

Diet in colonic diverticulosis: is it useful?

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ABSTRACT

Diverticulosis of the colon is the most common anatomic alteration of the human colon. Diet may be important in the management of diverticular disease (DD). It is known that high-fiber diet does not prevent diverticulosis, and there are conflicting data on the prevention and treatment of DD and acute diverticulitis. No association has been reported between nut, corn, or popcorn consumption and the development of diverticulosis, DD, and acute diverticulitis. However, there seems to be a mild association between high alcohol intake and diverticulosis, whereas alcohol dependence seems to be related to a lower risk of in-hospital mortality due to acute diverticulitis. Higher consumption of red meat was associated with a mild increase in the risk of acute diverticulitis, especially when consumed as unprocessed red meat (defined as consumption of “beef or lamb as main dish,” “pork as main dish,” “hamburger,” and “beef, pork or lamb as a sandwich or mixed dish”). On the other hand, higher consumption of poultry (white meat) was not associated with the risk of acute diverticulitis. Finally, higher fish intake was associated with a reduced risk of diverticulitis in an age-adjusted model but not after adjustment for other potential confounders.

Introduction Diverticulosis is an anatomical alteration mainly located in the colon, and it is characterized by the presence of pockets called “diverticula.” These diverticula may be detected through the entire colon, but they differ in structure according to the location: in the left colon, there is herniation of the mucosa and submucosa (“pseudodiverticula”), whereas in the right colon, herniation of all colonic layers is seen (“true diverticula”).¹

Currently, the real incidence and prevalence of diverticulosis are unknown, probably due to the lack of prospective population-based studies. According to available data, diverticulosis is common in the developed world; it is more common in the United States than in Europe; and it is quite rare in Africa. However, the frequency of this condition seems to be rapidly increasing in some developing countries, particularly in the Eastern world.²

The prevalence of diverticulosis in Europe varies greatly. The European countries with a lower socioeconomic status report an increasing frequency of diverticulosis with older age: 5.3% in patients aged 30 to 39 years, 8.7% in those aged 40 to 49 years, 19.4% in those between 50 and 59 years, and up to 29.6% in individuals older than

70 years, 40.2% in those aged 70 to 79 years, and as high as 57.9% in those above 80 years of age.²

Although advancing age is obviously associated with diverticulosis, this association is not strong per se, as it is not age but the prolonged time course during which the colonic wall is exposed that makes the colon more susceptible to other pathogenetic factors. Unfortunately, the pathological mechanisms that underlie the formation of colonic diverticula are not fully known. While the occurrence of the right-sided diverticulosis (“true diverticula”) seems to be linked to genetic factors,³ the occurrence of left-sided diverticulosis (“pseudodiverticula”) seems likely to be the result of complex interactions between genetic factors, age, diet, changes in colonic structure and motility, as well as changes in colonic microbiota. Currently, there is some evidence suggesting that the prevalence of colonic diverticulosis is increasing worldwide probably due to changes in lifestyle (mainly diet).⁴

Terminology and clinical picture of diverticular disease There are various terms used to describe diverticulosis and diverticular disease (DD), which may be confounding. Therefore, we provide the correct terminology below.

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Diverticulosis The term “diverticulosis” is used to describe merely the presence of colonic diverticula, which may or may not become symptomatic. The detection of diverticulosis is generally incidental during a routine examination of the abdomen (eg, during computed tomography [CT]), often performed due to other causes than suspicion of gastrointestinal disease.¹ If detected incidentally, diverticulosis does not require management.

Diverticular disease The term “diverticular disease” describes clinically significant and symptomatic diverticulosis. The term implies that the anatomical lesion (ie, diverticulosis) progresses to a disease state. It entails symptoms such as low-grade inflammation (ie, symptomatic uncomplicated diverticular disease [SUDD]) or acute inflammation of the diverticula (ie, acute diverticulitis), or other less well-understood manifestations (eg, colonic visceral hypersensitivity in the absence of inflammation).

Symptomatic uncomplicated diverticular disease It is estimated that about 15% to 20% of individuals with diverticulosis suffer from SUDD. It is the main subtype of DD, manifested as persistent abdominal symptoms attributed to diverticula but in the absence of macroscopically overt colitis or diverticulitis. This definition is generally uniform across Europe but not in other countries, such as the United States, probably because SUDD is characterized by nonspecific symptoms. In particular, SUDD presents with attacks of abdominal pain without endoscopic or radiologic evidence of diverticular inflammation; it could be colicky in nature but can also last several hours, and it is not relieved by passing flatus or having a bowel movement. Other nonspecific symptoms, ranging from bloating to changes in bowel habits, can also occur due to bacterial overgrowth, and diarrhea seems to be less frequent than constipation. Fullness or tenderness in the left lower abdominal fossa is often detected on physical examination.¹ Finally, these symptoms may occur several times a year. There are reports of patients presenting with these symptoms who suffer from a type of IBS affecting some people with diverticulosis.⁵ Thus, IBS has been recently reported to occur significantly more frequently (4.7-fold) in patients after an episode of acute diverticulitis than in controls.⁶ Several pathophysiological findings may explain the occurrence of symptoms in those patients, including the significant attenuation in serotonin transporter expression,⁷ increased neuropeptide expression in colonic mucosa,⁸ detection of low-grade inflammation,^{9,10} and, finally, elevated levels of fecal calprotectin.¹¹ As a consequence, the current guidelines identify SUDD as a distinct clinical syndrome.¹²

Acute diverticulitis The term “acute diverticulitis” describes the macroscopic inflammation of diverticula. The condition can be uncomplicated or complicated. It is uncomplicated when CT shows

colonic wall thickening with fat stranding, while it is complicated when CT detects features of inflammatory complications (abscess, peritonitis, obstruction, fistulas).¹² The management of acute diverticulitis is associated with significant direct and indirect costs for national health systems.¹³ Even if the disease seems to occur less frequently than previously estimated,¹⁴ it has been shown to relapse more frequently. In fact, a recent study found that the overall disease relapse rate during a 10-year follow-up was up to 40%.¹⁵ According to older guidelines, recurrent episodes (≥ 2 episodes) of acute diverticulitis lead to complicated diverticulitis with increased mortality.¹⁶ However, increased mortality or a higher risk of complicated diverticulitis do not seem to be linked to multiple episodes of diverticulitis, and the recommendation for surgery after at least 2 episodes of recurrent diverticulitis is now questionable.^{12,17} The outcome of patients varies depending on whether the disease is uncomplicated or complicated. The overall mortality rate for patients with uncomplicated acute diverticulitis was 2.5%, which was significantly lower than that for patients with complicated diverticulitis at presentation (10%).¹⁸ In addition, elective sigmoid resection in all patients undergoing elective surgery is associated with a higher risk of colostomy and mortality (14.2% and 2.3%, respectively).¹⁹⁻²¹ Of note, a large majority of patients with perforated diverticulitis (78%) had no history of acute diverticulitis.²²

Risk factors for diverticular disease of the colon

Aging, obesity, and sedentary lifestyle are considered risk factors for diverticular disease. In the present review, we discuss the role of fiber, nut, corn, seed, meat, alcohol, and FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) consumption as a risk factor for diverticulosis as well as the cause of symptom onset and disease progression.

Fiber intake High dietary fiber intake may improve gastrointestinal function and is recommended in patients with diverticulosis and SUDD. Numerous clinicians recommend the use of spasmolytic agents as well as a high-fiber diet or fiber supplementation as the first-line treatment for SUDD in line with current World Gastroenterology Organisation guidelines.²³ However, a recent systematic review did not identify high-quality evidence for a high-fiber diet in the treatment of DD, while it found that most of the current recommendations are based on levels 2 and 3 of evidence (not adequate evidence).²⁴ However, despite this scientific evidence, a high-fiber diet is still recommended. Good-quality studies that investigated fiber consumption in this population are reported in [TABLE 1](#).

Fiber intake and occurrence of diverticulosis Altered fiber intake could underlie the development of diverticulosis. However, it is difficult to identify and investigate the possible link between dietary fiber

TABLE 1 Fibers in diverticulosis and diverticular disease

Study, year	Trial design	No. of patients	Randomization	Outcomes	Length of follow-up	Results
Brodibb et al, 1977 ^a	Double-blind	18	Wheat crispbread, 0.6 g/d vs bran crispbread, 6.7 g/d	Reduction in global symptom score in SUDD	3 mo	Significant reduction in symptom score in high-fiber vs low-fiber group (34.3–8.1 vs 42–35.1, $P < 0.002$)
Ornstein et al, 1981 ^a	Randomized, crossover, double-blind, placebo-controlled	58	Bran (6.99 g/d) vs ispaghula (9.04 g/d) vs placebo (2.34 g/d)	Reduction in global symptom score in SUDD	16 wk	No difference between the 3 arms ($P = \text{NS}$); no difference between bran and ispaghula consumption (5.9 vs 6.7)
Hodgson et al, 1972 ^a	Double-blind, randomized, placebo-controlled	30	Methylcellulose 2 tablets/d vs placebo 2 tablets/d	Reduction in global symptom score in SUDD	3 mo	Reduced symptom score in methylcellulose group (mean [SD], 19 [6] to 13 [4], $P < 0.01$) but not in the placebo group (mean [SD], from 21 [7] to 17 [9], $P = \text{NS}$)
Crowe et al, 2011 ^b	Prospective cohort study	47 033	Vegetarian vs nonvegetarian diet (≥ 25.5 g/d for women and ≥ 26.1 g/d for men) vs lower fiber consumption	Occurrence of DD; hospital admission for DD complications	11.6 y	Vegetarians had a 31% lower risk of DD ($P = 0.001$); patients with high fiber intake had a 26% lower risk of DD ($P = 0.018$); hospital admission or death due to DD was 4.4% for meat eaters and 3% for vegetarians or vegans.
Peery et al, 2012 ^c	Cross-sectional study	2104	Fiber or high-fiber consumption (> 50 g/d) vs normal diet	Occurrence of diverticulosis	12 y	High fiber consumption associated with a higher risk of diverticulosis ($P = 0.004$); soluble fiber associated with a higher risk of diverticulosis ($P = 0.038$)
Strate et al, 2008 ^d	Prospective cohort study	47 228	Lower (less than once per month) vs higher (at least twice per week) nut, corn, or popcorn consumption	Occurrence of diverticulitis and diverticular bleeding	18 y	Higher nut, corn, or popcorn consumption associated with a lower risk of diverticulitis ($P = 0.034$); no difference in diverticular bleeding occurrence between higher or lower consumption of nut, corn, or popcorn ($P = 0.56$, $P = 0.64$, and $P = 0.52$, respectively)
Leahy et al, 1985 ^a	Prospective case-control study	56	Low- (< 25 g/d) vs high- (> 25 g/d) fiber diet	Symptom recurrence; occurrence of complications; surgery due to DD	66 months	High-fiber diet associated with significantly lower symptom recurrence (19.35% vs 44%, $P < 0.05$), occurrence of complications (6.45% vs, 20.25%, $P < 0.05$), and surgery due to DD (6.45% vs 32%) than low-fiber diet

a Data extracted from Carabotti et al²⁴

b Data extracted from Crowe et al⁴⁸

c Data extracted from Peery et al⁴⁹

d Data extracted from Strate et al⁴³

Abbreviations: DD, diverticular disease; SUDD, symptomatic uncomplicated diverticular disease

intake and this condition due to the time required for diverticulosis to develop as well as its asymptomatic course in a large majority of patients.

A recent systematic review of population- and colonoscopy-based studies assessed the role of fiber in diverticulosis.²⁴ The authors reported that preliminary studies conducted in the 1970s found a higher incidence of diverticulosis in the African population, where fiber intake was significantly higher than in the Western population. Moreover, they found that diverticulosis was more frequent in nonvegetarians than in vegetarians (33% vs 12%, $P < 0.01$), and nonvegetarians with or without diverticulosis had similar fiber intake (22.8 vs 22.1 g/d), suggesting that other factors than fiber

intake may be involved in the occurrence of diverticulosis. Colonoscopy-based studies revealed no differences in dietary fiber intake between patients with and without diverticulosis, as assessed by mini dietary assessment index scores.²⁴ These results seem to confirm the findings from a cross-sectional colonoscopy-based study by Peery et al.²⁵ Analyzing data from 539 individuals with diverticulosis and 1569 controls, the authors found that people with less frequent bowel movements (< 7 evacuations/wk) had a lower risk of diverticulosis compared with those with regular bowel movements (7 evacuations/wk) (odds ratio [OR], 0.56; 95% CI, 0.4–0.8). Patients reporting hard stools also had a lower risk (OR, 0.75; 95% CI,

0.55–1.02). Finally, no association was found between diverticulosis and straining (OR, 0.85; 95% CI, 0.59–1.22) or incomplete bowel movements (OR, 0.85; 95% CI, 0.61–1.2).²⁵ These results were confirmed by Japanese investigators. Yamada et al²⁶ performed a cross-sectional colonoscopy-based study including 1066 patients (648 men and 418 women; ratio, 1.55:1; mean [SD] age, 63.9 [13] years). After adjustment for age and sex, the prevalence of diverticulosis was significantly lower in people with constipation than in those with regular bowel habit (OR, 0.7; 95% CI, 0.52–0.93). When assessed according to the location of diverticula, again the prevalence of left-sided diverticulosis was significantly lower in patients with constipation than in those with regular bowel habit (OR, 0.39; 95% CI, 0.16–0.93), but it was higher in patients with right-sided diverticulosis (OR, 1.1; 95% CI, 0.48–2.53). Finally, stool form was not associated with the presence or absence of diverticula.²⁶

Fiber intake and occurrence of diverticular disease
The Million Women Study, a recent large population-based prospective study, investigated the relationship between fiber intake and occurrence of DD in 1.3 million women aged 50 to 65 years, who were invited to attend the National Health Service Breast Screening Programme.²⁷ Using a questionnaire asking about lifestyle factors, the authors found that women taking less than 9.6 g/d of fiber were at higher risk of DD than those taking at least 17.6 g/d of fiber ($P < 0.0001$). Moreover, the risk varied depending on the source of fiber, with differences between the 4 main sources of fiber ($P < 0.0001$). In particular, the relative risk for the occurrence of DD, adjusted for each of the other sources of dietary fiber, was 0.84 (95% CI, 0.81–0.88) per 5 g/d for cereal, 0.81 (95% CI, 0.77–0.86) per 5 g/d for fruit, 1.03 (95% CI, 0.93–1.14) per 5 g/d for vegetables, and 1.04 (95% CI, 1.02–1.07) per 1 g/d for potatoes.²⁷ However, these results were not confirmed by the systematic review by Carabotti et al.²⁴ The benefit of dietary or supplemental fiber in these patients still remains to be established.

Fiber intake and occurrence of acute diverticulitis
Altered fiber intake could also be an important cofactor associated with the occurrence of acute diverticulitis. Recently, a prospective cohort study of 46 295 men who were free of diverticulitis and known diverticulosis in 1986 (baseline) was performed, using data from the Health Professionals Follow-up Study.²⁸ Each study participant completed a detailed medical and dietary questionnaire at baseline, which was resubmitted to men reporting incident diverticulitis on biennial follow-up questionnaires. Patients were differentiated between those with Western dietary pattern (high in red meat, refined grains, and high-fat dairy) and those with prudent pattern (high in fruit, vegetables, and whole grains). During a 14-year follow-up, 1063 incident cases of acute

diverticulitis were identified. After adjustment for other risk factors, a higher Western dietary pattern score was associated with an increased risk of acute diverticulitis ($P = 0.0001$), whereas higher prudent and alternative healthy eating index (AHEI, a score based on expert opinion to represent optimal dietary behavior for disease prevention) dietary pattern scores were associated with reduced risk ($P = 0.001$). Surprisingly, men in the highest quintile of the Western dietary pattern score had a multivariate hazard ratio of 1.55 (95% CI, 1.20–1.99) when compared with men in the lowest quintile, who had a multivariate hazard ratio of 1.28 (95% CI, 0.98–1.68). The corresponding multivariate hazard ratios comparing the extreme quintiles were 0.74 (95% CI, 0.60–0.91) for the prudent dietary pattern and 0.67 (95% CI, 0.55–0.82) for the AHEI pattern.²⁸ Moreover, in older adults with diverticulosis or SUDD, the existing guidelines recommend a high-fiber diet for the long-term primary prevention of diverticulitis based on expert opinion and observational cohort studies.^{29–31} However, these associations have not yet been sufficiently addressed by intervention studies, highlighting the need for more research in this area.³² A recent systematic review and meta-analysis by Eberhardt et al³³ on the role of dietary fiber in older adults identified 7 studies that investigated this dietary management strategy. However, only 1 study measured the effect of fiber intake on the incidence of diverticulitis, and it had a high risk of bias, no comparator group, and was published over 40 years earlier.³⁴

Considering the above data, the effect of a high dietary fiber intake on the prevention of diverticulitis in patients with asymptomatic diverticulosis or SUDD is largely unknown. Available data confirm that in the United States population the Western diet is associated with only a mild risk of acute diverticulitis, and it did not seem to significantly affect the occurrence of diverticulosis. However, several factors may have influenced these results, ranging from self-reported data to an imprecise assessment of dietary intake by the questionnaires provided.²⁹

In a recent systematic review, Dahl et al³⁵ investigated recovery from acute uncomplicated diverticulitis and prevention of its recurrence. However, they found no high-quality intervention research data examining the dietary management of adults with this condition.³⁵ The authors reported low confidence in the evidence that high dietary fiber intake would directly result in a lower risk of diverticulitis recurrence and/or gastrointestinal symptoms, but they also found no evidence supporting the use of a low-fiber diet. A high-fiber diet is recommended by dietary guidelines as a standard diet for all adults; therefore, this recommendation is valid even though there is no strong confidence in an added benefit for diverticulitis-related outcomes. However, the outcomes that could be evaluated by observational and/or lower-quality intervention research suggest that unrestricted and restricted diets are

equal in terms of recovery (both associated with a very low risk and incidence of treatment failure), disease recurrence, and persistence of gastrointestinal symptoms, with a tendency for the unrestricted diet to be used less often in everyday practice.³⁵ The very low quality of the evidence comparing unrestricted and restricted diets demonstrates that there has been no research showing any clinical benefit of implementing a diet restriction. Moreover, no studies supporting the hypothesis that bowel rest is required for resolution of an acute episode in uncomplicated cases have been identified.³⁵

Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols Recently, the adoption of a low-FODMAP diet, containing a low amount of fermentable oligosaccharides, disaccharides, monosaccharides, and polyols, has been proposed as a significant therapeutic tool in the management of patients with IBS due to the limitation or exclusion of gas-producing foods.³⁶

An interesting hypothesis has been recently developed by Uno and van Velkinburgh,³⁷ who claimed that the features of a high-fiber diet represent a logical contradiction for colon diverticulitis. According to the Bernoulli's principle, an enlarged diameter of the lumen results in increased pressure and decreased fluid velocity, which might contribute to the development of the diverticulum. Therefore, theoretically, prevention of high pressure in the colon would be beneficial and adoption of the low-FODMAP diet could help prevent the occurrence or recurrence of acute diverticulitis. As FODMAPs are not digested or absorbed in the small intestine,³⁸ their intake causes increased fluid in the ileum due to the corresponding high osmotic pressure and leads to a large amount of gas produced by fermentation in the colon. The daily adoption of the low-FODMAP diet by patients with IBS led to a significant improvement in symptoms.^{39,40} Since SUDD shares some symptoms with IBS,^{41,42} this therapeutic approach could be effective at least in patients with SUDD in whom bloating and altered bowel habits are the main symptoms. Although all these studies raise important questions about the actual role of fiber in the development of diverticulosis as well as DD and its complications, high-fiber diet and fiber supplementation are still recommended by current guidelines.

Nut, corn, and seed During the last 100 years, the consumption of nuts, corn, and seeds was contraindicated due to a belief that undigested fragments of these food products may lead to diverticular trauma and thus complications of the disease.

In 2008, Strate et al⁴³ assessed 47 000 men followed for 18 years as part of the Health Professionals Follow-up Study. The authors found negative correlations between nut and popcorn consumption and the risk of acute diverticulitis. In the multivariate analysis, the risk of

complications was lower for men with the highest intake (at least twice per week) than for those with the lowest intake (less than once per month) of each food (hazard ratio [HR], 0.8; 95% CI, 0.63–1.01; $P = 0.04$ for nuts and HR, 0.72; 95% CI, 0.56–0.92; $P = 0.007$ for popcorn). Finally, no associations were found between corn consumption and acute diverticulitis or between nut, corn, or popcorn consumption and diverticular bleeding or uncomplicated diverticulosis.⁴³

Alcohol Two case-control studies assessed the role of alcohol and smoking in the occurrence of diverticulosis and DD. Aldoori et al⁴⁴ assessed these associations in a prospective cohort of 47 678 men during a 4-year follow-up study (1988–1992). They recorded 382 new cases of symptomatic DD. After adjustment for age, physical activity, and energy-adjusted intake of dietary fiber and total fat, alcohol intake (assessed by comparing those who drink >30 g/d with nondrinkers) was not associated with the risk of symptomatic DD (relative risk [RR], 1.36; 95% CI, 0.94–1.97; P for trend = 0.37). Any association between caffeine, specific caffeinated beverages, and decaffeinated coffee and the risk of symptomatic DD was also recorded.⁴⁴

More recently, Nagata et al⁴⁵ conducted a prospective cross-sectional colonoscopy-based study on Japanese patients who underwent colonoscopy. Patients were interviewed on the day of colonoscopy about alcohol, alcohol-related flushing, smoking, medications, and comorbidities. Alcohol consumption was defined as nondrinking, light (1–180 g/wk), moderate (181–360 g/wk), and heavy (>361 g/wk) drinking. A univariate analysis found male sex, age, smoking status, alcohol consumption, aspirin, anticoagulant, and corticosteroid use, hypertension, as well as atherosclerotic disease as factors significantly linked to diverticulosis, while alcohol-related flushing was not associated with diverticulosis. A multivariate analysis found increasing age, increasing alcohol consumption, smoking, and atherosclerotic disease as risk factors for diverticulosis (all $P < 0.01$). In addition, alcohol and smoking were associated with right-sided and bilateral diverticula.⁴⁵ More recently, similar findings were reported by Shara et al.⁴⁶ Finally, a recent long-term population-based study (12.5-year follow-up) found that DD was less frequent in people with alcohol dependence than in controls.⁴⁷

Meat consumption It is estimated that the decrease in fiber intake typically seen with industrialization is paralleled by other dietary changes, in particular by a significant increase in meat intake. However, epidemiological studies have provided conflicting results.

Red meat Although a dose-response relationship was not confirmed, a significant association between red meat intake and an increased risk of DD was reported in the EPIC-Oxford study.⁴⁸

TABLE 2 Meat and fish consumption in diverticulosis and diverticular disease

Study	Trial design	No. of patients	Study population	Outcomes	Duration of follow-up	Results
Cao et al ⁵⁰	Prospective cohort study	51 529	Men enrolled in the Health Professionals Follow-up Study	Risk of diverticulitis	1986–2012	Red meat intake, particularly unprocessed red meat, was associated with an increased risk of diverticulitis (multivariable RR, 1.58; 95% CI, 1.19–2.11; <i>P</i> for trend = 0.01). The association was stronger for unprocessed red meat (RR for Q5 vs Q1, 1.51; 95% CI, 1.12–2.03; <i>P</i> for trend = 0.03) than for processed red meat (RR for Q5 vs Q1, 1.03; 95% CI, 0.78–1.35; <i>P</i> for trend = 0.26). Higher consumption of poultry or fish was not associated with the risk of diverticulitis (multivariable RR, 0.80; 95% CI, 0.63–0.99).
Peery et al ⁴⁹	Cross-sectional study	2104	Patients who underwent colonoscopy	Risk for asymptomatic diverticulosis	–	No associations between fat intake (PR, 0.97; 95% CI, 0.84–1.12), red meat intake (PR, 1.04; 95% CI, 0.90–1.19), and diverticulosis when comparing the highest quartile of each exposure to the lowest, after adjustment for potential confounders (eg, age, race, body mass index).
Crowe et al ⁴⁸	Prospective cohort study (EPIC-Oxford)	57 446	Vegetarian vs nonvegetarian diet	Occurrence of diverticular disease; hospital admission for DD complications	11.6 years	The association between the quantity of meat consumed and the risk of diverticular disease was not significant when people with intake of meat lower than 50 g/d were compared with those with the highest intake of meat (≥ 100 g/d); the risk of diverticular disease for those with an intake less than 50 g/d was 0.95 (0.76 to 1.18). The risk was not significantly lower among participants who ate some fish but no meat.

Abbreviations: PR, prevalence ratio; RR, relative risk; Q1, lowest quintile; Q5, highest quintile; others, see [TABLE 1](#)

The study found that red meat eaters had a higher risk of hospitalization for DD than vegetarians, but eaters consuming less than 50 g of meat per day had a surprisingly lower RR of 0.95.⁴⁸ On the contrary, Peery et al⁴⁹ did not find any association between red meat intake and diverticulosis in a colonoscopy-based study. A very recent prospective study assessed the association between the consumption of every type of meat (total red meat, red unprocessed meat, red processed meat, poultry, and fish) and the risk of incident acute diverticulitis.⁵⁰ The authors analyzed 46 461 men enrolled in the Health Professionals Follow-Up Study (from 1986 to 2012) and reported acute diverticulitis in 764 cases (1.64% of the population). Overall, red meat intake was associated with an increased risk of diverticulitis, but this risk increased only when the total red meat consumption was assessed. The risk was higher in men in the highest quintile than in those in the lowest quintile of total red meat consumption (RR, 1.58; 95% CI, 1.19–2.11; *P* for trend = 0.01). Moreover, this risk was nonlinear but plateaued after 6 servings per week (*P* for nonlinearity = 0.002). The authors found that the risk of diverticulitis linked with total red meat intake appeared primarily driven by the consumption of unprocessed red meat (which was defined as the consumption of “beef or lamb as main dish,” “pork as main dish,” “hamburger,” and “beef, pork or lamb as a sandwich or mixed dish”). In fact, the higher risk of acute diverticulitis with meat consumption was found for unprocessed red meat (RR for the highest vs lowest quintile, 1.51; 95% CI, 1.12–2.03;

P for trend = 0.03) than for processed red meat (defined as “bacon,” “beef or pork hot dogs,” “salami, bologna or other processed meat sandwiches,” and “other processed red meats, such as sausage, kielbasa, etc”) ([TABLE 2](#)).⁵⁰ The reason for this finding is unknown, but it is hypothesized that microbial composition, diversity, and richness could be altered by this kind of diet.

Some studies have indicated that certain dietary components, ranging from low fiber to high fat, can cause dysbiosis by decreasing the abundance of beneficial bacteria and promoting the growth of harmful bacteria, leading to an increased intestinal permeability and therefore intestinal inflammation.^{51,52} The role of inflammatory potential of diet was recently investigated by Ma et al.⁵³ They assessed the association between some potential inflammatory foods and chronic inflammation, represented by inflammatory and plasma levels of C-reactive protein and interleukin 6, and subsequent risk of diverticulitis. In a large prospective cohort of men, the authors found 1110 incident cases of acute diverticulitis over 992 589 person-years of follow-up. When they compared men with the lowest quintile of the empiric dietary inflammatory pattern (EDIP) score, after an age-adjusted analyses, those in the highest quintile had a 30% increased risk of acute diverticulitis (hazard ratio [HR], 1.3; 95% CI, 1.08–1.56; *P* for trend = 0.009). The risk did not change even after further adjustment for other lifestyle factors (HR, 1.31; 95% CI, 1.07–1.6; *P* for trend = 0.01). Both fiber and red meat consumption contributed to the EDIP score. In

fact, the authors found that the HRs in men with the highest EDIP quintile was higher than in those with the lowest quintile (HR, 1.23; 95% CI, 1–1.5; *P* for trend = 0.07), suggesting that the association between the EDIP score and acute diverticulitis could be attributed both to fiber and red meat intake. The Western dietary pattern also showed a significant association between the risk of diverticulitis and the EDIP score (HR for the highest vs lowest quintile, 1.27; 95% CI, 1.04–1.55; *P* for trend = 0.02). Finally, the adjustment for body mass index or vigorous physical activity did not change the positive association between the inflammatory potential of diet and risk of acute diverticulitis.⁵³

White meat and fish Cao et al.⁵⁰ have recently assessed the effect of white meat or fish consumption on the occurrence of acute diverticulitis. The risk of acute diverticulitis was not increased by a higher consumption of poultry (considered as white meat) (RR for the highest vs lowest quintile, 1.09; 95% CI, 0.86–1.39; *P* for trend = 0.55). On the other hand, the risk of acute diverticulitis in patients with higher fish intake was significantly reduced in an age-adjusted model but not after further adjustment for other potential confounders (RR for the highest vs lowest quintile: 0.87; 95% CI, 0.68–1.1; *P* for trend = 0.2). Moreover, the authors found that the risk of acute diverticulitis was also decreased after substitution of poultry or fish with one serving of unprocessed red meat per day (multivariable RR, 0.8; 95% CI, 0.63–0.99).⁵⁰

Conclusions There is no clear evidence on the role of low-fiber diet either in preventing the occurrence of diverticulosis and DD or their treatment. There is very low-quality evidence for the recommendation of unrestricted and restricted fiber diets for inpatient management to improve the hospital length of stay, recovery, gastrointestinal symptoms, and prevent disease recurrence after an episode of acute uncomplicated diverticulitis. There is very low-quality evidence for the recommendation of a high-fiber diet as opposed to a standard or low-fiber diet following the resolution of an episode of acute uncomplicated diverticulitis in order to improve gastrointestinal symptoms and prevent disease recurrence. On the contrary, there is some evidence that selected types of meat and higher alcohol consumption increase the risk of diverticulosis and DD. Future studies should investigate the role of new approaches to modifying dietary patterns to prevent acute diverticulitis or at least relieve SIDD symptoms, for example, by providing the low-FODMAP diet.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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REFERENCES

- 1 Tursi A, Papa A, Danese S. Review article: the pathophysiology and medical management of diverticulosis and diverticular disease of the colon. *Aliment Pharmacol Ther.* 2015; 42: 664-684. [↗](#)
- 2 Tursi A. Diverticulosis today: unfashionable and still under-researched. *Therap Adv Gastroenterol.* 2016; 9: 213-228. [↗](#)
- 3 Kupcinkas J, Strate LL, Bassotti G, et al. Pathogenesis of diverticulosis and diverticular disease. *J Gastrointest Liver Dis.* 2019; 28: 7-10. [↗](#)
- 4 Weizman AV, Nguyen GC. Diverticular disease: epidemiology and management. *Can J Gastroenterol.* 2011; 25: 295-302. [↗](#)
- 5 Yamada E, Inamori M, Uchida E, et al. Association between the location of diverticular disease and the irritable bowel syndrome: a multicenter study in Japan. *Am J Gastroenterol.* 2014; 109: 1900-1905. [↗](#)
- 6 Cohen E, Fuller G, Bolus R, et al. Increased risk for irritable bowel syndrome after acute diverticulitis. *Clin Gastroenterol Hepatol.* 2013; 11: 1614-1619. [↗](#)
- 7 Aldoori WH, Giovannucci EL, Rimm EB, et al. Prospective study of diet and the risk of symptomatic diverticular disease in men. *Am J Clin Nutr.* 1994; 60: 757-764. [↗](#)
- 8 Costedio MM, Coates MD, Danielson AB, et al. Serotonin signaling in diverticular disease. *J Gastrointest Surg.* 2008; 12: 1439-1445. [↗](#)
- 9 Humes DJ, Simpson J, Smith J, et al. Visceral hypersensitivity in symptomatic diverticular disease and the role of neuropeptides and low grade inflammation. *Neurogastroenterol Motil.* 2012; 24: 318-e163. [↗](#)
- 10 Tursi A, Elisei W, Giorgetti GM, et al. Detection of endoscopic and histological inflammation after an attack of colonic diverticulitis is associated with higher diverticulitis recurrence. *J Gastrointest Liver Dis.* 2013; 22: 13-19.
- 11 Tursi A, Brandimarte G, Elisei W, et al. Faecal calprotectin in colonic diverticular disease: a case-control study. *Int J Colorectal Dis.* 2009; 24: 49-55. [↗](#)
- 12 Tursi A, Brandimarte G, Di Mario F, et al. International Consensus on Diverticulosis and Diverticular Disease. Statements from the 3rd International Symposium on Diverticular Disease. *J Gastrointest Liver Dis.* 2019; 28: 57-66.
- 13 Sandler RS, Everhart JE, Donowitz M, et al. The burden of selected digestive disease in the United States. *Gastroenterology.* 2002; 122: 1500-1511. [↗](#)
- 14 Shahedi K, Fuller G, Bolus R, et al. Long-term risk of acute diverticulitis among patients with incidental diverticulosis found during colonoscopy. *Clin Gastroenterol Hepatol.* 2013; 11: 1609-1613. [↗](#)
- 15 Lahat A, Avidan B, Sakhnini E, et al. Acute diverticulitis: a decade of prospective follow-up. *J Clin Gastroenterol.* 2013; 47: 415-419. [↗](#)
- 16 Stollman N, Raskin JB. Diagnosis and management of diverticular disease of the colon in adults: Ad Hoc Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol.* 1999; 94: 3110-3121. [↗](#)
- 17 Stollman N, Smalley W, Hirano I, AGA Institute Clinical Guidelines Committee. American Gastroenterological Association Institute Guideline on the management of acute diverticulitis. *Gastroenterology.* 2015; 149: 1944-1949. [↗](#)
- 18 Chautems R, Ambrosetti P, Ludwig A, et al. Long term follow-up after first acute episode of sigmoid diverticulitis: is surgery mandatory? A prospective study of 118 patients. *Dis Colon Rectum.* 2002; 45: 962-966. [↗](#)
- 19 Hart AR, Kennedy HJ, Stebbings WS, Day NE. How frequently do large bowel diverticula perforate? An incidence and cross-sectional study. *Eur J Gastroenterol Hepatol.* 2000; 12: 661-665. [↗](#)
- 20 Richards R, Hammitt JK. Timing of prophylactic surgery in prevention of diverticulitis recurrence: a cost-effectiveness analysis. *Dig Dis Sci.* 2002; 47: 1903-1908.
- 21 Salem L, Veenstra DL, Sullivan SD, Flum DR. The timing of elective colectomy in diverticulitis: a decision analysis. *J Am Coll Surg.* 2004; 199: 904-912. [↗](#)
- 22 Chapman J, Dozois EJ, Wolff BG, et al. Diverticulitis: a progressive disease? Do multiple recurrences predict less favourable outcomes? *Ann Surg.* 2006; 243: 876-880. [↗](#)
- 23 World Gastroenterology Organisation. World Gastroenterology Organisation practice guidelines: diverticular disease. <https://www.worldgastroenterology.org/guidelines/global-guidelines/diverticular-disease/diverticular-disease-english>. Accessed January 21, 2020.
- 24 Carabotti M, Annibale B, Severi C, Lahner E. Role of Fiber in symptomatic uncomplicated diverticular disease: a systematic review. *Nutrients.* 2017; 9: E161. [↗](#)

- 25 Peery AF, Sandler RS, Ahnen DJ, et al. Constipation and a low-fiber diet are not associated with diverticulosis. *Clin Gastroenterol Hepatol*. 2013; 11: 1622-1627. [↗](#)
- 26 Yamada E, Inamori M, Watanabe S, et al. Constipation is not associated with colonic diverticula: a multicenter study in Japan. *Neurogastroenterol Motil*. 2015; 27: 333-338. [↗](#)
- 27 Crowe FL, Balkwill A, Cairns BJ, et al; Million Women Study Collaborators. Source of dietary fibre and diverticular disease incidence: a prospective study of UK women. *Gut*. 2014; 63: 1450-1456. [↗](#)
- 28 Strate LL, Keeley BR, Cao Y, et al. Western dietary pattern increases, and prudent dietary pattern decreases, risk of incident diverticulitis in a prospective cohort study. *Gastroenterology*. 2017; 152: 1023-1030. [↗](#)
- 29 Köhler L, Sauerland S, Neugebauer E. Diagnosis and treatment of diverticular disease: results of a consensus development conference. The Scientific Committee of the European Association for Endoscopic Surgery. *Surg Endosc*. 1999; 13: 430-436.
- 30 Feingold D, Steele SR, Lee S, et al. Practice parameters for the treatment of sigmoid diverticulitis. *Dis Colon Rectum*. 2014; 57: 284-294. [↗](#)
- 31 O'Leary D, Lynch N, Clancy C, et al. International, expert-based, consensus statement regarding the management of acute diverticulitis. *JAMA Surg*. 2015; 150: 899-904. [↗](#)
- 32 Mahmood MW, Abraham-Nordling M, Håkansson N, et al. High intake of dietary fibre from fruit and vegetables reduces the risk of hospitalisation for diverticular disease. *Eur J Nutr*. 2019; 58: 2393-2400. [↗](#)
- 33 Eberhardt F, Crichton M, Dahl C, et al. Role of dietary fibre in older adults with asymptomatic (AS) or symptomatic uncomplicated diverticular disease (SUDD): systematic review and metaanalysis. *Maturitas*. 2019; 130: 57-67. [↗](#)
- 34 Painter NS, Almeida AZ, Colebourne KW. Unprocessed bran in treatment of diverticular disease of the colon. *Br Med J*. 1972; 2: 137-140. [↗](#)
- 35 Dahl C, Crichton M, Jenkins J, et al. Evidence for dietary fibre modification in the recovery and prevention of recurrence of acute, uncomplicated diverticulitis: a systematic literature review. *Nutrients*. 2018; 10: E137. [↗](#)
- 36 Portincasa P, Bonfrate L, de Bari O, et al. Irritable bowel syndrome and diet. *Gastroenterol Rep (Oxf)*. 2017; 5: 11-19. [↗](#)
- 37 Uno Y, van Velkinburgh JC. Logical hypothesis: low FODMAP diet to prevent diverticulitis. *World J Gastrointest Pharmacol Ther*. 2016; 7: 503-512. [↗](#)
- 38 Gibson PR, Shepherd SJ. Food choice as a key management strategy for functional gastrointestinal symptoms. *Am J Gastroenterol*. 2012; 107: 657-666. [↗](#)
- 39 Halmos EP, Power VA, Shepherd SJ, et al. A diet low in FODMAPs reduces symptoms of irritable bowel syndrome. *Gastroenterology*. 2014; 146: 67-75. [↗](#)
- 40 Chumritazi BP, Cope JL, Hollister EB, et al. Randomised clinical trial: gut microbiome biomarkers are associated with clinical response to a low FODMAP diet in children with the irritable bowel syndrome. *Aliment Pharmacol Ther*. 2015; 42: 418-427. [↗](#)
- 41 Cuomo R, Barbara G, Andreati P, et al. Symptom patterns can distinguish diverticular disease from irritable bowel syndrome. *Eur J Clin Invest*. 2013; 43: 1147-1155. [↗](#)
- 42 Tursi A, Elisei W, Picchio M, et al. Moderate-to-severe and prolonged left lower abdominal pain is the best symptom characterizing symptomatic uncomplicated diverticular disease of the colon: a comparison with fecal calprotectin in clinical setting. *J Clin Gastroenterol*. 2015; 49: 218-221. [↗](#)
- 43 Strate LL, Liu YL, Syngal S, et al. Nut, corn, and popcorn consumption and the incidence of diverticular disease. *JAMA*. 2008; 300: 907-914. [↗](#)
- 44 Aldoori WH, Giovannucci EL, Rimm EB, et al. A prospective study of alcohol, smoking, caffeine, and the risk of symptomatic diverticular disease in men. *Ann Epidemiol*. 1995; 5: 221-228. [↗](#)
- 45 Nagata N, Niikura R, Shimbo T, et al. Alcohol and smoking affect risk of uncomplicated colonic diverticulosis in Japan. *PLoS One*. 2013; 8: e81137. [↗](#)
- 46 Sharara AI, El-Halabi MM, Mansour NM, et al. Alcohol consumption is a risk factor for colonic diverticulosis. *J Clin Gastroenterol*. 2013; 47: 420-425. [↗](#)
- 47 Schoepf D, Heun R. Alcohol dependence and physical comorbidity: increased prevalence but reduced relevance of individual comorbidities for hospital-based mortality during a 12.5-year observation period in general hospital admissions in urban North-West England. *Eur Psychiatry*. 2015; 30: 459-468. [↗](#)
- 48 Crowe FL, Appleby PN, Allen NE, Key TJ. Diet and risk of diverticular disease in Oxford cohort of European Prospective Investigation into Cancer and Nutrition (EPIC): prospective study of British vegetarians and non-vegetarians. *BMJ*. 2011; 343: d4131. [↗](#)
- 49 Peery AF, Barrett PR, Park D et al. A high-fiber diet does not protect against asymptomatic diverticulosis. *Gastroenterology*. 2012; 142: 266-272. [↗](#)
- 50 Cao Y, Strate LL, Keeley BR, et al. Meat intake and risk of diverticulitis among men. *Gut*. 2018; 67: 466-472. [↗](#)
- 51 Schroeder BO, Birchenough GMH, Stahlman M, et al. Bifidobacteria or fiber protects against diet-induced microbiotamediated colonic mucus deterioration. *Cell Host Microbe*. 2018; 23: 27-40. [↗](#)
- 52 Wan Y, Wang F, Yuan J, et al. Effects of dietary fat on gut microbiota and faecal metabolites, and their relationship with cardiometabolic risk factors: a 6-month randomised controlled-feeding trial. *Gut*. 2019; 68: 1417-1429. [↗](#)
- 53 Ma W, Jovani M, Nguyen LH, et al. Association between inflammatory diets, circulating markers of inflammation, and risk of diverticulitis. *Clin Gastroenterol Hepatol*. 2019. [Epub ahead of print]. [↗](#)