bowel motility is accelerated (e.g., ulcerative colitis). Obstructive disorders (e.g., Hirschsprung disease) also cause secondary malabsorption from enterocolitis.

Anatomic defects, such as extensive resection of the bowel or SBS, affect digestion by decreasing the transit time of substances and affect absorption by severely compromising the absorptive surface.

Celiac Disease (Gluten-Sensitive Enteropathy)

Celiac disease, also known as *gluten-induced enteropathy, gluten-sensitive enteropathy,* and *celiac sprue,* is a permanent intestinal intolerance to dietary gluten, a protein present in wheat, barley, rye, and oats that causes damage to the villi in the small intestine (Paul, Johnson, and Speed, 2013). The incidence is variable and has been reported in 1 in 141 people (Rubio-Tapia, Ludvigsson, Brantner, et al, 2012). The disease is seen more frequently in Europe and the United States; it is rarely reported in Asians or African Americans (Reilly and Green, 2012). As adults, it is more prevalent in women than men, but there is equal distribution of cases among children (Reilly and Green, 2012). Although the exact cause is unknown, it is generally accepted that celiac disease is an immunologically mediated small intestine enteropathy. The mucosal lesions contain features that suggest both humoral and cell-mediated immunologic overstimulation.

Pathophysiology

Celiac disease is characterized by villous atrophy in the small intestine in response to the protein gluten. When individuals are unable to digest the gliadin component of gluten (an accumulation of a toxic substance that is damaging to the mucosal cells), damage to the mucosa of the small intestine leads to villous atrophy, hyperplasia of the crypts, and infiltration of the epithelial cells with lymphocytes. Villous atrophy leads to malabsorption caused by the reduced absorptive surface area (Fig. 22-10).