




Diverticulitis—new evidence to share with patients

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Abstract

Diverticulitis is one of the most common gastrointestinal causes of hospitalization in Western society. While previously characterized as a disease of older patients, new literature highlights an increasing incidence among the younger population. Over the past few decades, the understanding of etiology and management of diverticulitis has changed drastically. New data refute past beliefs while promoting other novel recommendations to mitigate incidence and subsequent complications. Data now confirms the safety and possible protective benefit of particulate food, while highlighting evidence-based approaches for the use of diagnostic imaging and antibiotics. We recognize modifiable and non-modifiable risk factors that are commonly seen throughout the literature and play a significant role in the management and prevention of diverticulitis. Emerging evidence also links chronic inflammation with subsequent microbial dysbiosis and alterations in the neuroendocrine system, leading to visceral hypersensitivity and perturbation of the gut-brain axis. This review provides a comprehensive update on acute uncomplicated diverticulitis according to the most recent evidence-based literature, encompassing the risks, diagnostic modalities, and management treatment regimens.

Keywords

diverticulitis, NSAIDs, dysbiosis, vitamin D deficiency, modifiable risk, non-modifiable risk, visceral hypersensitivity

Introduction

The most recent evidence has changed the approach to diverticular disease, both prophylactically and in its medical management. Diverticulosis is defined by the presence of many diverticula, which are pouch-like externally directed openings in the colonic wall. Diverticulitis occurs when one or more adjacent diverticula become inflamed. About 85% of diverticulitis cases are considered uncomplicated and may present clinically with a change in bowel habits, low-grade fevers, and left-sided abdominal pain, with the exception

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being patients from Asian countries who largely experience right-sided pain [1, 2]. The remainder of patients experience a disease course complicated by abscess, free perforation, phlegmon, stricture, or fistula formation, constituting complicated diverticulitis [1, 3]. Many patients presenting with complicated diverticulitis will often require surgical intervention, although this number has been decreasing as our imaging and antibiotic capabilities have improved [4].

Diverticular disease is quite common in Western and industrialized societies, representing the sixth most common gastrointestinal (GI) cause of hospitalization [1]. Over half of the population aged 60 and older is reported to have a diagnosis of diverticulosis, with that number reaching over 70% at age 80 [5, 6]. Four percent of those affected are estimated to experience an episode of diverticulitis in their lifetime. Interestingly, Peery et al. [7] noted a 132% increase in incident cases of diverticulitis in patients aged 40–49 from 1980 to 2007, suggesting diverticulitis may no longer be considered a disease primarily encountered in the elderly. They also found that following an initial complicated event, the risk of most complications decreases with recurrent events. The risk of recurrence overall, however, increases with each subsequent episode [7]. Given how common diverticular disease is in the population, as well as the increasing incidence in the younger population, it is imperative to stay at the forefront of new evidence to prevent disease or mitigate debilitating complications in those affected. This article will review emerging evidence regarding the pathophysiology and risk factors of diverticulitis, as well as its diagnosis and treatment.

Epidemiology and pathophysiology

Rethinking dietary causes

The exact etiology of diverticulitis is unknown. Dietary intake, however, appears to play a significant role in both its prevention and provocation. Over time, as theories have evolved from new data, so have clinic recommendations. Decades ago, the idea that particulate food matter, such as corn, seeds, and nuts, could cause obstruction or mechanical damage to the mucosa of the colon led to the recommendation against eating such foods. This belief went unchallenged until 2008, when a large prospective cohort study, consisting of only men, showed no association of this diet with incident diverticulitis or diverticular bleeding [8]. On the contrary, due to their anti-inflammatory benefits, consumption of nuts was found to be inversely related to levels of inflammatory markers often studied alongside diverticulitis, including interleukin (IL)-6 and C-reactive protein (CRP), suggesting a possible protective role. Nuts have also been shown to confer some protective benefits in other inflammatory diseases such as cardiovascular disease and type 2 diabetes [9, 10]. Recently, a similar prospective cohort study consisting of only women, with a total of 415,103 person-years of follow-up, also did not find an association between nuts, seeds, and corn and incident diverticulitis risk [11]. Interestingly, they also looked at diet quality and its relationship to incident diverticulitis risk, suggesting a reduced risk of diverticulitis with improved dietary habits [11]. Additionally, diets lower in red meat and higher in fiber were associated with a decreased risk of diverticulitis [12]. Overall, the literature suggests that healthier diets confer a lower inflammatory burden on the body, leading to decreased incidence and more favorable outcomes in diverticulitis.

Microbiome

Evidence continues to mount that the gut microbiome is a critical facet of all GI conditions. Alterations in the gut microbiome, called dysbiosis, have demonstrated clear links with other conditions of chronic low-grade inflammation, such as inflammatory bowel disease (IBD) and cirrhosis [13, 14]. The gut microbiome is a well-regulated environment composed mainly of two different phyla, Bacteroidetes and Firmicutes. Linking diverticulitis to dysbiosis, a small proof-of-concept study found that patients with incident acute diverticulitis, compared to controls, had significantly altered microbial diversity, namely within the Proteobacteria phylum [15]. In a larger nested case-control study from the population of women in the Nurses' Health Study II, significant differences between microbial community structure and metabolomic profiles were noted between patients with diverticulitis and controls, particularly the presence of enriched pro-inflammatory species [16].

It has also been shown that a favorable microbiome promotes a reduction in inflammation measured by decreased inflammatory markers, such as IL-6, CRP, and tumor necrosis factor (TNF)- α [17]. In a small double-blind placebo-controlled randomized controlled trial among patients with acute uncomplicated diverticulitis (AUD), patients treated with probiotics and antibiotics compared to antibiotics alone had decreased abdominal pain and shorter hospitalization time [18]. The authors postulate this is due to probiotic mediation of pro- and anti-inflammatory IL and TNF [18]. Given these data and the pro-inflammatory environment harbored by gut dysbiosis, the gut microbiota is a potential future therapeutic target in the treatment and prevention of diverticulitis.

Modifiable risk factors for recurrent diverticulitis

Three broad categories of modifiable risk factors for diverticulitis have been suggested: diet, lifestyle, and medications (Table 1) [7]. The rate of recurrence of diverticulitis associated with these risk factors is high and one that warrants further discussion. Surprisingly, retrospective analysis suggests a 22% chance of the first recurrent event within 10 years following the incident event [19]. Furthermore, the long-term risk of recurrence increases with subsequent episodes; the cumulative incidence of recurrence increases to 19% at 1 year after the first recurrence and then to 24% at 1 year after the second recurrence [19]. When stratified by demographics, the risk of recurrence is higher in patients under the age of 50 and in women. While the incidence of diverticulitis had been consistent between 1980 and 1997, a marked increase in incidence was seen between 2000 and 2007 in patients under the age of 50. Although this number can partially be attributed to increased use of computed tomography (CT) during this time, it is also potentially reflective of the poor changes in lifestyle shown to contribute unfavorably to the incidence and recurrence of diverticulitis [19].

Table 1. Modifiable and non-modifiable risk factors for diverticulitis.

Classification of risk factors	Items
Modifiable risk factors	Diet Obesity Physical activity Smoking Use of NSAIDs Alcohol intake
Non-modifiable risk factors	Age Sex Genetics

NSAIDs: non-steroidal anti-inflammatory drugs.

Tobacco smoking and alcohol consumption

Tobacco and alcohol use are closely linked social behaviors and for decades have been shown to be associated with an increased risk of diverticular disease. Older literature on smoking highlights an increased risk of diverticulitis incidence, complications, severity of disease, as well as increased risk of colectomy [20, 21]. There is, however, a paucity of literature about the relationship between smoking and its influence on the recurrence of diverticulitis episodes. Two studies have identified a positive relationship between smoking and recurrence, although the sample sizes are relatively small and their significances are weak [22, 23]. Interestingly, one common trend seen throughout most of the smoking literature is the relationship between any smoking history, current or former, and increased risk of diverticulitis that is dose-independent.

Since the exact mechanism for diverticular disease is currently unknown and is likely multifactorial, many studies have postulated ways in which smoking disrupts the body's natural defenses and potentially contributes to an increased risk of disease. Smoking has effects on vaso-intestinal polypeptide (VIP), which affects motility and intraluminal pressure. Increased intraluminal pressure is often hypothesized to

eventually lead to the formation of diverticula at weak points in the colonic wall, therefore inciting disease [4, 22]. Other effects include reducing colonic mucus production, damaging endothelial function and therefore blood supply to the colon, and disrupting the gut microbiome, leading to dysbiosis [20]. Another indirect relationship between smoking and diverticulitis risk is its association with increased central adiposity, which is a risk factor for diverticular disease by itself [24].

Alcohol consumption has also been associated with increased risk for diverticulosis and diverticulitis [25–28]. Recent evidence in a large prospective study including 84,232 women from the Nurses' Health Study II showed that alcohol is associated with diverticulitis in a more dose-dependent fashion, with the greatest risk seen in women who consume two or more alcoholic beverages per day [28]. One unique finding in this study was that when alcohol use was stratified by type, individuals who predominantly consumed liquor were found to have a positive association with risk of diverticulitis when compared to those who predominantly consumed red wine. Considering the association between systemic inflammation and risk of incident diverticulitis [29], as well as the antioxidant and anti-inflammatory properties native to red wine (i.e., polyphenols) [30, 31], these findings highlight a potential avenue for further exploration and may help clinicians provide more nuanced lifestyle guidance. Finally, when controlling for other variables including lifestyle, diet, and medications, they analyzed the combined risk of smokers and those who consumed alcohol; the largest risk for diverticulitis was observed between smokers (current or past) and those who consumed more than 1 alcoholic beverage per day [multivariate hazard ratio (HR), 1.60; 95% CI, 1.16–2.21] [28].

There are also many ways in which alcohol can affect the gut and the body's natural defenses that may lead to the observed increased risk. The authors attribute this risk to the disruption in prostaglandin synthesis, which would decrease protection for the mucosal barrier, compromise its integrity, and ultimately promote translocation of bacteria from the gut into the blood. This is one known mechanism of propagation for a chronic systemic inflammatory state [14]. They also suggest injury may be driven by the production of advanced glycation end products, which are known proinflammatory agents associated with intestinal epithelial damage, reduced microbial diversity, and dysfunction of the gut barrier [32].

Obesity and physical activity

Obesity represents another prominent modifiable lifestyle factor strongly associated with diverticulitis. When followed prospectively for 18 years, men with a body mass index (BMI) ≥ 30 kg/m² had a significantly elevated risk for diverticulitis compared to men with a BMI < 21 kg/m² [33]. Furthermore, using waist circumference and waist-to-hip ratio as measures of central obesity, men in the highest quintiles were found to have significantly higher risk for diverticulitis compared to those in the lowest quintiles, with multivariate relative risks of 1.56 (95% CI, 1.18–2.07; $P = 0.002$) and 1.62 (95% CI, 1.23–2.14; $P = 0.0004$), respectively [33]. Extending these findings, a large prospective cohort study involving 46,079 women from the Nurses' Health Study found that adulthood weight gain of ≥ 20 kg had a 73% increased risk of developing diverticulitis compared to women who maintained weight from age 18 to present [34]. The biological basis for this relationship may lie in the pro-inflammatory properties of visceral fat, which is metabolically active tissue that secretes a variety of adipokines and produces a state of systemic inflammation [35]. These gross changes, which are implicated in the development of insulin resistance and diabetes, may also contribute to the pathogenesis of diverticulitis.

Given the data on obesity, it is reasonable to assume that physical activity level might then play a role in risk. Indeed, studies have identified activity level as a contributor [36, 37]. A difference between sexes was also identified, with risk as an inverse linear correlation with the level of exercise identified in females, compared with the risk in men being significantly reduced with only vigorous exercise [36]. Research has also shown that regular exercise reduces systemic inflammation [38]. With chronic systemic inflammation as a likely major driver for diverticulitis, exercise is a crucial point for counseling, especially as the sedentary lifestyle becomes more commonplace in Western societies and contributes to many other metabolic diseases.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Regular use of NSAIDs has been shown to increase the risk of diverticulitis through disruption of prostaglandin synthesis and direct topical injury, ultimately increasing mucosal permeability as well as impairing mucosal defense [39, 40]. In the context of diverticular bleeding and complicated diverticulitis (i.e., perforation, abscess), NSAID use has been shown to significantly increase risk in both case-control and cohort studies [41–43]. When stratified by NSAID type, regular use (i.e., at least 2 times per week) of non-aspirin NSAIDs demonstrated a stronger association with incident diverticulitis than aspirin [44, 45]. This difference in risk was thought to reflect aspirin's poor colonic penetration and subsequent reduction in topical efficacy, considering it is primarily absorbed in the stomach and duodenum [39]. Peery et al. [7] recommend avoiding the use of NSAIDs, except aspirin, when prescribed for secondary prevention of cardiovascular disease, to reduce the risk of diverticulitis. While there does not appear to be literature directly linking diverticulitis recurrence to NSAID use, it is reasonable to assume continued use of these medications will continue to disrupt the mucosal barriers and further contribute to the ongoing low-grade inflammatory state, predisposing individuals to subsequent episodes.

Non-modifiable risk factors

With mounting evidence for modifiable risk factors and their role in the development of diverticulitis, age, sex, and the importance of genetic predisposition also must be addressed. As we have seen, the prevalence of diverticulitis increases as we age, with rates similar between males and females [46]. As for genetics, population-based studies with twins and siblings have found that the relative risk of diverticulitis in siblings of index cases was roughly threefold higher than the general population [47]. Furthermore, the risk of diverticular disease in monozygotic twins was nearly triple that of dizygotic twins [47]. Delving deeper into which genetic markers may be implicated, one of the largest genome-wide association studies (GWAS) on diverticulitis identified 42 loci associated with diverticular disease; 39 of those loci were novel at the time [48]. Genes in these loci are linked to intestinal motility, cellular adhesion, and immunity; functions that have all been hypothesized to contribute to the etiopathogenesis of diverticulitis. Five years after the discovery of these loci, the same group developed a polygenic risk score (PRS) tool specific to diverticulitis, which combines the risk that each single nucleotide polymorphism (SNP) represents and provides insight into an individual's specific risk of developing the disease. They found that incidence and severity were associated with higher predicted risk based on the PRS [49].

Recently, another group analyzed the combination of the PRS and environmental risk factors in determining the risk of development of diverticulitis. They developed a healthy lifestyle score (scale of 0–5), which combined commonly studied modifiable risk factors: smoking, BMI, physical activity, fiber intake, and intake of red meat [36]. A higher score indicated a healthier lifestyle. They found that a higher healthy lifestyle score was associated with a reduced risk of diverticulitis, with the highest score of 5 being associated with as much as a 50% reduction in risk of acute disease when compared to those with a score of 0 [36]. Interestingly, they also concluded that the additive risk when genetic and environmental risks are both present is significantly greater than when compared to the sum of each risk considered independently [36]. Altogether, these findings underscore the advent of personalized medicine and of one day routinely incorporating family history as well as individual genetic risk into discussions of prophylaxis and potential treatment options, especially in high-risk groups.

Diagnosis and evaluation

Clinical presentation

The clinical presentation and initial management of acute diverticulitis are varied but principally depend on initial severity (uncomplicated vs. complicated), location of presentation (outpatient vs. emergency department), patient comorbidities, age, and availability of resources. Therefore, each case must be treated uniquely. Suspected AUD, consisting of left-sided abdominal pain, a change in bowel habits, low-grade fevers, and no red flag symptoms, in the outpatient setting in a patient with known diverticular disease,

may be managed conservatively with bowel rest and symptom management. When the patient presents to the emergency department with more severe illness, including signs of sepsis, high-grade fevers, peritonitis, fecaluria, and/or highly elevated CRP value > 150 mg/L, the clinician must consider hospital admission and the need for antibiotics, imaging, and surgical evaluation [4]. These objective markers of inflammation and immunonutrition status have also shown potential in their ability to prognosticate the clinical course of diverticulitis [50–52]. Recently, Barlas et al. [53] found that the hemoglobin, albumin, lymphocyte, and platelet (HALP) score—an index that integrates HALP levels—was significantly higher in patients diagnosed with complicated diverticulitis compared to those with uncomplicated diverticulitis. Furthermore, when stratified into two groups according to a defined cut-off value, those who had a low HALP score had a sixfold higher risk of complications than those who scored higher [53].

Clinical presentation may also differ on account of patient age. Given the increasing incidence of diverticulitis across all age groups [54], Han et al. [55] evaluated the disease profile of colonic diverticulitis in children and adolescents relative to adult disease. They found that colonic diverticulitis within this age group was predominantly localized to the cecum or ascending colon, presenting as a solitary lesion [55]. Furthermore, clinical presentation was less severe, with approximately 59% of patients classified as modified Hinchey stage 0, characterized by mild symptoms and CT findings demonstrative of diverticula with or without colonic wall thickening [55].

Imaging

Our imaging capabilities have greatly improved over the past few decades, becoming both more readily available and more accurate. In confirming the diagnosis of diverticulitis, CT has been the gold standard, with a sensitivity and specificity of 98% and 99%, respectively [56]. Other imaging modalities, namely magnetic resonance imaging (MRI) and ultrasound (US), can also be used; however, are not as accurate. MRI is limited in its ability to identify extraluminal air, and US can be very user-dependent. The US should be used primarily to rule out other causes of abdominal pain instead of diagnosing diverticulitis [57].

The most recent guidelines from the American College of Physicians (ACP) suggest abdominal CT imaging when diagnostic uncertainty remains after a detailed history and physical exam [2]. The American Gastroenterological Association (AGA) echoes this recommendation in their guidelines, especially in patients without previous image-confirmed disease, and to evaluate for signs of complicated diverticulitis [7]. They also cite the accuracy of diagnosis solely by clinical suspicion as 40–65%, which inherently supports their recommendation of routine imaging except in very typical, uncomplicated outpatient cases lacking alarm symptoms who present with recurrent episodes [7]. Both guidelines, as well as other literature, support imaging or re-imaging when the diagnosis is unclear, patient comorbidities are too great, the patient is immunocompromised, there is worsening of symptoms to suggest progression to complicated disease (i.e., abscess formation), or symptoms have not improved after five days of treatment [2, 7, 56].

Moreover, since CT imaging has become much more feasible, one must seriously consider its necessity according to the present guidelines and the risks to patient health over time. Ordering CT scans when not indicated unnecessarily increases the patient's exposure to ionizing radiation, which is identified as a carcinogen. Although each scan provides only a small amount of radiation, the risk of cancer in this setting is thought to be from cumulative radiation exposure. Surprisingly, since the use of CT has increased by about 30% since 2007, CT imaging now represents a more prominent risk factor for future cancer, with potentially up to 5% of all new cancer diagnoses attributed to CT scanning [58]. This shocking projection emphasizes the importance of triaging those who need imaging versus a clear diagnosis without red flag signs and symptoms.

Management and treatment

Antibiotic stewardship

The initial belief that diverticulitis was due to an infectious process led to the routine administration of antibiotics in the treatment of AUD and complicated diverticulitis. However, data have demonstrated that the use of antibiotics as part of AUD treatment does not necessarily provide a benefit [59–61]. In fact, when not indicated, antibiotics may cause unnecessary side effects and will contribute to increasing rates of antibiotic resistance. A recent systematic review and meta-analysis, including 2,505 patients with AUD from nine studies, showed shorter hospitalizations for patients treated without antibiotics. Importantly, there was no significant difference between groups in the need for additional treatment or intervention during the initial episode, rate of readmission, need for surgical or radiological intervention, or recurrence of diverticulitis [59]. These data effectively refute the long-held belief that treating AUD requires antibiotics and allow a shift in clinical practice to a more selective, patient-specific approach.

Antibiotics, however, are indicated in complicated diverticulitis and certain situations of AUD, such as patients who are immunocompromised, frail, have many comorbidities, have refractory symptoms or vomiting, or have elevated lab values, including white blood cell count $> 15 \times 10^9$ cells per liter and/or CRP > 140 mg/L [7]. Also, other risk factors for progression to complicated diverticulitis, including fluid collection or longer segments of inflammation seen on CT, are also indications to initiate antibiotic treatment. When indicated, antibiotics normally include broad-spectrum coverage for gram-negative bacteria and anaerobes in either oral or intravenous forms [7].

Post-diverticulitis colonoscopy

Historically, routine colonoscopy was recommended 6–8 weeks after resolution of an episode of diverticulitis to exclude underlying malignancy, regardless of disease severity or colorectal cancer (CRC) screening history. This practice was largely due to a lack of specificity in diagnosis at that time and the ability to rule out potential malignancy as a cause for disease [62]. More recently, as imaging techniques have advanced, our diagnostic accuracy has also improved, challenging this long-held practice. Current data suggest that the prevalence of CRC in patients with diverticulitis is low. Indeed, in a meta-analysis of eleven studies across 7 countries, Sharma et al. [62] found the number of patients with malignancy was only 22 out of a pooled population of 1,970 patients. When these data were stratified by severity, 5 out of 1,497 patients with uncomplicated diverticulitis were found to have cancer compared to 6 out of 79 patients with complicated diverticulitis [62]. In a similar meta-analysis, the pooled prevalence of CRC in patients with acute diverticulitis was also found to be low at 1.9%; the cancer risk being greater in patients with complicated disease (7.9%) versus uncomplicated (1.3%) [63]. As such, many medical societies have since revised this practice to recommend against routine colonic evaluation in certain circumstances (Table 2) [2, 7, 64–67]. Patients who have CT-confirmed diverticular disease that is benign, uncomplicated, and successfully treated do not require follow-up colonoscopy if they have had a recent high-quality exam (~1–3 years). Complicated cases presenting with alarm features (systemic symptoms, worsening disease course, rectal bleeding, abscess, etc.) that are managed non-operatively, or those which are uncomplicated but lack diagnostic certainty, are recommended to undergo follow-up colonoscopy [2, 7, 64–67]. The widely accepted recommended timeframe for follow-up is about 6–8 weeks following resolution of symptoms due to the theoretical increased risk for perforation, a more technically difficult exam, and increased patient discomfort [7]. Therefore, just as is the case for antibiotic use, each management plan must be tailored to the patient and their history, so they are not unnecessarily exposed to invasive procedures that carry their own inherent risks.

Visceral hypersensitivity

Upon resolution of an acute diverticulitis episode or in the presence of smoldering disease, there may be lingering or intermittent abdominal pain due to lowered pain thresholds referred to as visceral hypersensitivity [68]. Clinically, this presents as abdominal pain without evidence of inflammation on imaging or endoscopy. Available studies have investigated the presence and impact of inflammation on the

Table 2. Recommendations by society on the indication of colonoscopy after uncomplicated and complicated diverticulitis episodes [2, 7, 64–67].

Society	Colonoscopy indicated after an uncomplicated diverticulitis?	Colonoscopy indicated after complicated diverticulitis?	Time frame for follow-up colonoscopy
American Gastroenterological Association (AGA) 2020	Yes; however, it may be deferred if the first episode and a high-quality exam within the past year	Yes	Minimum 6–8 weeks or until complete resolution of symptoms, whichever is longer
American College of Physicians (ACP) 2022	Not indicated for those with up-to-date recommended CRC screening	Yes, in those with diagnostic uncertainty in ruling out CRC	Minimum 6–8 weeks after complete resolution of acute symptoms
American Society of Colon and Rectal Surgeons (ASCRS) 2020	Not indicated unless atypical imaging, symptoms, or recovery ensues	Yes, if a colonoscopy has not been performed recently	About 6 weeks after the acute episode
European Society of Coloproctology (ESCP) 2020	May not be necessary after a single episode, CT-verified, and symptom-free recovery. Indicated in all other patients treated without resection, without a colonoscopy in the past 3 years		At least 6 weeks after the acute episode
World Society of Emergency Surgery (WSES) 2020	Not if diagnosed by CT imaging	Yes, in patients with diverticular abscesses treated non-operatively	Within about 4–6 weeks
European Association of Endoscopic Surgery (EAES)/Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) 2018	Not indicated if successfully treated, and no high-risk features present	Not explicitly stated	

CRC: colorectal cancer; CT: computed tomography.

enteric nervous system. Increases in the neuropeptides substance P, galanin, and neuropeptide K (NK) have been found in increased amounts in neural tissue following acute diverticulitis episodes [68, 69]. Humes et al. [68] took the evidence of increased inflammatory modulators and neuropeptides one step further and correlated them with patient presentation. They started by collecting sigmoid colon biopsies and blood samples. Then, after five days, they performed rectal balloon distension and measured the pain threshold between patients with symptomatic and asymptomatic diverticular disease. They found that the symptomatic patients had significantly lower thresholds for discomfort and higher expression of NK1 receptor, IL-6, and TNF- α on tissue analysis [68]. These data suggest a role for inflammation in the modulation of neuropeptides and the development of visceral hypersensitivity.

Visceral hypersensitivity is also seen in several conditions that result from an imbalance or alteration of the brain-gut axis, which is a complex system wherein there is bidirectional crosstalk between the brain and the gut influenced by many external factors [70]. Disorders of gut-brain interaction (DGBI) or functional GI disorders (FGID) encompass the many conditions associated with alterations of this axis. These conditions include irritable bowel syndrome (IBS), functional dyspepsia, and functional constipation, among many other variations of symptoms, including changes in bowel habits, nausea, and vomiting that do not appear to have a gross pathological basis [71]. Although not fully understood, many studies have identified links between psychosocial factors, diet, recent GI infection, and obesity as contributors to a low-grade inflammatory state and resulting decreased pain threshold [70–72]. Systemic inflammation is shown to alter GI immune function (B- and T-cell populations, IL-6 and IL-8, and changes in mast cell density and activity), the neuroendocrine system (changes in serotonergic functioning), and the integrity of the GI barrier (loss of tight junctions and increased permeability, dysbiosis) [72]. There are also stigmata associated with disorders termed “functional”, so it is common for anxiety and depression to develop because of diagnostic uncertainty or unsatisfying diagnoses, further contributing to 5-HT dysfunction [71]. When taken all together, this system can be very easily perturbed by either an organic GI disorder (i.e., bacterial or viral gastroenteritis) or by external psychosocial forces (i.e., depression), predisposing the patient to further complications.

Importance of vitamin D

While vitamin D is well recognized for its role in regulating calcium homeostasis, recent studies have identified novel connections between this vitamin and GI integrity (Table 3). This role is particularly important in the context of pathological GI conditions such as IBD, IBS, and celiac disease. In the intestine, vitamin D exerts a broad range of gut-protective effects, including promoting the differentiation of antigen-presenting cells and enhancing the overall adaptive immune response [73]. Vitamin D also affects the structural integrity of the intestinal barrier by preserving the genetic expression of tight junctions in colonic epithelial cells and strengthening intercellular connections [73, 74]. Findings from in vivo murine models further established the protective role of vitamin D, in which deficient mice were more susceptible to *C. rodentium* colonic injury, which is characterized by tight junction instability at a cellular level [74]. Interestingly, vitamin D has also been shown to affect fecal microbiota makeup, stability, and metabolites [75]. Supplementation of individuals with moderate-dose vitamin D was sufficient to alter the composition of the microbiome and favor the growth of specific taxa [75]. While evidence suggests that these changes may be beneficial, the precise mechanism driving benefits to the immune system is unclear and requires further investigation.

Table 3. Summary of the role and importance of vitamin D in GI disease.

Category	The role and importance of vitamin D
Implicated GI diseases	IBD, IBS, CRC, diverticular disease, celiac disease, many others
Role in the GI system	Enhances immune response, improves intestinal epithelial barrier integrity, promotes favorable microbiome diversity, and regulates intestinal proliferation
Sources	Diet, supplementation, sunlight
Recommended dosing	Not definitive; 2,000 IU to 40,000 IU weekly dosing escalation has shown positive therapeutic efficacy correlation without serious adverse reactions
Recommended serum 25(OH)D levels	Greater than 30 ng/mL

CRC: colorectal cancer; GI: gastrointestinal; IBD: inflammatory bowel disease; IBS: irritable bowel syndrome.

Taken together, these cellular mechanisms provide a framework to contextualize broader epidemiological trends related to vitamin D status. Considering ultraviolet (UV) light exposure is requisite for endogenous vitamin D synthesis, investigators compared the rate of diverticulitis admissions across different geographical regions and seasons. Consistent with the notion that vitamin D confers gut-protective effects, rates of diverticulitis admissions were significantly higher in areas of low UV light exposure compared with areas of high UV light exposure [76]. In addition, admission rates were lowest in winter months (January–March) and highest in summer months (June–August) [76]. Although seemingly counterintuitive, this finding could be explained by a latent period between vitamin D deficiency and clinical presentation. In further qualifying seasonal variation in admissions, the increased admission rate observed during the summer was more pronounced in areas with greater annual UV fluctuation (i.e., higher latitude regions) [76].

These seasonal and geographic patterns underscore the impact of individual vitamin D status, as higher pre-diagnostic levels of vitamin D have been associated with significantly lower risk of diverticulitis [77]. In fact, when compared with individuals with uncomplicated diverticulosis, mean levels of pre-diagnostic vitamin D were significantly lower in patients with acute diverticulitis and diverticulitis with sequelae (i.e., abscess, recurrent diverticulitis, or requiring emergent laparotomy). Further work is needed to delineate how much risk is attributed to various levels of deficiency of vitamin D. This, however, is a lab test that is simple to order and worth correcting in most patients, as it can possibly reduce the risk of incident or recurrent episodes of diverticulitis.

Conclusions

Diverticular disease continues to affect the lives of many patients, and over the past two decades, new data have changed the way we approach preventive measures, discussions on risk factors, and management.

Given the variability in presentation and the unique risk that each patient carries, the treatment and management plans moving forward must take into consideration individual risk profiles. Large studies have effectively refuted the misbelief that particulate food incites diverticular events, as well as amended our views on the routine use of antibiotics and follow-up colonoscopies. We have also seen that CT-induced cancer risk is increasing to a non-negligible level. Although CT greatly increases diagnostic accuracy and can help mitigate the need for follow-up colonoscopy, thoughtful use of imaging that exposes patients to ionizing radiation is paramount. Additionally, the importance of inflammation as a contributor to acute events and lingering symptoms is becoming clear, providing new avenues for exploration into new treatment modalities and understanding aggravating factors and comorbidities. Many of these risks are in the patient's control, including diet, exercise, and cessation of alcohol and tobacco use. Explaining their role and the impact of everyday decisions on their disease course is imperative. Additionally, research uncovering risk attributed to non-modifiable factors is important to establish baseline risk. This knowledge allows the patient to become more involved in their condition and empowers them to work towards more favorable long-term outcomes.

At a time when pivotal research is emerging and evidence-based practices are changing just as fast, it is the job of the gastroenterologist to stay at the forefront to advise effectively and provide the safest and most up-to-date care. Knowledge of this data is integral when counseling our patients about proper diet and supplementation, the importance of genetics in diverticulitis, and treatment options for acute episodes and lingering symptoms. Finally, continued research into specific inflammatory markers indicating disease severity and progression, and effective ways to optimize the health of the gut microbiome, will be key in improving outcomes.

Abbreviations

AUD: acute uncomplicated diverticulitis

BMI: body mass index

CRC: colorectal cancer

CRP: C-reactive protein

CT: computed tomography

GI: gastrointestinal

HALP: hemoglobin, albumin, lymphocyte, and platelet

IBD: inflammatory bowel disease

IBS: irritable bowel syndrome

IL: interleukin

MRI: magnetic resonance imaging

NK: neuropeptide K

NSAIDs: non-steroidal anti-inflammatory drugs

PRS: polygenic risk score

TNF: tumor necrosis factor

US: ultrasound

UV: ultraviolet

Declarations

Author contributions

NBDA: Writing—original draft, Writing—review & editing, Project administration. TV: Writing—original draft. ECOIV: Writing—review & editing. DAJ: Conceptualization, Supervision, Project administration, Writing—review & editing. All authors read and approved the submitted version.

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The authors declare that there are no conflicts of interest.

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References

1. Hall JK, Supiano MA, Cohan JN. Diverticulitis in Older Adults: A Review of Etiology, Diagnosis, and Management. *J Am Geriatr Soc.* 2025;73:1598–607. [DOI] [PubMed] [PMC]
2. Qaseem A, Etzeandia-Ikobaltzeta I, Lin JS, Fitterman N, Shamliyan T, Wilt TJ, et al.; Clinical Guidelines Committee of the American College of Physicians. Diagnosis and Management of Acute Left-Sided Colonic Diverticulitis: A Clinical Guideline From the American College of Physicians. *Ann Intern Med.* 2022;175:399–415. [DOI] [PubMed]
3. Wilkins T, Embry K, George R. Diagnosis and management of acute diverticulitis. *Am Fam Physician.* 2013;87:612–20. [PubMed]
4. Garfinkle R, Boutros M. Current Management of Diverticulitis. *Adv Surg.* 2024;58:87–106. [DOI] [PubMed]
5. Mathews SN, Lamm R, Yang J, Kang L, Telem D, Pryor AD, et al. Factors Associated with Repeated Health Resource Utilization in Patients with Diverticulitis. *J Gastrointest Surg.* 2017;21:112–20. [DOI] [PubMed]
6. Shahedi K, Fuller G, Bolus R, Cohen E, Vu M, Shah R, et al. Long-term risk of acute diverticulitis among patients with incidental diverticulosis found during colonoscopy. *Clin Gastroenterol Hepatol.* 2013;11:1609–13. [DOI] [PubMed] [PMC]

7. Peery AF, Shaukat A, Strate LL. AGA Clinical Practice Update on Medical Management of Colonic Diverticulitis: Expert Review. *Gastroenterology*. 2021;160:906–11.e1. [DOI] [PubMed] [PMC]
8. Strate LL, Liu YL, Syngal S, Alloori WH, Giovannucci EL. Nut, corn, and popcorn consumption and the incidence of diverticular disease. *JAMA*. 2008;300:907–14. [DOI] [PubMed] [PMC]
9. Jiang R, Jacobs DR Jr, Mayer-Davis E, Szklo M, Herrington D, Jenny NS, et al. Nut and seed consumption and inflammatory markers in the multi-ethnic study of atherosclerosis. *Am J Epidemiol*. 2006;163:222–31. [DOI] [PubMed]
10. Hu FB, Stampfer MJ. Nut consumption and risk of coronary heart disease: a review of epidemiologic evidence. *Curr Atheroscler Rep*. 1999;1:204–9. [DOI] [PubMed]
11. Barlowe T, Anderson C, Nichols HB, Salvador AC, Sandler RS, Sandler DP, et al. Diet and Risk for Incident Diverticulitis in Women: A Prospective Cohort Study. *Ann Intern Med*. 2025;178:788–95. [DOI] [PubMed] [PMC]
12. Lemes VB, Galdino GG, Romão P, Reis ST. THE RELATION BETWEEN THE DIET AND THE DIVERTICULITIS PATHOPHYSIOLOGY: AN INTEGRATIVE REVIEW. *Arq Gastroenterol*. 2021;58:394–8. [DOI] [PubMed]
13. Qiu P, Ishimoto T, Fu L, Zhang J, Zhang Z, Liu Y. The Gut Microbiota in Inflammatory Bowel Disease. *Front Cell Infect Microbiol*. 2022;12:733992. [DOI] [PubMed] [PMC]
14. Lei Y, Li S, He M, Ao Z, Wang J, Wu Q, et al. Oral Pathogenic Bacteria and the Oral-Gut-Liver Axis: A New Understanding of Chronic Liver Diseases. *Diagnostics (Basel)*. 2023;13:3324. [DOI] [PubMed] [PMC]
15. Daniels L, Budding AE, de Korte N, Eck A, Bogaards JA, Stockmann HB et al.. Fecal microbiome analysis as a diagnostic test for diverticulitis. *Eur J Clin Microbiol Infect Dis*. 2014;33:1927–36. [DOI] [PubMed]
16. Ma W, Wang Y, Nguyen LH, Mehta RS, Ha J, Bhosle A, et al. Gut microbiome composition and metabolic activity in women with diverticulitis. *Nat Commun*. 2024;15:3612. [DOI] [PubMed] [PMC]
17. Piccioni A, Franza L, Brigida M, Zanza C, Torelli E, Petrucci M, et al. Gut Microbiota and Acute Diverticulitis: Role of Probiotics in Management of This Delicate Pathophysiological Balance. *J Pers Med*. 2021;11:298. [DOI] [PubMed] [PMC]
18. Petruzzello C, Migneco A, Cardone S, Covino M, Saviano A, Franceschi F, et al. Supplementation with *Lactobacillus reuteri* ATCC PTA 4659 in patients affected by acute uncomplicated diverticulitis: a randomized double-blind placebo controlled trial. *Int J Colorectal Dis*. 2019;34:1087–94. [DOI] [PubMed]
19. Bharucha AE, Parthasarathy G, Ditah I, Fletcher JG, Ewelukwa O, Pendlimari R, et al. Temporal Trends in the Incidence and Natural History of Diverticulitis: A Population-Based Study. *Am J Gastroenterol*. 2015;110:1589–96. [DOI] [PubMed] [PMC]
20. Diamant MJ, Schaffer S, Coward S, Kuenzig ME, Hubbard J, Eksteen B, et al. Smoking Is Associated with an Increased Risk for Surgery in Diverticulitis: A Case Control Study. *PLoS One*. 2016;11:e0153871. [DOI] [PubMed] [PMC]
21. Hjern F, Wolk A, Håkansson N. Smoking and the risk of diverticular disease in women. *Br J Surg*. 2011;98:997–1002. [DOI] [PubMed]
22. Turunen P, Wikström H, Carpelan-Holmström M, Kairaluoma P, Kruuna O, Scheinin T. Smoking increases the incidence of complicated diverticular disease of the sigmoid colon. *Scand J Surg*. 2010;99:14–7. [DOI] [PubMed]
23. Sağıroğlu J, Beyazadam D, Gök AFK, Ökmen H, Özpek A, Atalay S, et al. Factors predicting severity level, progression and recurrence risk of acute left colonic diverticulitis in Turkey: A multicenter study. *Ulus Travma Acil Cerrahi Derg*. 2021;27:132–8. [DOI] [PubMed]
24. Barrett-Connor E, Khaw KT. Cigarette smoking and increased central adiposity. *Ann Intern Med*. 1989;111:783–7. [DOI] [PubMed]
25. Nagata N, Niikura R, Shimbo T, Kishida Y, Sekine K, Tanaka S, et al. Alcohol and smoking affect risk of uncomplicated colonic diverticulosis in Japan. *PLoS One*. 2013;8:e81137. [DOI] [PubMed] [PMC]

26. Sharara AI, El-Halabi MM, Mansour NM, Malli A, Ghaith OA, Hashash JG, et al. Alcohol consumption is a risk factor for colonic diverticulosis. *J Clin Gastroenterol.* 2013;47:420–5. [DOI] [PubMed]
27. Tønnesen H, Engholm G, Moller H. Association between alcoholism and diverticulitis. *Br J Surg.* 1999; 86:1067–8. [DOI] [PubMed]
28. Gunby SA, Ma W, Levy MJ, Giovannucci EL, Chan AT, Strate LL. Smoking and Alcohol Consumption and Risk of Incident Diverticulitis in Women. *Clin Gastroenterol Hepatol.* 2024;22:1108–16. [DOI] [PubMed] [PMC]
29. Ma W, Jovani M, Nguyen LH, Tabung FK, Song M, Liu PH, et al. Association Between Inflammatory Diets, Circulating Markers of Inflammation, and Risk of Diverticulitis. *Clin Gastroenterol Hepatol.* 2020;18:2279–86.e3. [DOI] [PubMed] [PMC]
30. Chiva-Blanch G, Urpi-Sarda M, Llorach R, Rotches-Ribalta M, Guillén M, Casas R, et al. Differential effects of polyphenols and alcohol of red wine on the expression of adhesion molecules and inflammatory cytokines related to atherosclerosis: a randomized clinical trial. *Am J Clin Nutr.* 2012; 95:326–34. [DOI] [PubMed]
31. Estruch R, Sacanella E, Badia E, Antúnez E, Nicolás JM, Fernández-Solá J, et al. Different effects of red wine and gin consumption on inflammatory biomarkers of atherosclerosis: a prospective randomized crossover trial. Effects of wine on inflammatory markers. *Atherosclerosis.* 2004;175:117–23. [DOI] [PubMed]
32. Rungratanawanich W, Qu Y, Wang X, Essa MM, Song BJ. Advanced glycation end products (AGEs) and other adducts in aging-related diseases and alcohol-mediated tissue injury. *Exp Mol Med.* 2021;53: 168–88. [DOI] [PubMed] [PMC]
33. Strate LL, Liu YL, Aldoori WH, Syngal S, Giovannucci EL. Obesity increases the risks of diverticulitis and diverticular bleeding. *Gastroenterology.* 2009;136:115–22.e1. [DOI] [PubMed] [PMC]
34. Ma W, Jovani M, Liu PH, Nguyen LH, Cao Y, Tam I, et al. Association Between Obesity and Weight Change and Risk of Diverticulitis in Women. *Gastroenterology.* 2018;155:58–66.e4. [DOI] [PubMed] [PMC]
35. Shoelson SE, Herrero L, Naaz A. Obesity, inflammation, and insulin resistance. *Gastroenterology.* 2007; 132:2169–80. [DOI] [PubMed]
36. Ma W, Ha J, Neylan CJ, Munro H, Skerrett D, Downie JM, et al. Lifestyle factors, genetic susceptibility and risk of incident diverticulitis: an integrated analysis of four prospective cohort studies and electronic health records-linked biobank. *Gut.* 2025;74:2004–11. [DOI] [PubMed]
37. Strate LL, Morris AM. Epidemiology, Pathophysiology, and Treatment of Diverticulitis. *Gastroenterology.* 2019;156:1282–98.e1. [DOI] [PubMed] [PMC]
38. Valenzuela PL, Ruilope LM, Santos-Lozano A, Wilhelm M, Kränkel N, Fiuza-Luces C, et al. Exercise benefits in cardiovascular diseases: from mechanisms to clinical implementation. *Eur Heart J.* 2023; 44:1874–89. [DOI] [PubMed]
39. Lanás A, Sopena F. Nonsteroidal anti-inflammatory drugs and lower gastrointestinal complications. *Gastroenterol Clin North Am.* 2009;38:333–52. [DOI] [PubMed]
40. Kim KO. Recent updates on classification and unsolved issues of diverticular disease: a narrative review. *J Yeungnam Med Sci.* 2024;41:252–60. [DOI] [PubMed] [PMC]
41. Wilson RG, Smith AN, Macintyre IM. Complications of diverticular disease and non-steroidal anti-inflammatory drugs: a prospective study. *Br J Surg.* 1990;77:1103–4. [DOI] [PubMed]
42. Longo S, Altobelli E, Castellini C, Vernia F, Valvano M, Magistroni M, et al. Non-steroidal anti-inflammatory drugs and acetylsalicylic acid increase the risk of complications of diverticular disease: a meta-analysis of case-control and cohort studies. *Int J Colorectal Dis.* 2022;37:521–9. [DOI] [PubMed]
43. Yuhara H, Corley DA, Nakahara F, Nakajima T, Koike J, Igarashi M, et al. Aspirin and non-aspirin NSAIDs increase risk of colonic diverticular bleeding: a systematic review and meta-analysis. *J Gastroenterol.* 2014;49:992–1000. [DOI] [PubMed]

44. Strate LL, Liu YL, Huang ES, Giovannucci EL, Chan AT. Use of aspirin or nonsteroidal anti-inflammatory drugs increases risk for diverticulitis and diverticular bleeding. *Gastroenterology*. 2011;140:1427–33. [DOI] [PubMed] [PMC]
45. Reichert MC, Krawczyk M, Appenrodt B, Casper M, Friesenhahn-Ochs B, Grünhage F, et al. Selective association of nonaspirin NSAIDs with risk of diverticulitis. *Int J Colorectal Dis*. 2018;33:423–30. [DOI] [PubMed]
46. Brown RF, Lopez K, Smith CB, Charles A. Diverticulitis: A Review. *JAMA*. 2025;[Epub ahead of print]. [DOI] [PubMed]
47. Strate LL, Erichsen R, Baron JA, Mortensen J, Pedersen JK, Riis AH, et al. Heritability and familial aggregation of diverticular disease: a population-based study of twins and siblings. *Gastroenterology*. 2013;144:736–42.e1: quiz e14. [DOI] [PubMed]
48. Maguire LH, Handelman SK, Du X, Chen Y, Pers TH, Speliotes EK. Genome-wide association analyses identify 39 new susceptibility loci for diverticular disease. *Nat Genet*. 2018;50:1359–65. [DOI] [PubMed] [PMC]
49. De Roo AC, Chen Y, Du X, Handelman S, Byrnes M, Regenbogen SE, et al. Polygenic Risk Prediction in Diverticulitis. *Ann Surg*. 2023;277:e1262–8. [DOI] [PubMed] [PMC]
50. Jeger V, Pop R, Forudastan F, Barras JP, Zuber M, Piso RJ. Is there a role for procalcitonin in differentiating uncomplicated and complicated diverticulitis in order to reduce antibiotic therapy? A prospective diagnostic cohort study. *Swiss Med Wkly*. 2017;147:w14555. [DOI] [PubMed]
51. Kiewiet JJS, Andeweg CS, Laurell H, Daniels L, Laméris W, Reitsma JB, et al. External validation of two tools for the clinical diagnosis of acute diverticulitis without imaging. *Dig Liver Dis*. 2014;46:119–24. [DOI] [PubMed]
52. Tursi A, Elisei W, Picchio M, Brandimarte G. Increased faecal calprotectin predicts recurrence of colonic diverticulitis. *Int J Colorectal Dis*. 2014;29:931–5. [DOI] [PubMed]
53. Barlas AM, Altiner S, Başpınar A, Aydın SM, Doğanay E, Kaymak Ş, et al. Predicting complicated acute diverticulitis with the HALP score: A retrospective study. *Asian J Surg*. 2025;48:287–91. [DOI] [PubMed]
54. Weizman AV, Nguyen GC. Diverticular disease: epidemiology and management. *Can J Gastroenterol*. 2011;25:385–9. [DOI] [PubMed] [PMC]
55. Han JW, Son J, Oh C. Features of colonic diverticulitis in children and adolescents: A multicenter study. *Asian J Surg*. 2024;47:2195–9. [DOI] [PubMed]
56. Langenfeld SJ. Evaluation and Medical Management of Uncomplicated Diverticulitis. *Clin Colon Rectal Surg*. 2021;34:86–90. [DOI] [PubMed] [PMC]
57. Sacks OA, Hall J. Management of Diverticulitis: A Review. *JAMA Surg*. 2024;159:696–703. [DOI] [PubMed]
58. Smith-Bindman R, Chu PW, Azman Firdaus H, Stewart C, Malekheadayat M, Alber S, et al. Projected Lifetime Cancer Risks From Current Computed Tomography Imaging. *JAMA Intern Med*. 2025;185:710–9. [DOI] [PubMed] [PMC]
59. Au S, Aly EH. Treatment of Uncomplicated Acute Diverticulitis Without Antibiotics: A Systematic Review and Meta-analysis. *Dis Colon Rectum*. 2019;62:1533–47. [DOI] [PubMed]
60. Chabok A, Pählman L, Hjern F, Haapaniemi S, Smedh K; Group AS, et al.; AVOD Study Group. Randomized clinical trial of antibiotics in acute uncomplicated diverticulitis. *Br J Surg*. 2012;99:532–9. [DOI] [PubMed]
61. Isacson D, Andreasson K, Nikberg M, Smedh K, Chabok A. No antibiotics in acute uncomplicated diverticulitis: does it work? *Scand J Gastroenterol*. 2014;49:1441–6. [DOI] [PubMed]
62. Sharma PV, Eglinton T, Hider P, Frizelle F. Systematic review and meta-analysis of the role of routine colonic evaluation after radiologically confirmed acute diverticulitis. *Ann Surg*. 2014;259:263–72. [DOI] [PubMed]

63. Meyer J, Orci LA, Combescure C, Balaphas A, Morel P, Buchs NC, et al. Risk of Colorectal Cancer in Patients With Acute Diverticulitis: A Systematic Review and Meta-analysis of Observational Studies. *Clin Gastroenterol Hepatol*. 2019;17:1448–56.e17. [DOI] [PubMed]
64. Francis NK, Sylla P, Abou-Khalil M, Arolfo S, Berler D, Curtis NJ, et al. EAES and SAGES 2018 consensus conference on acute diverticulitis management: evidence-based recommendations for clinical practice. *Surg Endosc*. 2019;33:2726–41. [DOI] [PubMed] [PMC]
65. Sartelli M, Weber DG, Kluger Y, Ansaloni L, Coccolini F, Abu-Zidan F, et al. 2020 update of the WSES guidelines for the management of acute colonic diverticulitis in the emergency setting. *World J Emerg Surg*. 2020;15:32. [DOI] [PubMed] [PMC]
66. Schultz JK, Azhar N, Binda GA, Barbara G, Biondo S, Boermeester MA, et al. European Society of Coloproctology: guidelines for the management of diverticular disease of the colon. *Colorectal Dis*. 2020;22 Suppl 2:5–28. [DOI] [PubMed]
67. Hall J, Hardiman K, Lee S, Lightner A, Stocchi L, Paquette IM, et al.; Prepared on behalf of the Clinical Practice Guidelines Committee of the American Society of Colon and Rectal Surgeons. The American Society of Colon and Rectal Surgeons Clinical Practice Guidelines for the Treatment of Left-Sided Colonic Diverticulitis. *Dis Colon Rectum*. 2020;63:728–47. [DOI] [PubMed]
68. Humes DJ, Simpson J, Smith J, Sutton P, Zaitoun A, Bush D, et al. Visceral hypersensitivity in symptomatic diverticular disease and the role of neuropeptides and low grade inflammation. *Neurogastroenterol Motil*. 2012;24:318-e163. [DOI] [PubMed]
69. Simpson J, Sundler F, Humes DJ, Jenkins D, Scholefield JH, Spiller RC. Post inflammatory damage to the enteric nervous system in diverticular disease and its relationship to symptoms. *Neurogastroenterol Motil*. 2009;21:847-e58. [DOI] [PubMed]
70. Yanping W, Gao X, Cheng Y, Liu M, Liao S, Zhou J, et al. The interaction between obesity and visceral hypersensitivity. *J Gastroenterol Hepatol*. 2023;38:370–7. [DOI] [PubMed]
71. Black CJ, Drossman DA, Talley NJ, Ruddy J, Ford AC. Functional gastrointestinal disorders: advances in understanding and management. *Lancet*. 2020;396:1664–74. [DOI] [PubMed]
72. Ng QX, Soh AYS, Loke W, Lim DY, Yeo W. The role of inflammation in irritable bowel syndrome (IBS). *J Inflamm Res*. 2018;11:345–9. [DOI] [PubMed] [PMC]
73. Wang J, Mei L, Hao Y, Xu Y, Yang Q, Dai Z, et al. Contemporary Perspectives on the Role of Vitamin D in Enhancing Gut Health and Its Implications for Preventing and Managing Intestinal Diseases. *Nutrients*. 2024;16:2352. [DOI] [PubMed] [PMC]
74. Assa A, Vong L, Pinnell LJ, Rautava J, Avitzur N, Johnson-Henry KC, et al. Vitamin D deficiency predisposes to adherent-invasive *Escherichia coli*-induced barrier dysfunction and experimental colonic injury. *Inflamm Bowel Dis*. 2015;21:297–306. [DOI] [PubMed]
75. Wyatt M, Choudhury A, Von Dohlen G, Heilesen JL, Forsse JS, Rajakaruna S, et al. Randomized control trial of moderate dose vitamin D alters microbiota stability and metabolite networks in healthy adults. *Microbiol Spectr*. 2024;12:e0008324. [DOI] [PubMed] [PMC]
76. Maguire LH, Song M, Strate LL, Giovannucci EL, Chan AT. Association of geographic and seasonal variation with diverticulitis admissions. *JAMA Surg*. 2015;150:74–7. [DOI] [PubMed] [PMC]
77. Maguire LH, Song M, Strate LE, Giovannucci EL, Chan AT. Higher serum levels of vitamin D are associated with a reduced risk of diverticulitis. *Clin Gastroenterol Hepatol*. 2013;11:1631–5. [DOI] [PubMed] [PMC]