the University of North Carolina has everyone interested in nutrition and gastrointestinal disease scratching their heads.[1] It also has motivated many nutrition professionals to go back to the original theory linking fiber to gastrointestinal disease and reevaluate earlier studies that generated or supported the theory.

Diverticular disease subsumes an array of clinical states that begins with herniation of the colonic mucosa and muscularis mucosa through the intestinal wall, and although it can occur in any part of the intestine, the colon—particularly the sigmoid colon—is the area typically affected.[2] The presence of one herniation—a saccular protrusion or outpouching—is a diverticulum (plural: diverticula), and the presence of diverticula indicates diverticulosis.

Diverticulitis is a complication of diverticulosis that indicates inflammation of one or more diverticulum. Other complications that can arise from diverticulitis include intestinal obstruction, bleeding, abscess, fistula, and perforation. In addition to being a complication, diverticulitis represents a flare-up of diverticulosis and, after it subsides into a period of remission, reverts back to the state of diverticulosis.

Diverticular disease certainly warrants closer scrutiny based on the toll it exacts on the people it affects, the substantial health care costs incurred for its treatment, and the serious complications it can cause. According to a 2008 report from the National Institutes of Health (NIH), it was the underlying cause of death among 58% of death certificates on which it was listed.[3] A 2004 report, cited by most recent reviews, stated that the economic burden of diverticular disease exceeds $4 billion every year; the actual current figure likely is significantly higher.

As life expectancy has increased since diverticular disease was first described in 1920, the prevalence of the disease also has grown. It was relatively rare in 1930, with a prevalence of only 5% to 10%. It now affects 33% of all Americans by the age of 50, 50% by age 60, and 66% by age 80, with similar rates in Canada.[4,5] As the North American population continues to age, the burden of diverticular disease certainly will rise.

This continuing education course reviews diverticular disease, including its epidemiology, and the evidence for nutrition and lifestyle factors in disease development, exacerbation, and recurrence.

## Symptoms, Clinical Manifestations, and Diagnosis

The majority of people with diverticular disease have asymptomatic diverticulosis. However, 25% of those with diverticulosis experience occasional bloating, flatulence, pain, and disordered intestinal motility resulting in either diarrhea or constipation, known as symptomatic diverticulosis.[6,7] For most people, the symptoms usually improve after a bowel movement. However, because the symptoms are nonspecific, there's considerable overlap with irritable bowel syndrome (IBS), an important disease in differential diagnosis.[8]

Of those with symptomatic diverticulosis, about 25% will develop complications such as diverticulitis, termed complicated diverticular disease.[2] The symptoms of diverticulitis include nausea, vomiting, abdominal distension, left lower quadrant pain, intestinal spasms, fever, and bleeding. Of these symptoms, left lower quadrant pain is most common, affecting 93% to 100% of patients seeking treatment.[7]

Some studies have indicated that the symptoms and disease are more severe in people younger than the age of 40, although others haven't reported this correlation,[9-11] and diverticular disease is more common among men and people who are obese.[12]

In Asia, diverticular disease is more common in the right colon, in contrast to Western countries where the left colon typically is affected, suggesting that genetic and environmental factors may play a role in disease development.[13,14] When the disease occurs in the right colon, it can be misdiagnosed as appendicitis. In Asians under the age of 40, it's more common in the left colon, with one study showing that 97.5% of patients in that age group had the left-sided type.[13]

Since most people with diverticulosis are asymptomatic, diagnosis typically occurs as the result of undergoing a colonoscopy or double-contrast barium enema for routine screening or investigating other symptoms.[15]

Diverticulitis most commonly is diagnosed when an acute attack results in an emergency hospital admission. In this setting, endoscopy usually isn't indicated and, if performed, requires extreme caution to prevent perforation, as even air insufflation can cause a sealed perforation to rupture.[7]

Typically, a diverticulitis diagnosis begins with a comprehensive history and physical examination, complete blood count, abdominal radiography, and urinalysis. Physicians may order other tests when these clinical assessments fail to provide a diagnosis, including ultrasound, CT, and water-soluble contrast enema, which is safer in emergency settings. Differential diagnosis becomes important when distinguishing diverticulitis from appendicitis, bowel obstruction, IBS, and colorectal cancer, which all can coincide with diverticular disease.

## Complications

Diverticulitis represents a complication of diverticulosis, in that it arises from diverticulum inflammation and/or infection. In uncomplicated or simple diverticulitis, which represents about 75% of cases of the condition, the resulting inflammation isn't severe and if the perforation is

small, it's enveloped by pericolic fat, or the fat surrounding the colon that includes subserosal, retroperitoneal, and mesenteric fat, reducing the risk of infection or more severe complications.

However, several complications can arise when the original perforation isn't well contained, such as complicated diverticulitis. Other serious complications include a major perforation, bleeding, bowel obstruction, fistula, abscess, phlegmon (diffuse inflammation with purulent exudate), abscess, adjacent organ involvement, and sepsis.

**Medical Treatment**

The rate of surgical intervention in complicated diverticulitis dropped from 17.4% to 14.4% from 1999 to 2005, with more emphasis on conservative treatment when possible.16 Conservative treatment for simple diverticulitis consists of consuming a low-fiber diet and taking oral antibiotics on an outpatient basis.[17]

If abdominal pain and tenderness are more severe, the patient will require hospitalization, particularly if the patient can't tolerate oral feedings and continues to experience a fever. In this case, treatment includes bowel rest, IV fluids, and antibiotics. Surgery may be required for serious complications such as obstruction, major perforations, abscesses, fistula, and phlegmon.

**Nutrition Recommendations**

In the hospital, a patient suspected of having diverticulitis typically should consume nothing by mouth before testing. Once the diagnosis is made, the patient may require enteral nutrition if bowel obstruction occurs or if the ileocecal valve is incompetent and distension arises in the small intestine. If there are no other serious complications, the patient will progress to nutrition therapy.

The medical nutrition therapy for acute diverticulitis consists of instructing the patient to follow a low-fiber diet (10 to 15 g/day) for a short time after the attack and then increase fiber gradually to reach and maintain a high-fiber diet with adequate hydration.[18] The current recommendation for daily fiber intake is 25 g for most adult women and up to 38 g for men. This actually represents a high fiber intake, as the average male and female consume approximately 50% and 62%, respectively, of the recommended level.[19]

Many older patients who have had diverticular disease for several years still avoid certain foods such as small seeds, nuts, and popcorn that once were believed to precipitate an attack of diverticulitis, intestinal bleeding, or both. However, both the NIH and a large prospective study of more than 47,000 men reported no evidence to support that belief.[20] To the contrary, the study results indicated that these foods actually may be associated with a lower risk of diverticular disease.

**Etiology, Pathophysiology, and Risk Factors**

The search for the cause of diverticular disease began with a hypothesis by two British surgeons, Neil Painter and Denis Burkitt, published in 1971.[21] Their review of medical textbooks revealed no mention of the disease since 1916, but they noted that the prevalence had skyrocketed during the years before their paper. They observed that diverticular disease

practically was nonexistent in less economically developed countries such as Africa while common in Western countries.

Furthermore, as less economically developed countries became more industrialized, the rate of diverticular disease increased concomitantly. Painter and Burkitt theorized that this geographic variation was linked to dietary fiber, with intake decreasing as countries became more industrialized.

They postulated that as diets become more Westernized, they contained significantly less fiber, precipitating the development of diverticular disease. Indeed, the title of their article labels diverticular disease as “a deficiency disease of Western civilization.” This “fiber hypothesis” has prevailed since their paper was published and, although it was somewhat controversial in research circles, only recently has it been challenged in earnest.

The fiber hypothesis posits that the disease’s pathogenesis begins with colonic segmentation, the process by which the contents of the large intestine move forward, a nonpropagated rhythmic contraction. This is different from peristalsis, the process that moves the food mass forward in the small intestine, which is a propagated wave.

The fiber hypothesis proposes that a low-fiber diet causes segmentation to occur more frequently and efficiently, which results in higher localized intraluminal pressure. Higher intraluminal pressure, in conjunction with weakened colonic musculature, which occurs in aging, favors the development of diverticular disease.

Painter and Burkitt proposed that fiber protects against high intraluminal pressure by producing higher-volume feces, which in turn increases colonic diameter. This may seem counterintuitive, but it’s based on a variation of the Law of LaPlace. This law states that the pressure within a cylinder, which the intestine essentially is, equals the tension on the wall divided by the radius.[22] Relative to the colon, intraluminal pressure then is inversely proportional to the radius, so as fiber distends the colon and increases its radius, pressure inside the colon is reduced.

In addition, Painter and Burkitt noted that because of the higher fiber content of the African diet, which was the focus of their study, “Food residue passes through the African’s gut within 48 hours, whereas in an Englishman this may take more than twice as long.” The shorter transit time results in lower water reabsorption, so the colon propels a less viscous fecal mass that generates lower pressure, much less conducive to the formation of diverticula.

Given this presumably reasonable rationale and epidemiologic evidence, it’s not surprising that the fiber hypothesis has been so resilient.

### *Effects of Aging*

The strong association of age with the presence of diverticular disease argues for the important role of the aging process in the disease. In 2005, the American Gastroenterological Association issued a committee report on the impact of the aging population on gastroenterology practice, education, and research.[23] The authors noted that aging causes an increase in collagen in the colonic wall, with a concomitant reduction in tensile strength, that

makes herniation more likely. In addition, with advancing age, intestinal motility slows, although studies have demonstrated conflicting results regarding this.[24] Slower motility results in higher colonic water reabsorption and harder feces, which causes excessive straining and higher intraluminal pressure.

Another factor in aging that makes herniation more likely is the reduction in neurons containing nitric oxide in the myenteric plexus.[25] These nerve cells play a role in receptive relaxation, a vagovagal reflex. These reflexes control muscular contraction to propel food through the gastrointestinal tract, and colonic receptive relaxation is important in allowing for expansion to accommodate and propel the fecal mass with lower intraluminal pressure. In addition, the residual nitric oxide–containing neurons may be less functional.

Chronic use of laxatives over a long period of time could potentially damage the colon, a condition gastroenterologists have dubbed the “cathartic colon,” making it more susceptible to diverticular disease and other gastrointestinal disorders.[26] Although most practitioners subscribe to this belief and caution patients based on it, some researchers believe evidence is lacking.[27]

### *Genetics*

The geographic variation in right or left colon diverticular disease suggests to some researchers that genetic differences in those populations are important in disease development.[28] In addition, the fact that individuals who have certain genetic disorders are more susceptible to developing diverticula suggests a role for genetics in diverticular disease. These disorders include Ehlers-Danlos syndrome, Williams-Beuren syndrome, Coffin-Lowry syndrome, and polycystic kidney disease, and researchers have identified the specific genetic mutations. One study reported that among people with end-stage polycystic kidney disease, the rate of diverticula was 83%.[29] The defective genes in these disorders may be related to diverticular disease via the smooth muscle accumulation of collagen and elastin.

A recent study in Denmark analyzed diverticular disease cases using a national patient registry, which included 10,420 siblings and 923 twins.[30] The study of familial aggregation of diverticular disease showed that the relative risk among siblings was 2.92 compared with the general population. The relationship was even stronger for diverticular disease cases requiring hospitalization or surgery.

When one monozygotic twin had diverticular disease, the relative risk for the other twin was 14.5. In dizygotic twins, the relative risk was 5.5, and the effect was stronger for females compared with males. On the basis of their results, the researchers concluded that genetic factors contribute 53% of the susceptibility to diverticular disease.

### *Obesity*

Although fiber has been the dietary focus related to diverticular disease, with researchers citing the rise in the disease’s prevalence as fiber intake declined, several aspects of diet and lifestyle have paralleled this relationship. Chief among these is BMI and obesity.

A national task force on obesity that convened in 2000 pointed to an association between obesity and diverticular disease, and researchers have provided evidence for an association between diverticular disease and BMI.[31] Strate and colleagues conducted a prospective study of 47,228 male health professionals and evaluated several parameters with respect to diverticular disease that included BMI, waist circumference, and waist-to-hip ratio.[12] All three parameters increased the risk of diverticular disease and diverticular bleeding, with relative risks ranging from 1.56 for waist circumference and diverticulitis to 3.19 for those with a BMI higher than 30 and diverticular bleeding.

The Strate study showed a link between obesity and both diverticulitis and diverticular bleeding but not diverticulosis. This may suggest that obesity affects diverticular bleeding risk via metabolic pathways related to vascular integrity.[32] Another possible mechanism is that obesity alters gut microbiota, which would be conducive to diverticular disease, since researchers have proposed that microflora may affect the development of diverticular disease.[33,34]

Perhaps a more likely mechanism linking obesity to diverticular disease relates to inflammation, particularly in light of numerous recent studies describing a state of chronic low-grade systemic inflammation in people who are obese.[35,36] Whether inflammation is an etiologic process or a consequence of obesity, it's clear that adipose tissue secretes various cytokines, proinflammatory compounds with local and systemic effects.

And relative to diverticular disease, both human and animal studies have reported a rise in plasma inflammatory markers with aging.[37,38] Chronic inflammation also can cause changes in intestinal motility and sensation, which are important in diverticular disease, by affecting alterations in the function of smooth muscle, intestinal nerves, and epithelial cells.[39]

Further support for this mechanism are data from the Strate study showing that waist-to-hip ratio—a superior indicator of visceral adipose tissue, which is more metabolically active than subcutaneous fat—was significantly correlated to diverticular disease. However, research evidence is lacking regarding an association between inflammation and diverticular disease, with some data showing no difference in fecal inflammatory markers between diverticular disease subjects and controls.[40] Further, one study reported a reduced risk of diverticular disease in patients with inflammatory bowel disease, suggesting that inflammation actually may be protective.[41]

***IBS***

In addition to being important for a differential diagnosis, IBS appears to significantly increase the risk of diverticular disease. A 2009 cross-sectional survey reported that subjects with IBS were much more likely to have diverticulosis than were subjects without IBS, and subjects aged 65 and older with IBS had a ninefold increased risk of diverticulosis.[8]

Although the reason for the association is unclear, the authors suggested several possible mechanisms. Noting the higher risk in older individuals, they posited that IBS may act in conjunction with the aging-related changes in smooth muscle and neurons to promote the development of diverticular disease.[23-25] Another possible mechanism is that bacterial overgrowth, a consequence of diverticula-induced stasis of colonic contents, may cause

chronic low-grade inflammation. In turn, inflammation sensitizes afferent neurons, giving rise to visceral hypersensitivity and hypermotility, which are hallmark features of IBS.

### *Other Risk Factors*

Studies have pointed to several other risk factors for diverticular disease, including physical inactivity,[42] red meat consumption,[43] a meat- vs. plant-based diet,[44] higher socioeconomic status,[45] hypertension,[46] and parity.[45] Some of these variables may be related to other risk factors (eg, physical inactivity and obesity, hypertension and obesity, vegetarian diet and fiber). However, the studies for these other factors are limited in number and scope.

## Fiber Hypothesis Revisited

Although many embraced Painter and Burkitt's fiber hypothesis, some researchers were skeptical. Most objections began with the obvious: Aside from dietary fiber intake, there are numerous differences between populations in industrialized countries and those in less economically developed countries that could relate to diverticular disease. These differences range from life expectancy to posture during bowel emptying.

The latter difference formed the basis for a compelling case for sitting vs. squatting as promoting diverticular disease.[47] In 1988, Sikirov argued that the level of straining in the sitting posture during bowel emptying was three times higher than that in the squatting posture.[47]

Most of the evidence for the fiber hypothesis comes from epidemiologic studies and small clinical trials, and even these have yielded conflicting results. The potential flaw in the former types of studies (ecologic fallacy) points to the problem of using population-based data to reflect an individual's risk. Reviews of the studies arrived at similar conclusions regarding the fact that the hypothesis represents a plausible physiologic mechanism, although the evidence isn't as strong as one might prefer.[2,28,48]

The Health Professionals Follow-Up Study provided some of the more compelling evidence for the fiber hypothesis.[42] In this large prospective study, insoluble fiber intake was inversely correlated with the risk of diverticular disease, particularly for cellulose.

However, there had been several missing links between the hypothesis and the data. For example, many assume that constipation, which leads to straining and high intraluminal pressure, plays a causative role in the development of diverticular disease. But Jung's large population-based study on the association between IBS and diverticular disease showed that diarrhea-predominant IBS was the strongest predictor of diverticular disease.[8] In addition, a recent study reported that fiber didn't improve stool consistency or painful defecation,[49] and a meta-analysis reported that constipation was reduced by lowering dietary fiber intake.[50]

While these studies showing that constipation isn't linked to diverticular disease and that fiber doesn't provide a benefit for constipation seem counterintuitive, they set the stage for a study that perhaps more than any other calls into question the validity of the fiber hypothesis. Peery and colleagues conducted a cross-sectional study of 2,104 subjects aged 30 to 80.[1] The researchers had access to colonoscopy reports on the subjects, a parameter typically not included in previous studies.

The results didn't show a correlation between diverticulosis and red meat intake or physical inactivity, as had previous studies. In addition, low fiber intake wasn't associated with diverticulosis; rather, high fiber intake was positively correlated to the disease. Even more stunning was the dose-response relationship between fiber intake and the number of diverticula, with the highest quartile of intake associated with the presence of more than three diverticula. In addition, constipation wasn't a risk factor, and subjects who had more frequent bowel movements (more than 15 per week vs. fewer than seven per week) had a 70% greater risk of diverticulosis.[1]

In attempting to reconcile the study results with the fiber hypothesis, the first possible consideration is that patients seeking a colonoscopy may have had previous symptoms and either started a high-fiber diet on their own or on the advice of their physicians. However, most of the colonoscopies were for routine screening. In addition, the researchers excluded a subset of subjects who previously had undergone colonoscopies with no change in the data.

The finding that frequent bowel movements were associated with a higher risk, while seeming to counter the fiber hypothesis, supports Jung's study that reported higher diverticulosis risk with diarrhea-predominant IBS.[8] In addition, since the sitting vs. squatting theory[47] suggests that the sitting position causes high intraluminal pressure, more frequent bowel movements would be expected to result in a condition of higher colonic pressures.

The Peery study results don't challenge the value of fiber for people who have diverticulosis or who have had diverticulitis and other diverticular complications. The reason high fiber intake may not help prevent diverticulosis may be related to different physiologic mechanisms that lead to the development of diverticulosis rather than diverticulitis. However, the results do raise an intriguing conundrum: High fiber may be beneficial for patients who have diverticulitis, but it actually may promote the development of diverticulosis in individuals with no prior diverticular disease, putting them at risk of diverticulitis.

**Treatment: Nutrition and Lifestyle Recommendations**

Although the Peery study has raised questions about the benefits of dietary fiber for preventing diverticulosis, the recommendations at this time remain the same. The main rationale for this is that although a high intake of dietary fiber may not help prevent diverticulosis, and indeed may promote it, still there's evidence that fiber reduces the risk of diverticular disease complications. So for patients who have diverticulosis and those who have had diverticulitis, which is the triggering event for most patient contact with an RD, high fiber intake is beneficial. And for patients recovering from diverticulitis, the short-term low-fiber and long-term high-fiber approach still applies.

Moreover, the abundance of research shows other health benefits of fiber, such as the prevention and treatment of cardiovascular disease, diabetes, and some cancers. Some of these benefits specifically are related to soluble fiber, which also appears to positively affect intestinal flora. In addition, soluble fiber is a substrate for the production of short-chain fatty acids, which serve as a fuel source for colonic cells. In light of the gut microorganisms' potential role in diverticular disease, it may be useful to ensure that patients include adequate

amounts of soluble fiber as they increase total fiber intake. Since most Americans consume about 50% of the recommended level of fiber, attaining the full amount would be an appropriate goal.

Obesity—especially abdominal obesity—appears to be an important risk factor for diverticular disease. In addition, as with fiber, strong evidence links obesity to several chronic diseases. For both reasons, modest weight loss in patients who are obese and maintaining a healthy body weight in others is an important component of nutrition and lifestyle recommendations in diverticular disease. Another aspect of lifestyle that may be helpful, despite scant evidence linking it to diverticular disease, is to engage in consistent physical activity in an effort to achieve and maintain a healthy weight.

Lastly, if further evidence strengthens inflammation's potential role in the development of diverticular disease, it may indicate an important dietary consideration. Some foods, such as fruits and vegetables, lower the level of plasma inflammatory markers, while other foods and cooking methods increase these markers. Foods that promote inflammation are those containing significant amounts of starch and sugar but small amounts of fiber. The grilling method of cooking meats, poultry, and fish also promotes inflammation by forming advanced glycation end products. Even without more evidence, as with fiber and physical activity, foods that lower systemic inflammation also are associated with a reduced risk of several chronic diseases.

**Advice for RDs**

Until further research corroborates the Peery study, RDs can be confident in current nutritional recommendations for patients and clients who have had diverticulitis, if not diverticulosis. The nutrition and lifestyle recommendations that may be beneficial for diverticular disease parallel those that RDs routinely emphasize in various practice settings.

As with many controversial topics in the field of nutrition and dietetics, it's important for RDs to continue to stay abreast of current research. Dietetic practice must be based on available evidence and not on concepts that have simply become accepted over time.

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*Click here for patient handouts "FAQs on Diverticular Disease" and "Fast Facts on Fiber."*

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**Examination**

**1. In distinguishing the various terms related to diverticulosis, diverticulitis, and diverticular disease, to what does the latter term refer?**
A. The inflammation and infection of diverticula lining the intestinal tract
B. The presence of more than one diverticulum typically in the sigmoid colon
C. Any complication caused by the inflammation and infection of diverticula
D. Various clinical states with herniation of intestinal mucosa through the wall

**2. Painter and Burkitt's fiber hypothesis posits that dietary fiber played a causative role in diverticulosis under which of the following conditions of intake?**
A. Total fiber excess
B. Insoluble fiber excess
C. Soluble fiber deficiency
D. Total fiber deficiency

**3. A 50-year-old patient has undergone her first colonoscopy, which showed no abnormalities. Your assessment of her dietary intake shows no deficient levels of any nutrients. However, she has a family history of diverticulitis, and she asks you whether she should begin fiber supplementation. Based on recent evidence, what is your answer?**
A. No, high fiber intake may promote diverticulosis.
B. Yes, high fiber intake may prevent diverticulitis.
C. No, red meat is more likely to cause diverticulosis.
D. Yes, you have a strong family history of diverticulitis.

**4. In light of recent studies, the role of a high-fiber diet in potentially promoting diverticulosis may be related to high intraluminal pressure as a result of which of the following?**
A. Lower colonic microbial flora levels
B. Higher frequency of bowel movements
C. Higher water reabsorption in the colon
D. Lower sensitization of colonic mucosa

**5. Most people with diverticulosis are asymptomatic, but among the 25% with symptomatic disease, the mortality rate for specific complications is as high as what percentage?**
A. 5
B. 20
C. 35
D. 75

**6. What is one mechanism by which obesity has emerged as an important risk factor for the development of diverticular disease?**
A. Promotion of inflammation
B. Excessive caloric intake
C. Reduction in physical activity
D. Increased intraluminal pressure

**7. You're instructing a patient admitted with diverticulitis about nutrition recommendations for discharge. He has a long history of diverticular disease and tells you that he avoids nuts and popcorn because they may trigger an attack. What should you tell him?**
A. There's sound evidence for avoiding these foods.
B. Those foods may reduce the risk.
C. The risk depends on the type of nuts.
D. Avoid popcorn if it causes diarrhea.

**8. Based on current evidence, physical inactivity may be an important risk factor for diverticular disease for which of the following reasons?**
A. It promotes low fiber intake.
B. It weakens colonic muscle.
C. It contributes to obesity.
D. It causes high colonic pressure.

**9. Which of the following gastrointestinal disorders is associated with a higher risk of diverticular disease?**
A. Idiopathic constipation
B. Irritable bowel syndrome
C. Inflammatory bowel disease
D. Gastroesophageal reflux

**10. A 40-year-old man is admitted to the hospital via the emergency department on nothing-by-mouth status with a diagnosis of diverticulitis. Testing has revealed that the ileocecal valve is incompetent with small-bowel distension. What may be the appropriate medical nutrition therapy?**
A. Parenteral nutrition
B. Low-fiber oral diet
C. Enteral nutrition
D. Full liquid oral diet