children older than 10 years old, and secondary ulcers are more common in infants and children with underlying disease, and children taking nonsteroidal antiinflammatory drugs (NSAIDs), corticosteroids, or sodium valproate medications (Sullivan, 2010).

Etiology

The exact cause of PUD is unknown, although infectious, genetic, and environmental factors are important. There is an increased familial incidence, likely due to *H. pylori,* which is known to cluster in families (Sullivan, 2010). *H. pylori* is a microaerophilic, gram-negative, slow-growing, spiral-shaped, and flagellated bacterium known to colonize the gastric mucosa in about half of the population of the world (Ertem, 2012). *H. pylori* synthesizes the enzyme urease, which hydrolyses urea to form ammonia and carbon dioxide. Ammonia then absorbs acid to form ammonium, thus raising the gastric pH. *H. pylori* may cause ulcers by weakening the gastric mucosal barrier and allowing acid to damage the mucosa. It is believed that it is acquired via the fecal–oral route, and this hypothesis is supported by finding viable *H. pylori* in feces.

In addition to ulcerogenic drugs, both alcohol and smoking contribute to ulcer formation. There is no conclusive evidence to implicate particular foods, such as caffeine-containing beverages or spicy foods, but polyunsaturated fats and fiber may play a role in ulcer formation. Psychological factors may play a role in the development of PUD, and stressful life events, dependency, passiveness, and hostility have all been implicated as contributing factors.

Pathophysiology

Most likely, the pathology is caused by an imbalance between the destructive (cytotoxic) factors and defensive (cytoprotective) factors in the GI tract. The toxic mechanisms include acid, pepsin, medications such as aspirin and NSAIDs, bile acids, and infection with *H. pylori.* The defensive factors include the mucus layer, local bicarbonate secretion, epithelial cell renewal, and mucosal blood flow. Prostaglandins play a role in mucosal defense because they stimulate both mucus and alkali secretion. The primary mechanism that prevents the development of peptic ulcer is the secretion of