

Review Article

Lifestyles and their Close Relationship with Gastrointestinal Diseases (Part I: Diet)

Shashi K. Agarwal, MD^{1*} ¹2227 US Highway 1, Suite 309, North Brunswick, NJ 08902, USA. **ORCID:** 0000-0003-0007-5582**Article History****Received:** 07.01.2022**Accepted:** 10.02.2022**Published:** 18.02.2022**Journal homepage:**<https://www.easpublisher.com>**Quick Response Code**

Abstract: The gastrointestinal tract (GI) is a continuous hollow twisting tube from the mouth to the anus. Its hollow organs include the mouth, esophagus, stomach, small intestine, large intestine, and anus. The liver, pancreas, and gallbladder (solid organs) are also considered part of the GI tract. The principal functions of the GI tract are digestion, absorption, excretion, and protection. Digestion and absorption occur primarily in the stomach and small intestine. Desiccation and compaction of waste occur in the large intestine. The waste products are then stored in the sigmoid colon and rectum before their elimination. The GI tract is influenced by several lifestyles, including the amount and the composition of the diet. The macronutrients and micronutrients in the diet, if prudent, are important for maintaining good GI health. However, unhealthy choices may cause or influence the development of GI pathology (such as esophageal reflux, peptic ulcer, inflammatory bowel disease, dietary intolerance, or even GI cancers). The lifestyle GI connection is reviewed in this two-part manuscript.

Keywords: Gastrointestinal diseases, diet, plant-based diet, meat-based diet, alcohol, coffee.

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INTRODUCTION

The gastrointestinal (GI) tract is primarily responsible for acquiring and digesting food, absorbing nutrients and water, and expelling wastes from the body as feces [1]. A proper diet and a normally functioning GI tract are important for the delivery of nutrients, prevention of nutrient deficiencies and malnutrition, repair of any damaged intestinal epithelium, restoration of normal luminal bacterial populations, promotion of normal GI motility, and maintenance of normal immune functions [2-5]. The caloric quantity of diet also affects the body weight [6]. The GI tract is extremely susceptible to lifestyles and an improper diet may result in or aggravate several GI diseases [7]. These include GI reflux esophagitis, peptic ulcer disease (PUD), irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), GI cancer, and pancreatitis. Hepatic disorders and their relationship with lifestyles have been discussed by me in a recent publication [7]. Gastro-esophageal reflux disease (GERD) is defined based on chronic and recurrent typical symptoms, i.e., pyrosis and acid regurgitation as well as extra-esophageal manifestation, demonstrated to impair quality of life (QoL) [8]. GERD can be classified as

non-erosive reflux disease or erosive reflux disease based on the presence or absence of esophageal mucosal damage seen on endoscopy. According to endoscopic findings and esophageal pH monitoring, some patients with GERD are asymptomatic [9]. Delayed gastric emptying and dysfunction of the lower esophageal sphincter is the main underlying pathogenesis [10]. Typical symptoms include heartburn and acid regurgitation which have a high specificity but low sensitivity for GERD [11]. Other symptoms include epigastric pain, dyspepsia, nausea, bloating, and belching. Extraesophageal symptoms are sometimes seen and include chronic cough, asthma, laryngitis, and dental erosions [12]. Effective treatments include lifestyle modification, proton pump inhibitors, and surgery. However, diet and lifestyle modifications are receiving increasing attention in their influence on the prevention and treatment of GERD [13-15]. Functional dyspepsia is a GI functional disorder characterized by symptoms, such as epigastric fullness and bloating, nausea, discomfort, and vomiting, which are provoked following food consumption [16]. Peptic ulcer disease (PUD) is a global problem with a lifetime risk of development ranging from 5% to 10% [17-21]. Peptic ulcer disease is characterized by discontinuation in the

*Corresponding Author: Shashi K. Agarwal, MD

2227 US Highway 1, Suite 309, North Brunswick, NJ 08902, USA

inner lining of the stomach (stomach ulcer) or proximal duodenal tract (duodenal ulcer) and may extend into the muscularis propria layer of the epithelium. Sometimes it may involve the lower esophagus, distal duodenum, or jejunum [22]. Duodenal ulcers are four times more common than gastric ulcers and appear more frequently in men. *H. pylori* – a gram-negative bacillus, is responsible for 90% of the duodenal ulcers and 70% to 90% of the gastric ulcers [23, 24]. Peptic ulcers may result in various complications such as bleeding, perforation, and gastric outlet obstruction [22]. Although the prevalence of PUD caused by *H. pylori* is on the decline, the prevalence of PUD induced by NSAIDs, or aspirin is increasing because of the worldwide increase in the use of these drugs by the growing aging population [25-27]. Acute pancreatitis (AP) is an inflammatory disease involving the pancreatic parenchyma and peripancreatic tissues [28]. It results from exocrine cell destruction by infiltrating inflammatory cells. Chronic pancreatitis (CP) is diagnosed if there is severe or recurring inflammation of the pancreas. The incidence of AP and CP has increased in recent decades [29-31]. It is a common cause of hospitalization and accounts for approximately 2.6 billion dollars in annual inpatient costs in the United States [32, 33]. In up to 20% of these cases, there are serious complications, and the mortality rate ranges from 10% to 30% [34-36]. Currently, there is no specific pharmacotherapy for AP. Cholelithiasis, excessive alcohol intake, hyperlipidemia, pancreatic trauma, infections, and medications are common risk factors [30, 31, 36, 37]. Chronic pancreatitis (CP) usually results from recurrent attacks of acute pancreatitis, leading to the development of pancreatic insufficiency, steatorrhea, diabetes, pancreatic calcification, and fibrosis. Although the incidence of CP is lower, patients with CP have a lower QoL and shorter lifespan than the general population [36]. Celiac disease results from exposure to gluten in susceptible individuals. It is relieved by eliminating gluten from the diet [38].

Irritable bowel syndrome (IBS) is a chronic GI functional disorder characterized by recurrent abdominal pain, as well as a change in bowel movements. This results in either diarrhea, constipation, or both without any known organic causes [39-41]. Patients with IBS are divided into four subtypes according to the stool pattern: diarrhea-predominant IBS, constipation-predominant IBS, mixed-diarrhea-and-constipation, and unclassified IBS [39, 42]. Patients with IBS are usually diagnosed at a young age, and IBS is more common in women than in men [43]. Although IBS is not associated with increased mortality, it reduces the quality of life to a significant degree [44]. It is also a major economic burden on society [45]. In contrast, inflammatory bowel disease (IBD) [46] is a relapsing inflammatory disease and includes both ulcerative colitis (UC) [47] and Crohn's disease (CD) [48]. UC is localized primarily in the large intestine [49,

50], whereas CD can affect any area of the GI tract [51, 52]. Both incidence and prevalence of IBD are currently increasing worldwide [53]. Diverticulosis usually results from obstruction of the diverticular ostium by a stool fragment or food particles and subsequent inflammation. Diverticula is characterized by the presence of extroflexions that occur when colonic mucosa and sub-mucosa herniate through defects in the muscle layer of the colon wall [54]. Inflammatory bowel diseases (IBDs) are a biologically complex set of conditions characterized by chronic, relapsing inflammation of the gastrointestinal (GI) tract. The two main types of IBDs are UC and CD. They are characterized by chronic inflammation of the GI tract. Ulcerative colitis usually affects the colon and rectum, while CD tends to involve the mouth, esophagus, stomach, colon, rectum, and anus. Both diseases can present with persistent diarrhea, rectal bleeding, vomiting, abdominal pain, weight loss, and fatigue. CD patients tend to have porridge like defecation while UC patients often have bowel movements with mucus and blood. UC patients may also experience rectal urgency and tenesmus. Colorectal cancer (CRC) is the third most common cause of cancer death in both men and women in the United States and ranks second when men and women are combined [55]. Colorectal cancer is more likely to be seen in elderly individuals. However, while overall CRC incidence rates have remained stable or declined in many high-income countries, the incidence of early-onset CRC (diagnosed before the age of 50 years) has recently been increasing worldwide [56].

DISCUSSION

Consuming a healthy diet helps prevent malnutrition as well as a range of noncommunicable diseases [57, 58]. However, changing lifestyles have resulted in higher consumption of foods rich in energy, fats, free sugars, and salt/sodium. The consumption of fruit, vegetables, and dietary fiber (whole grains) has decreased. A healthy diet for adults, according to the World Health Organization [59], should include at least 400 grams of fruits and vegetables per day (excluding starchy roots such as potatoes and cassava). They also recommend that free sugars (all sugars added to foods or drinks, as well as sugars naturally present in honey, syrups, fruit juices, and fruit juice concentrates) should be limited to 10% of total energy intake. Fats should be less than 39% of total energy intake. Unsaturated fats (found in fish, avocado, and nuts, and in sunflower, soybean, canola, and olive oils) are preferable. Saturated fats (found in fatty meat, butter, palm and coconut oil, cream, cheese, ghee, and lard) should be less than 10% of total energy intake. Industrially produced trans fats (found in baked and fried foods, and pre-packaged snacks and foods, such as frozen pizza, pies, cookies, biscuits, wafers, and cooking oils and spreads) should be avoided. Salt intake should be less than 5 g of salt (equivalent to about one teaspoon) per day. Salt should be iodized. A healthy diet (both in

calorie amount and in quality) helps prevent the development and progression of most GI diseases. The effect of diet on these ailments is the topic of discussion in this Part I of this two-part manuscript. Other four major lifestyles (smoking, alcohol intake, obesity, and exercise) and their relationship with GI diseases are discussed in part II.

Diet

Diet has a major influence on the GI tract. Zhang *et al.* in a review of 72 articles, found that GERD was less frequent in individuals on plant-based diets (Odds Ratio or OR=0.34) and not eating meat (OR=0.841), and more frequent in omnivores (daily meat, fish, and egg intake: OR=1.088) and with a high intake of saturated fat (high-fat diet: OR=7.568) [60]. Many patients find that GERD is aggravated by citrus fruits and tomatoes, mint, and spicy foods [61]. Chocolate, caffeine, and alcohol may also increase GERD symptoms in some [62]. Besides the type of food consumed, poor eating habits can also affect GERD. Zhang *et al.* reported that GERD is aggravated by midnight snacking (OR=5.08), skipping breakfast (OR=2.7), eating quickly (OR=4.06), eating very hot foods (OR=1.81), and eating beyond fullness: OR=2.85) [60]. Sleeping soon after dinner also worsens GERD - in their study, Zhang *et al.* noted that an interval of fewer than three hours between dinner and bedtime (OR=7.45) worsened GERD [60]. The American Gastroenterological Association and the American College of Gastroenterology do not recommend any special diet to prevent peptic ulcer formation or promote its healing [63]. However, as noted with GERD, certain nutritional component intake may help reduce the symptoms. Some patients feel better with the avoidance of pepper, caffeine, tea, peppermint, spearmint, chocolate, citrus foods, and tomatoes [64]. Fiber intake helps reduce symptoms [65]. *H. Pylori* is the main culprit behind PUD, and fermented foods, such as yogurt, kefir, sauerkraut, and kimchi, help inhibit the activity of *H. pylori* [66]. Fermented foods use several microorganisms for the fermentation process, and often contain helpful probiotics, (such as *Lactobacillus*, *Streptococcus*, *Pediococcus*, and *Leuconostoc*, *Saccharomyces cerevisiae*, *Penicillium* spp, and *Aspergillus* spp,) which often help mitigate peptic ulcers [67].

Alcohol is a major risk factor for pancreatitis and its role is discussed in part II of this manuscript. During acute pancreatitis (AP), early oral feeding helps reduce inflammation and improve outcomes [68]. Early feedings with low fat, soft oral diet, lead to a shorter length of stay, fewer complications, and lower costs. However, if oral feeding is not tolerated, supplemental enteral nutrition through gastric or jejunal feeding may be required. Although superior to parenteral nutrition, certain complications such as bowel obstructions, abdominal compartment syndrome, prolonged ileus, or mesenteric ischemia may necessitate the use of the

latter [69]. Diet also remains important during the recovery period, and a low-fat diet (void of fried and processed foods), eaten in smaller amounts, usually, six times a day, may be better tolerated [70]. Nutritional management in chronic pancreatitis (CP) is unclear as studies are scarce. Avoidance of alcohol and consumption of a balanced diet is important in these patients. They are often underweighted or sarcopenic [71] and may require a high protein, high calorie diet [72, 73]. They also often have several micronutrient deficiencies especially that of vitamin D [74], and these should be corrected with supplementation [75].

Celiac disease involves the small intestine and is due to an immune reaction to eating gluten [38]. Patients with this disease may experience symptoms like diarrhea, bloating, gas, anemia, and growth problems. Gluten is the main structural protein of wheat and is also present in several other cereal grains, such as barley, rye, wheat berries, spelt, durum, emmer, semolina, farina, farro, graham, khorasan wheat, einkorn, and triticale. Cereals that are free of gluten include quinoa, brown, black, or red rice, buckwheat, amaranth, millet, corn, sorghum, teff, and gluten-free oats. Avoidance of gluten in the diet is an effective treatment for this disease [76, 77].

IBS patients often find intolerance to several food items [78, 79]. Studies have shown that the intake of low-fermentable oligo-, di-, monosaccharides, and polyols (FODMAP) foods lead to a positive clinical response in 50%-80% of IBS patients. These patients find improvements in several symptoms including bloating, flatulence, diarrhea, and chronic abdominal pain [80, 81]. FODMAP also improves the QoL in these patients [82, 83]. The list of foods that are high or low in FODMAP is extensive and can be obtained by visiting the website of the American College of Gastroenterology [84]. IBS patients are at risk of developing deficiencies in some vitamins, minerals, and naturally occurring antioxidants, and if FODMAP diet is continued for a long time, these may need to be supplemented [85, 86].

Inflammatory bowel disease is also affected by diet [87]. An improper diet can worsen IBD by several mechanisms, including dysregulating the immune system and promoting intestinal inflammation. It can also alter intestinal permeability and contribute to microbial dysbiosis. There is an increased risk of IBD, especially UC among people who consume greater amounts of n-6 polyunsaturated fatty acids (PUFA) — and a lower risk among people with diets high in fiber, fruits, vegetables, and n-3 PUFA [88, 89]. The Western diet has more red/processed meat, saturated fat, refined grains, sugar, beer, and spirits. It has a significant amount of linoleic acid (n-6 PUFA) which is a precursor for arachidonic acid, which in turn is a precursor of inflammatory mediators such as prostaglandins and leukotrienes [90]. In contrast, long-

chain n-3 PUFAs (eicosapentaenoic acid and docosahexaenoic acid) in a plant-based/fish diet, are anti-inflammatory [91]. The benefits of n-3 PUFA rich diet was noted in the Nurses' Health Study (NHS) [92]. In this study, a higher ratio of n-3: n-6 PUFAs decreased the incidence of UC. Fiber intake is also important in IBD [93] and is often low in IBD patients [94]. In a meta-analysis, Hou *et al.* found that a high intake of dietary fiber was associated with a decreased risk of IBD [95]. In the NHS study, a large amount of fiber intake resulted in an approximately 40% reduction in the diagnosis of Crohn's disease [92]. Dietary fiber is associated with luminal production of short-chain fatty acids with immunomodulatory properties. They also help lower colonic pH, which benefits helpful microflora and inhibits potential pathogens. An adequate intake of dietary fiber can thus help reduce intestinal lesions, accelerate healing and regeneration, and help maintain remission [96]. IBD patients may also suffer from weight loss and deficiencies in essential vitamins, minerals, and other nutrients due to malabsorption [97]. Deficiencies have been noted with vitamin A [98, 99] and vitamin D [100]. Calcium deficiency is also sometimes noted [101]. Correction of these via supplementation is associated with better outcomes [102]. The increased level of n-6 PUFA in the Western diet has been mentioned before and this diet increases the risk of IBD and leads to a poorer prognosis [95, 103]. On the other hand, the Mediterranean-type dietary pattern consists mainly of fiber-rich sources, such as fruit and vegetables, and ω -3 fatty acid-rich food sources - and is associated with reduced risk of IBD development and progression [104-106].

Diet plays an important role in cancer [107, 108] including those of the GI tract [109]. It has been repeatedly documented that suboptimal dietary intake is associated with an increased risk of several GI cancers, including oral, esophageal, stomach, pancreatic, and colon/rectum [110]. Diets that are cancer preventive are primarily plant-based [111, 112]. Several studies have noted that greater fruit and vegetable consumption is preventive for GI cancers [113, 114]. Conversely, a diet rich in meat and animal products is associated with higher rates of GI cancers [115]. According to Islami *et al.* red meat consumption was associated with 5.4% of colorectal cancers [112]. Processed meat intake is especially harmful [116]. It is estimated that processed meat consumption is associated with 8.2% of colorectal cancers [112]. Fiber in the diet is also important [117]. Islami *et al.* found that low dietary fiber accounted for 10.3% of colorectal cancer cases [118]. A proper diet also helps reduce several associated symptoms of cancer and its treatment and helps improve the quality of life in these individuals [119]. Mortality is also reduced [120, 121]. Cancer survivors are at an increased risk of secondary cancers [122], and a plant-based diet has been shown to retard the development of these [123].

A prudent diet is rich in vegetables, fruit, cereals and legumes, whole grains, rice/pasta, fish, low-fat dairy, poultry, and water. It discourages processed meat, refined carbohydrates, and saturated fats. There are several mechanisms by which a prudent diet influences GI cancer. A healthy plant-based diet allows decreased exposure to carcinogens including N-nitroso compounds and decreased formation of cyto- and genotoxic aldehydes. There is less obesity, decreased insulin levels, reduced inflammation, increased antioxidative capacity, and improved DNA repair. Circulating sex and growth hormones are more balanced. It also diminishes exposure to carcinogenic heterocyclic aromatic amines and polycyclic aromatic hydrocarbons that are formed during high heat cooking of meat [124, 125]. Vegetables diversify gut microbiomes and provide better immunity, decrease tumorigenesis, and potentiate immunotherapeutic effects in cancer prevention and treatment [126].

CONCLUSIONS

Healthy lifestyles can dramatically reduce the development and progression of major GI diseases. These include GERD, PUD, pancreatitis, IBS and IBD, and several GI cancers. Obesity worsens all GI diseases and increases the risk of cancer. A prudent diet (primarily plant-based) is overall beneficial for GI disorders. Certain dietary components may aggravate GI disorders and should be avoided depending upon the nature of the disease, and the experience of the patient. Part II of this manuscript reviews the influence of four other lifestyle factors on GI diseases – smoking, obesity, alcohol intake, and exercise.

REFERENCES

1. Ogobuiro I, Gonzales J, Tuma F. Physiology, Gastrointestinal. [Updated 2021 Apr 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK537103/>.
2. Greenwood-Van Meerveld B, Johnson AC, Grundy D. Gastrointestinal Physiology and Function. *Handb Exp Pharmacol*. 2017;239:1-16. doi: 10.1007/164_2016_118.
3. Camilleri M, Madsen K, Spiller R, Greenwood-Van Meerveld B, Verne GN. Intestinal barrier function in health and gastrointestinal disease [published correction appears in *Neurogastroenterol Motil*. 2012 Oct;24(10):976. doi:10.1111/j.1365-2982.2012.01921.x.
4. Camilleri M, Madsen K, Spiller R, Greenwood-Van Meerveld B, Verne GN. Intestinal barrier function in health and gastrointestinal disease [published correction appears in *Neurogastroenterol Motil*. 2012 Oct;24(10):976. doi:10.1111/j.1365-2982.2012.01921.x
5. Kjk Tobias A, Sadiq NM. Physiology, Gastrointestinal Nervous Control. [Updated 2021 Oct 1]. In: StatPearls [Internet]. Treasure Island

- (FL): StatPearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK545268/>.
6. Golay A, Bobbioni E. The role of dietary fat in obesity. *Int J Obes Relat Metab Disord*. 1997 Jun;21 Suppl 3:S2-11.
 7. Agarwal SK (2021). Lifestyles and Diseases of the Liver. *East African Scholars J Med Sci*, 4(10), 239-249.
 8. Richter J.E., Rubenstein J.H. Presentation and Epidemiology of Gastroesophageal Reflux Disease. *Gastroenterology*. 2018; 154:267–276. doi: 10.1053/j.gastro.2017.07.045.
 9. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. *Am J Gastroenterol*. 2006;101(8):1900–1920. doi:10.1111/j.1572-0241.2006.00630.x.
 10. Chen J, Brady P. Gastroesophageal Reflux Disease: pathophysiology, Diagnosis, and Treatment. *Gastroenterol Nurs*. 2019;42(1):20–28. doi:10.1097/SGA.0000000000000359.
 11. Klauser AG, Schindlbeck NE, Müller-Lissner SA. Symptoms in gastro-oesophageal reflux disease. *Lancet*. 1990; 335:205–208.
 12. [Hom C, Vaezi MF. Extra-esophageal manifestations of gastroesophageal reflux disease: diagnosis and treatment. *Drugs*. 2013; 73:1281–1295.
 13. Freedberg DE, Kim LS, Yang YX, Risks T. and Benefits of Long-term Use of Proton Pump Inhibitors: expert Review and Best Practice Advice from the American Gastroenterological Association. *Gastroenterology*. 2017;152(4):706–715. doi:10.1053/j.gastro.2017.01.031.
 14. Sethi S, Richter JE. Diet and gastroesophageal reflux disease: role in pathogenesis and management. *Curr Opin Gastroenterol*. 2017;33(2):107–111. doi:10.1097/MOG.0000000000000337.
 15. Patti MG. An evidence-based approach to the treatment of gastroesophageal reflux disease. *JAMA Surg*. 2016; 151(1):73–78. doi:10.1001/jamasurg.2015.4233.
 16. Tack J., Talley N.J. Functional dyspepsia--symptoms, definitions and validity of the Rome III criteria. *Nat. Rev. Gastroenterol. Hepatol*. 2013; 10:134–141. doi: 10.1038/nrgastro.2013.14.
 17. Snowden FM. Emerging and reemerging diseases: a historical perspective. *Immunol Rev*. 2008 Oct;225:9-26..
 18. Lanas A, Chan FKL. Peptic ulcer disease. *Lancet*. 2017 Aug 05;390(10094):613-624.
 19. Ford AC, Gurusamy K, Delaney B, Forman D, Moayyedi P. Eradication therapy for peptic ulcer disease in -positive people. *Cochrane Database of Systematic Reviews* 2016, Issue 4. Art. No.: CD003840. DOI: 10.1002/14651858.CD003840.pub5.
 20. Malfertheiner P., Chan F.K., McColl K.E. Peptic ulcer disease. *Lancet*. 2009; 374:1449–1461. doi: 10.1016/S0140-6736(09)60938-7.
 21. Yuan Y., Padol I.T., Hunt R.H. Peptic ulcer disease today. *Nat. Clin. Pract. Gastroenterol. Hepatol*. 2006; 3:80–89. doi: 10.1038/ncpgasthep0393.
 22. Milosavljevic T, Kostić-Milosavljević M, Jovanović I, Krstić M. Complications of peptic ulcer disease. *Dig Dis*. 2011; 29:491–493.
 23. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs. *N Engl J Med*. 1999; 340:1888–1899.
 24. Hopkins RJ, Girardi LS, Turney EA. Relationship between Helicobacter pylori eradication and reduced duodenal and gastric ulcer recurrence: a review. *Gastroenterology*. 1996; 110:1244–1252.
 25. Potamitis GS, Axon AT. Helicobacter pylori and Nonmalignant Diseases. *Helicobacter*. 2015;20 Suppl 1:26–29.
 26. Thorat MA, Cuzick J. Prophylactic use of aspirin: systematic review of harms and approaches to mitigation in the general population. *Eur J Epidemiol*. 2015;30:5–18.
 27. Sasaki H, Nagahara A, Hojo M. Ten-year trend of the cumulative Helicobacter pylori eradication rate for the ‘Japanese eradication strategy’ *Digestion*. 2013;88:272–278.
 28. Petrov M.S., Yadav D. Global epidemiology and holistic prevention of pancreatitis. *Nat. Rev. Gastroenterol. Hepatol*. 2019; 16:175–184. doi: 10.1038/s41575-018-0087-5.
 29. Hirota M, Shimosegawa T, Masamune A, Kikuta K, Kume K, Hamada S. The sixth nationwide epidemiological survey of chronic pancreatitis in Japan. *Pancreatol*. 2012;12: 79–84. doi:10.1016/j.pan.2012.02.005.
 30. Yadav D, Timmons L, Benson JT, Dierkhising RA, Chari ST. Incidence, prevalence, and survival of chronic pancreatitis: a population-based study. *Am J Gastroenterol*. 2011;106: 2192–2199. doi:10.1038/ajg.2011.328.
 31. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. *Pancreas*. 2006;33: 323–330.
 32. Peery AF, Dellon ES, Lund J, Crockett SD, McGowan CE. Burden of gastrointestinal disease in the United States: 2012 update. *Gastroenterology*. 2012;143:1179–1187.e1-3.
 33. Whitcomb DC. Clinical practice. Acute pancreatitis. *N Engl J Med*. 2006;354:2142–2150.
 34. Pandol SJ, Saluja AK, Imrie CW, Banks PA. Acute pancreatitis: bench to the bedside. *Gastroenterology*. 2007;132:1127–1151.
 35. Wang GJ, Gao CF, Wei D, Wang C, Ding SQ. Acute pancreatitis: etiology and common pathogenesis. *World J Gastroenterol*. 2009;15:1427–1430.

36. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013;144: 1252–1261. doi: 10.1053/j.gastro.2013.01.068.
37. Hirota M, Shimosegawa T, Masamune A, Kikuta K, Kume K, Hamada S. The sixth nationwide epidemiological survey of chronic pancreatitis in Japan. *Pancreatol*. 2012;12: 79–84. doi: 10.1016/j.pan.2012.02.005.
38. Posner EB, Haseeb M. Celiac Disease. [Updated 2021 Aug 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK441900/>.
39. Longstreth G.F., Thompson W.G., Chey W.D., Houghton L.A., Mearin F., Spiller R.C. Functional bowel disorders. *Gastroenterology*. 2006;130:1480–1491. doi: 10.1053/j.gastro.2005.11.061.
40. Spiller R, Aziz Q, Creed F, Emmanuel A, Houghton L. Clinical Services Committee of The British Society of Gastroenterology Guidelines on the irritable bowel syndrome: Mechanisms and practical management. *Gut*. 2007;56:1770–1798. doi: 10.1136/gut.2007.119446.
41. Thompson WG, Irvine EJ, Pare P, Ferrazzi S, Rance L. Functional gastrointestinal disorders in Canada: First population-based survey using Rome II criteria with suggestions for improving the questionnaire. *Dig Dis Sci*. 2002;47:225–235. doi: 10.1023/A:1013208713670.
42. Spiller R, Aziz Q, Creed F, Emmanuel A, Houghton L. Clinical Services Committee of The British Society of Gastroenterology Guidelines on the irritable bowel syndrome: Mechanisms and practical management. *Gut*. 2007;56:1770–1798. doi: 10.1136/gut.2007.119446.
43. Miller V, Whitaker K, Morris JA, Whorwell PJ. Gender and irritable bowel syndrome: The male connection. *J Clin Gastroenterol*. 2004;38:558–560. doi: 10.1097/00004836-200408000-00004.
44. Whitehead WE, Burnett CK, Cook EW, III, Taub E. Impact of irritable bowel syndrome on quality of life. *Dig Dis Sci*. 1996;41:2248–2253. doi: 10.1007/BF02071408.
45. El-Salhy M. Irritable bowel syndrome: Diagnosis and pathogenesis. *World J Gastroenterol*. 2012;18:5151–5163.
46. Baumgart DC, Carding SR. Inflammatory bowel disease: cause and immunobiology. *Lancet*. 2007;369:1627–1640.
47. Kk Turner D, Ricciuto A, Lewis A, D'Amico F, Dhaliwal J. International Organization for the Study of IBD. STRIDE-II: An Update on the Selecting Therapeutic Targets in Inflammatory Bowel Disease (STRIDE) Initiative of the International Organization for the Study of IBD (IOIBD): Determining Therapeutic Goals for Treatment-Target strategies in IBD. *Gastroenterology*. 2021 Apr;160(5):1570-1583. doi: 10.1053/j.gastro.2020.12.031.
48. Franke A, McGovern DPB, Barrett JC. Genome-wide meta-analysis increases to 71 the number of confirmed Crohn's disease susceptibility loci. *Nat Genet*. 2010;42:1118–1125.
49. Kornbluth A, Sachar DB. Practice Parameters Committee of the American College of Gastroenterology. Ulcerative colitis practice guidelines in adults: American College of Gastroenterology, Practice Parameters Committee. *Am J Gastroenterol*. 2010;105:501–523.
50. Broome U, Bergquist A. Primary sclerosing cholangitis, inflammatory bowel disease, and colon cancer. *Semin Liver Dis*. 2006;26:31–41.
51. Baumgart DC, Sandborn WJ. Inflammatory bowel disease: clinical aspects and established and evolving therapies. *Lancet*. 2007; 369:1641–1657.
52. Lichtenstein GR, Hanauer SB, Sandborn WJ; Practice Parameters Committee of American College of Gastroenterology. Management of Crohn's disease in adults. *Am J Gastroenterol*. 2009;104:465–483, quiz 464-484.
53. JMoodecky NA, Soon IS, Rabi DM, Ghali WA, Ferris M. Increasing incidence and prevalence of the inflammatory bowel diseases with time, based on systematic review. *Gastroenterology*. 2012;142:46–54.e42. doi: 10.1053/j.gastro.2011.10.001.].
54. Karen M. Horton, Frank M. Corl, and Elliot K. Fishman. CT Evaluation of the Colon: Inflammatory Disease. *RadioGraphics* 2000 20:2, 399-418.
55. Siegel RL, Miller KD, Goding Sauer A, Fedewa SA, Butterly LF. Colorectal cancer statistics, 2020. *CA Cancer J Clin*. 2020 May;70(3):145-164. doi: 10.3322/caac.21601.
56. Siegel RL. Global patterns and trends in colorectal cancer incidence in young adults. *Gut* 68, 2179–2185 (2019).
57. Agarwal SK. Diet and Non-communicable Diseases: Part I. Cardiovascular Diseases, Respiratory Diseases, Obesity, Depression, Liver Diseases. *Clinical Medicine Insights*. 2021; 158-180. <http://doi.org/10.52845/CMI/2021-2-3-7>.
58. Agarwal SK. Diet and Non-communicable Diseases: Part II. Cardiovascular Diseases, Respiratory Diseases, Obesity, Depression, Liver Diseases. *Clinical Medicine Insights*. 2021; 181-200. <http://doi.org/10.52845/CMI/2021-2-3-8>.
59. <https://www.who.int/news-room/factsheets/detail/healthy-diet> - accessed January 18, 2022.
60. Zhang M, Hou ZK, Huang ZB, Chen XL, Liu FB. Dietary and Lifestyle Factors Related to Gastroesophageal Reflux Disease: A Systematic Review. *Ther Clin Risk Manag*. 2021 Apr 15; 17:305-323. doi: 10.2147/TCRM.S296680.
61. <https://www.niddk.nih.gov/>.

62. Kaltenbach T, Crockett S, Gerson LB. Are lifestyle measures effective in patients with gastroesophageal reflux disease? An evidence-based approach. *Arch Intern Med*. 2006; 166:965–971.
63. Caufield SP, Schafer TW. Peptic Ulcer Disease. American College of Gastroenterology. <https://gi.org/topics/peptic-ulcer-disease/>. Published 2012. Accessed July 24, 2019, 2019. <https://gi.org/topics/peptic-ulcer-disease/>.
64. Ryan-Harshman M, Aldoori W. How diet and lifestyle affect duodenal ulcers. Review of the evidence. *Can Fam Physician*. 2004;50: 727–732.
65. Anderson JW, Baird P, Davis RH Jr, Ferreri S, Knudtson M, Koraym A. Health benefits of dietary fiber. *Nutr Rev*. 2009;67(4): 188–205. doi: 10.1111/j.1753-4887.2009.00189.x.
66. Nair MR, Chouhan D, Sen Gupta S, Chattopadhyay S. Fermented Foods: Are They Tasty Medicines for Helicobacter pylori Associated Peptic Ulcer and Gastric Cancer? *Front Microbiol*. 2016 Jul 25;7:1148. doi: 10.3389/fmicb.2016.01148.
67. Marco ML, Heeney D, Binda S. Health benefits of fermented foods: microbiota and beyond. *Curr Opin Biotechnol*. 2017;44:94–102.
68. Wu P., Li L., Sun W. Efficacy comparisons of enteral nutrition and parenteral nutrition in patients with severe acute pancreatitis: A meta-analysis from randomized controlled trials. *Biosci. Rep*. 2018;38 doi: 10.1042/BSR20181515.
69. Arvanitakis M., Ockenga J., Bezmarevic M., Gianotti L., Krznaric Z. Espen guideline on clinical nutrition in acute and chronic pancreatitis. *Clin. Nutr*. 2020;39:612–631. doi: 10.1016/j.clnu.2020.01.004.
70. <https://health.clevelandclinic.org/best-and-worst-foods-for-pancreatitis-pain/>.
71. Kuan LL, Dennison AR, Garcea G. Prevalence and Impact of Sarcopenia in Chronic Pancreatitis: A Review of the Literature. *World J Surg*. 2021; 45:590–597.
72. Arvanitakis M, Ockenga J, Bezmarevic M, Gianotti L, Krznarić Ž. ESPEN guideline on clinical nutrition in acute and chronic pancreatitis. *Clin Nutr*. 2020;39:612–631.
73. Löhr JM, Dominguez-Munoz E, Rosendahl J, Besselink M, Mayerle J. EU/UEG Working Group. United European Gastroenterology evidence-based guidelines for the diagnosis and therapy of chronic pancreatitis (HaPanEU) United European Gastroenterol J. 2017;5:153–199.
74. Roberts KM, Golian P, Nahikian-Nelms M, Hinton A, Madril P. Does the Healthy Eating Index and Mediterranean Diet Score Identify the Nutritional Adequacy of Dietary Patterns in Chronic Pancreatitis? *Dig Dis Sci*. 2019;64:2318–2326.
75. Lindkvist B, Dominguez-Munoz JE, Luaces-Regueira M. Serum nutritional markers for prediction of pancreatic exocrine insufficiency in chronic pancreatitis. *Pancreatology* 2012;12:305–10.
76. Bascuñán KA, Vespa MC, Araya M. Celiac disease: understanding the gluten-free diet. *Eur J Nutr*. 2017 Mar;56(2):449-459. doi: 10.1007/s00394-016-1238-5.
77. Itzlinger A, Branchi F, Elli L, Schumann M. Gluten-Free Diet in Celiac Disease—Forever and for All? *Nutrients*. 2018 Nov 18;10(11):1796. doi: 10.3390/nu10111796.
78. Simren M, Mansson A, Langkilde AM. Food-related gastrointestinal symptoms in the irritable bowel syndrome. *Digestion*. 2001;63:108–15.
79. Ostgaard H, Hausken T, Gundersen D, El-Salhy M. Diet and effects of diet management on quality of life and symptoms in patients with irritable bowel syndrome. *Mol Med Rep*. 2012;5:1382–1390.
80. El-Salhy M, Gundersen D. Diet in irritable bowel syndrome. *Nutr J*. 2015;14:36.
81. Bohn L, Storsrud S, Liljebo T. Diet low in FODMAPs reduces symptoms of irritable bowel syndrome as well as traditional dietary advice: a randomized controlled trial. *Gastroenterology*. 2015;149:1399–407.
82. Schumann D, Klose P, Lauche R, Dobos G, Langhorst J, Cramer H. Low fermentable, oligo-, di-, mono-saccharides and polyol diet in the treatment of irritable bowel syndrome: A systematic review and meta-analysis. *Nutrition*. 2018;45:24–31.
83. Dionne J, Ford AC, Yuan Y. A systematic review and meta-analysis evaluating the efficacy of a gluten-free diet and a low FODMAPs diet in treating symptoms of irritable bowel syndrome. *Am J Gastroenterol*. 2018;113:1290–300.
84. <https://gi.org/topics/low-fodmap-diet/>.
85. Catassi G, Lionetti E, Gatti S, Catassi C. The Low FODMAP Diet: Many Question Marks for a Catchy Acronym. *Nutrients*. 2017;9:292.
86. Staudacher HM, Whelan K. The low FODMAP diet: recent advances in understanding its mechanisms and efficacy in IBS. *Gut*. 2017;66:1517–1527.
87. Asakura H, Suzuki K, Kitahora T, Morizane T. Is there a link between food and intestinal microbes and the occurrence of Crohn's disease and ulcerative colitis? *J Gastroenterol Hepatol*. 2008;23:1794–801.
88. Hou JK, Abraham B, El-Serag H. Dietary intake and risk of developing inflammatory bowel disease: a systematic review of the literature. *American Journal of Gastroenterology*. 2011;106:563–73)
89. Investigators IBDiES. Tjonneland A, Overvad K. Linoleic acid, a dietary n-6 polyunsaturated fatty acid, and the aetiology of ulcerative colitis: a nested case-control study within a European prospective cohort study. *Gut*. 2009;58:1606–11.

90. Schmitz, G. and Ecker, J. (2008) The opposing effects of n-3 and n-6 fatty acids. *Prog. Lipid Res.* 47, 147–155.
91. Patterson E, Wall R, Fitzgerald GF, Ross RP, Stanton C. Health implications of high dietary omega-6 polyunsaturated Fatty acids. *J Nutr Metab.* 2012;2012:539426. doi:10.1155/2012/539426.
92. Ananthkrishnan AN, Khalili H, Konijeti GG. Long-term intake of dietary fat and risk of ulcerative colitis and Crohn's disease. *Gut.* 2013.
93. Brown AC, Rampertab SD, Mullin GE. Existing dietary guidelines for Crohn's disease and ulcerative colitis. *Expert Rev Gastroenterol Hepatol.* 2011;5:411–25.
94. Lambert K, Pappas D, Miglioretto C, Javadpour A, Reveley H. Systematic review with meta-analysis: dietary intake in adults with inflammatory bowel disease. *Aliment Pharmacol Ther.* 2021 Sep;54(6):742-754. doi: 10.1111/apt.16549.
95. Hou JK, Abraham B, El-Serag H. Dietary intake and risk of developing inflammatory bowel disease: a systematic review of the literature. *Am J Gastroenterol.* 2011;106:563–573. doi: 10.1038/ajg.2011.44.
96. Pituch-Zdanowska A, Banaszkiwicz A, Albrecht P. The role of dietary fibre in inflammatory bowel disease. *Prz Gastroenterol.* 2015;10(3):135-141. doi:10.5114/pg.2015.52753.
97. <https://www.medicalnewstoday.com/articles/nutritional-deficiencies-and-crohns-disease#signs-of-malnutrition>.
98. Main AN, Mills PR, Russell RI. Vitamin A deficiency in Crohn's disease. *Gut* 1983;24:1169-1175
99. Soares-Mota M, Silva TA, Gomes LM. High prevalence of vitamin A deficiency in Crohn's disease patients according to serum retinol levels and the relative dose-response test. *World J Gastroenterol.* 2015;21(5):1614-1620. doi:10.3748/wjg.v21.i5.1614.
100. Toriki M, Gholamrezaei A, Mirbagher L, Danesh M, Kheiri S, Emami MH. Vitamin D deficiency associated with disease activity in patients with inflammatory bowel diseases. *Dig Dis Sci.* 2015;60:3085–3091. doi: 10.1007/s10620-015-3727-4.
101. Vernia P, Loizos P, Di Giuseppeantonio I, Amore B, Chiappini A, Cannizzaro S. Dietary calcium intake in patients with inflammatory bowel disease. *J Crohns Colitis.* 2014 Apr;8(4):312-7. doi: 10.1016/j.crohns.2013.09.008.
102. Jorgensen SP, Agnholt J, Glerup H. Clinical trial: vitamin D3 treatment in Crohn's disease – a randomized double-blind placebo-controlled study. *Aliment Pharmacol Ther.* 2010;32:377–83.
103. Manzel A, Muller DN, Hafler DA. Role of “Western diet” in inflammatory autoimmune diseases. *Curr Allergy Asthma Rep* 2014;14:404.
104. Ananthkrishnan AN, Khalili H, Song M. High school diet and risk of Crohn's disease and ulcerative colitis. *Inflammatory Bowel Diseases* 2015:1.
105. Khalili H, Håkansson N, Chan SS. Adherence to a Mediterranean diet is associated with a lower risk of later-onset Crohn's disease: results from two large prospective cohort studies. *Gut* 2020;gutjnl-2019–319505.
106. Mozaffari H., Daneshzad E., Larijani B., Bellissimo N., Azadbakht L. Dietary intake of fish, n-3 polyunsaturated fatty acids, and risk of inflammatory bowel disease: A systematic review and meta-analysis of observational studies. *Eur. J. Nutr.* 2020;59:1–17. doi: 10.1007/s00394-019-01901-0.
107. Potter J., Brown L., Williams R.L., Byles J., Collins C.E. (2016). Diet Quality and Cancer Outcomes in Adults: A Systematic Review of Epidemiological Studies. *Int J Mol Sci*, 17(7), 1052. Published 2016 Jul 5. <https://doi:10.3390/ijms17071052>.
108. Bodén S., Myte R., Wennberg M., Harlid S. (2019). The Inflammatory Potential of Diet in Determining Cancer Risk; A Prospective Investigation of Two Dietary Pattern Scores. *PLoS ONE*, 14, e0214551. <https://doi:10.1371/journal.pone.0214551>.
109. Johnson IT. New approaches to the role of diet in the prevention of cancers of the alimentary tract. *Mutat Res.* 2004 Jul 13;551(1-2):9-28. doi: 10.1016/j.mrfmmm.2004.02.017.
110. Zhang FF, Cudhea F, Shan Z, Michaud DS, Imamura F, et al. Preventable Cancer Burden Associated With Poor Diet in the United States. *JNCI Cancer Spectr.* 2019 May 22;3(2):pkz034. doi: 10.1093/jncics/pkz034.
111. Steinmetz KA, Potter JD. Vegetables, fruit, and cancer prevention: a review. *J Am Diet Assoc.* 1996;96:1027–1039,
112. Islami F, Goding Sauer A, Miller KD. Proportion and number of cancer cases and deaths attributable to potentially modifiable risk factors in the United States. *CA Cancer J Clin.* 2018;68(1):31–54. doi: 10.3322/caac.21440.
113. Donaldson M.S. Nutrition and Cancer: A Review of the Evidence for an Anti-Cancer Diet. *Nutr. J.* 2004;3:19. doi: 10.1186/1475-2891-3-19.
114. Behrens G, Gredner T, Stock C, Leitzmann MF, Brenner H, Mons U. Cancers Due to Excess Weight, Low Physical Activity, and Unhealthy Diet. *Dtsch Arztebl Int.* 2018;115(35-36):578-585. doi:10.3238/arztebl.2018.0578.
115. Xu X, Yu E, Gao X. Red and processed meat intake and risk of colorectal adenomas: a meta-analysis of observational studies. *Int J Cancer* 2013;132:437-48.
116. World Cancer Research Fund, American Institute for Cancer Research. Diet, nutrition, physical

- activity and cancer: a global perspective. Continuous update project expert report 2018.
117. Veettil SK, Wong TY, Loo YS, Playdon MC, Lai NM, Giovannucci EL, Chaiyakunapruk N. Role of Diet in Colorectal Cancer Incidence: Umbrella Review of Meta-analyses of Prospective Observational Studies. *JAMA Netw Open*. 2021 Feb 1;4(2):e2037341. doi: 10.1001/jamanetworkopen.2020.37341.
118. <http://pressroom.cancer.org/IslamiPAF2017> – accessed October 29, 2021.
119. Kassianos, A. P., Raats, M. M. , Gage, H., & Peacock, M. (2015). Quality of life and dietary changes among cancer patients: A systematic review. *Quality of Life Research*, 24, 705–719.
120. Thomson CA, McCullough ML, Wertheim BC. Nutrition and physical activity cancer prevention guidelines, cancer risk, and mortality in the Women’s Health Initiative. *Cancer Prev Res*. 2014; 7: 42- 53.
121. Schwedhelm C, Boeing H, Hoffmann G, Aleksandrova K, Schwingshackl L. Effect of diet on mortality and cancer recurrence among cancer survivors: a systematic review and meta-analysis of cohort studies. *Nutr Rev*. 2016 Dec;74(12):737-748. doi: 10.1093/nutrit/nuw045.
122. Murphy CC, Gerber DE, Pruitt SL. Prevalence of prior cancer among persons newly diagnosed with cancer: an initial report from the surveillance, epidemiology, and end results program. *JAMA Oncology*. 2017; doi:10.1001/jamaoncol.2017.3605.
123. Kushi LH, Doyle C, McCullough M. American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin*. 2012;62:30-67.
124. Norat T, Scoccianti C, Boutron-Ruault MC. European Code Against Cancer 4th Edition: diet and cancer. *Cancer Epidemiol*. 2015;39(1):S56–S66.
125. Sinha R, Knize MG, Salmon CP. Heterocyclic amine content of pork products cooked by different methods and to varying degrees of doneness. *Food Chem Toxicol*. 1998; 36: 289- 297.
126. Berg G, Erlacher A, Smalla K, Krause R. Vegetable microbiomes: is there a connection among opportunistic infections, human health and our 'gut feeling'?. *Microb Biotechnol*. 2014;7(6):487-495. doi:10.1111/1751-7915.12159.

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