# AN OVERVIEW OF SELECTED DIGESTIVE SYSTEM DISORDERS WITH IMPLICATIONS FOR NOURISHING THE BODY

In Chapter 2, digestion is defined as a process by which food is broken down mechanically and chemically in the gastrointestinal (GI) tract. Digestion ultimately provides nutrients ready for absorption into the body through the cells of the GI tract, principally the cells (enterocytes) of the small intestine. Secretions required to digest nutrients are produced by multiple organs of the GI tract. These secretions include primarily enzymes, but also hydrochloric acid important for gastric digestion, and bicarbonate and bile important for digestion and absorption in the intestine. If one or more organs malfunctions because of disease, fewer secretions may be synthesized and released into the GI tract. Without secretions, or with less than normal amounts of secretions, nutrient digestion may be impaired, resulting in nutrient malabsorption.

Many conditions or diseases alter the function of organs of the GI tract and thus affect digestion. For example, some GI tract diseases may cause decreased synthesis and release of secretions needed for nutrient digestion. Other conditions or diseases that affect the GI tract—for example, malfunction of sphincters—can alter motility or clearing of the GI contents through the organs of the GI tract. Clearing problems may cause back fluxes (refluxes) of secretions from, for example, the stomach into the esophagus (remember, normally the contents of the GI tract move from the esophagus to the stomach, and not vice versa). Conditions in which the GI mucosa is inflamed or damaged, as well as conditions that increase transit time or speed up the movement of GI contents (food and nutrients) through the GI tract, typically result in nutrient malabsorption because the body does not have enough time to digest and absorb nutrients.

An understanding of the physiology of the GI tract and its accessory organs, and of the diseases affecting the GI tract, is essential to understanding how to modify a person's diet from the standard dietary recommendations for healthy populations of the United States. This perspective addresses, in a general fashion, four disorders that affect the gastrointestinal tract and outlines the implications of these conditions for nourishing the body.

## GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux disease (GERD) is a disorder marked by reflux or backward flow of gastric contents (acidic chyme) from the stomach to the esophagus. After food is chewed and swallowed, the food enters the esophagus and then passes through the gastroesophageal sphincter into the stomach. Normally, the gastroesophageal sphincter displays a relatively high pressure that prevents the reflux of stomach contents into the esophagus. However, changes or decreases in the gastroesophageal sphincter pressure, sometimes called lower esophageal sphincter incompetence, can ultimately result in GERD. Increases in abdominal pressure, such as may occur with overeating, bending, lifting, lying down, vomiting, or coughing, also can increase reflux and cause GERD.

Recurrent reflux of gastric contents, including hydrochloric acid, into the esophagus from the stomach can damage and inflame the esophageal mucosa and result in reflux esophagitis (inflammation of the esophagus caused by the refluxed gastric contents). The severity of the esophagitis depends in part on the volume and acidity of the gastric contents that are refluxed and on the length of time the gastric contents are in contact with the esophageal mucosa. The more acidic the contents, and the longer the contents are in contact with the mucosa, the more damage results. Weak peristalsis and delays in gastric emptying are likely to prolong contact time and increase damage. The resistance of the esophageal mucosa also affects the severity of the damage. Repeated bouts of GERD resulting in reflux esophagitis cause, to varying degrees, esophageal edema (swelling); esophageal tissue damage, including erosion and ulceration; blood vessel (usually capillary) damage; spasms; and fibrotic tissue formation, which can cause a narrowing (stricture) within the esophagus.

A person experiencing GERD or reflux esophagitis typically complains of heartburn, that is, a burning sensation in the midchest region, but may also complain of excessive belching and/or coughing. The symptoms usually occur within an hour of eating and worsen if the person lies down soon after eating.

To address nutrition implications of this condition, we first need to reexamine some of the foods, nutrients, or substances in foods that influence gastroesophageal sphincter pressure, that may promote increased acid production, and that may irritate an inflamed esophagus. Several substances decrease gastroesophageal sphincter pressure, including high-fat foods, chocolate, nicotine, alcohol, and carminatives. Carminatives are volatile oil extracts of plants, most often oils of spearmint and peppermint. Other substances increase gastric secretions, especially acid production. Alcohol, calcium, decaffeinated and caffeinated coffee, and tea (specifically, methylxanthines) stimulate gastric secretions, including hydrochloric acid. Citrus products and other acidic foods or beverages, as well as some spices, are known to directly irritate an inflamed esophagus. Ingesting these substances or foods is likely to aggravate irritated esophageal mucosa.

Based on this knowledge, some of the recommendations for the patient with GERD or reflux esophagitis include:

- avoiding substances that can further decrease gastroesophageal sphincter pressure, which is already low because of the condition
- avoiding substances that may promote the secretion of acid, which would be present in higher concentrations than normal if refluxed
- avoiding foods or substances that may irritate an inflamed esophagus

To implement these recommendations, people with GERD or reflux esophagitis must be told which foods or substances to avoid, such as high-fat foods or meals, chocolate, coffee, tea, alcohol, carminatives such as peppermint and spearmint, citrus products, acidic foods, and spices such as red and black pepper, nutmeg, cloves, and chili powder.

In addition to avoiding substances that reduce gastroesophageal sphincter pressure, that promote the secretion of acid, and that can irritate an inflamed esophagus, recommendations can also include increasing the intake of foods or nutrients that increase gastroesophageal sphincter pressure. Protein is a nutrient that increases gastroesophageal sphincter pressure. Consequently, a higher than normal protein intake is encouraged; however, excessive protein intakes, especially from foods high in calcium such as dairy products, are not recommended. The reason for avoiding excessively high intakes of dairy foods relates to the fact that the amino acids and peptides (generated by digesting the protein in the dairy products) and calcium in dairy products are known to stimulate gastrin release. Although gastrin increases gastroesophageal sphincter pressure, it is also a potent stimulator of hydrochloric acid secretion.

In addition to noting the previously stated nutrition recommendations, remember that reflux is more likely to occur with increased gastric volume (i.e., eating large meals), increased gastric pressure (e.g., from obesity), and placement of gastric contents near the sphincter (i.e., bending, lying down, or assuming a recumbent position). Thus, recommendations for people with GERD or reflux esophagitis should include:

- eating smaller meals (and avoiding large ones)
- drinking fluids between meals, instead of with a meal, to help minimize large increases in gastric volume

- losing weight, if the person is overweight or obese
- avoiding tight-fitting clothes
- avoiding lying down, lifting, or bending for at least 2 hours after eating

## INFLAMMATORY BOWEL DISEASES

Inflammatory bowel diseases (IBDs) include ulcerative colitis and Crohn's disease and are characterized by acute, relapsing, or chronic inflammation of various segments of the GI tract, especially the intestines. Although the causes of IBDs are unclear, nutrient malabsorption is a significant problem for several reasons. First, because of the disease-associated inflammation of the mucosa, brush border disaccharidase and peptidase activities are diminished, and thus nutrient digestion is impaired. Second, nutrient transit time is typically decreased—that is, GI tract contents move through the GI tract more quickly than usual and thus leave little time for absorption. Third, malabsorption occurs because of direct damage to the absorptive mucosa cells (enterocytes). Exacerbating the poor nutrient absorption is poor food intake, which is especially common during acute attacks.

Manifestations of IBDs include excessive diarrhea and steatorrhea (large amounts of fat in the feces), which may occur up to 20 times per day. Diarrhea is associated with increased losses of fluid and electrolytes (especially potassium) from the body. Fluid and electrolyte imbalance or even dehydration can result. Blood is often present in the feces, especially if deeper areas of the GI mucosa are severely inflamed or ulcerated. Loss of blood impairs the body's protein and mineral (especially iron) status. If IBD has affected the ileum (as is common with Crohn's disease), vitamin $B_{12}$ absorption may be impaired (this vitamin is absorbed in the ileum), reabsorption of bile salts from the ileum may be diminished, and fat malabsorption may occur. Although pancreatic lipase is available to hydrolyze dietary triacylglycerols, the lack of sufficient bile or diminished bile function caused by bacterial alteration of bile can decrease micelle formation and thus decrease absorption of fatty acids and fat-soluble vitamins into the enterocyte. Unabsorbed fatty acids bind to calcium and magnesium in the lumen of the intestine; the resulting insoluble complex, sometimes called a soap, is excreted in the feces.

Dietary recommendations for people with IBD are aimed at replacing nutrient losses, correcting nutrient imbalances, and improving nutrition status. Some of these dietary recommendations include:

- increasing iron intake above the Recommended Dietary Allowance (RDA) because of increased iron losses with the bloody diarrhea and decreased absorption
- following a low-fat diet because fat absorption is impaired
- increasing calcium and magnesium intake because absorption of these nutrients is diminished by soap formation and overall malabsorption with diarrhea
- consuming a higher amount of protein than normal because protein is lost from the blood into the feces with bloody diarrhea and malabsorption of amino acids
- taking fat-soluble vitamin supplements, possibly in a water-miscible form to improve absorption
- increasing fluid and electrolyte intakes to rehydrate and restore electrolyte balance
- increasing overall intake of nutrients to meet energy and nutrient needs

Easily digestible, carbohydrate-rich foods that are low in fiber, high-protein low-fat foods, and lactose-free foods should provide the bulk of the person's energy needs if oral intake is deemed appropriate. Medium-chain triacylglycerol (MCT) oil, which is absorbed directly into portal blood and does not need bile for absorption, may be added in small amounts to different foods throughout the day to increase energy intake. Sometimes, however, complete rest of the GI tract is needed, and a person with IBD may need to be fed intravenously (by parenteral nutrition).

## CELIAC DISEASE

Celiac disease, also called gluten- or gliadin-sensitive enteropathy or celiac sprue, results from an intolerance to gluten. Gluten is the general name for storage proteins, also called prolamins, in grains. Grains vary in their storage proteins, however, and in people with celiac disease, three storage proteins—secalin in rye, hordein in barley, and gliadin in wheat—appear to elicit or trigger the problems.

Consuming any of these grains alone, or foods made with any of these grains, triggers both immune and inflammatory responses in a person with celiac disease. Although the severity of the condition varies, the small intestine of someone with celiac disease becomes inflamed; lymphocytes and other immune system cells and the cytokines produced by the cells invade and attack the mucosa. The villi typically become atrophied or blunted, with corresponding changes in the crypt to villous height ratio. Because of the villi destruction, digestion and absorption become severely impaired. Manifestations of celiac disease include diarrhea, abdominal pain, malabsorption, and weight loss. Over time and if untreated, an infant or child with celiac disease may even exhibit signs of protein-energy malnutrition characterized by poor somatic muscle mass, hypotonia, abdominal distension, peripheral edema, depleted subcutaneous fat stores, and poor growth. Older children also may complain of constipation, nausea, reflux, and vomiting. This disorder affects other parts of the body as well as the intestines. Extraintestinal symptoms often include skin rashes, and muscle and joint pain. Fertility problems, especially in women with celiac disease, have been noted, along with bone problems including delayed bone growth and development and ultimately osteoporosis.

The cause of celiac disease is not clear, but it is thought to have a genetic component. The condition has been linked to the presence of several specific human leukocyte antigens. Diagnosis of celiac disease is based on the presence of a combination of serum antibody markers and biopsy of the small intestine.

Treatment of celiac disease requires lifelong exclusion of any form of any product that contains rye, barley, or wheat. However, because many foods contain combinations of grains, the list of foods to exclude is extensive. For example, grains such as triticale, which is a combination of rye and wheat, and malt, which is a partial hydrolysate of barley, cannot be consumed. A list of all the foods allowed and not allowed with celiac disease is beyond the scope of this perspective. Fortunately, food labels now must state if products contain wheat, and products that are gluten free often advertise this on the label. Further, the tremendous increase in the availability of gluten-free products in grocery stores has made living with celiac disease a little easier.

## CHRONIC PANCREATITIS

Pancreatitis, or inflammation of the pancreas, provides an excellent example of the nutritional ramifications of a condition affecting an accessory organ of the GI tract. Remember that the exocrine portion of the pancreas produces several enzymes needed to digest all nutrients. *Chronic* refers to an ongoing or long-lasting situation.

Chronic pancreatitis can result from long-term excessive use of alcohol, gallstones, liver disease, viral infections, and use of certain medications, among other factors. With time, sections of pancreatic tissue become dysfunctional. Acinar cells, for example, can ultimately fail to produce sufficient digestive enzymes and juices. Consequently, a person with chronic pancreatitis experiences pain, especially with eating, as well as nausea, vomiting, and diarrhea. The diarrhea results in part from the maldigestion, with resulting malabsorption of several nutrients.

Diminished secretion of pancreatic lipase into the duodenum, caused by the chronic pancreatitis, results in maldigestion of fat and thus malabsorption of fat and fat-soluble vitamins. Fat is malabsorbed because not enough pancreatic lipase is available to hydrolyze the fatty acids from the triacylglycerols. This hydrolysis is necessary for fatty acids and monoacylglycerols to form micelles, the form in which the fatty acids are carried into the enterocyte for absorption. Thus, with pancreatitis, the insufficiency of enzymes available for fat hydrolysis necessitates a low-fat diet.

In addition to insufficient pancreatic lipase secretion, bicarbonate secretion into the duodenum is also diminished with pancreatitis. Bicarbonate, in part, increases the pH of the small intestine. Intestinal enzymes function best at an alkaline pH, which is provided by the release of bicarbonate into the intestine.

Oral supplements of pancreatic enzymes may be needed to replace the diminished output of these enzymes by the malfunctioning, inflamed pancreas. Medications such as antacids, $H_2$ receptor blockers, or proton pump

inhibitors may also be needed. The medications are taken to diminish acid production and thus increase intestinal pH. In effect, they replace the bicarbonate and thus help maintain an appropriate pH for enzyme function. Exogenous insulin may also have to be administered if insulin is no longer produced in sufficient quantities by the damaged pancreatic endocrine cells.

These four conditions illustrate how diseases that affect the GI tract—malfunction of a sphincter (GERD and reflux esophagitis); destruction of enterocyte function (IBD), especially destruction of the enterocyte absorptive surface (celiac disease); and chronic malfunction of a GI tract accessory organ that provides secretions needed for nutrient digestion (pancreatitis)—affect the body's ability to digest and absorb nutrients. Furthermore, these conditions illustrate how nutrient intakes must deviate from recommended levels—in some cases to lower levels, and in other cases to higher levels—depending on the condition. Such dietary modifications are typical of many conditions that affect not only the gastrointestinal tract but also other organ systems.