Figure 3.1. Pathophysiology of Increased Intestinal Permeability.

Figure 3.2. Intestinal Barrier Defenses in the Healthy Gut. The gut lining functions as a selective gate or barrier, allowing the passage and transport of small and medium-sized molecules of essential nutrients and communication peptides. At the same time, the membrane bars the passage of larger molecules, including many toxins and allergenic substances. The cells of the gut lining play an essential role in this process by allowing some substances through and barring others.
Figure 3.3. Injury to the Intestine and Altered Permeability. Damage to the gut can cause defects to its barrier function with resultant systemic dissemination of toxic bacterial by-products and food antigens.

Figure 3.4. Gut Flora and Intestinal Proinflammatory Responses. As a consequence of disruptions in the bacterial flora of the gut, mucosal inflammation can be divided into two categories with respect to effector versus regulatory T-cell imbalance. In the first type, bacterial-drive responses are abnormally robust and the intrinsic homeostasis of the mucosal immune system (regulatory) is overwhelmed, and there is a bias towards inflammation. Alternatively, effector-cell responses to gut flora can be normal, but regulatory-cell responses (i.e., genetically determined) can be weak; again, inflammation is the result.

Figure 3.5. Schematic representation of the pattern of bidirectional brain–gut–microbe interactions. The brain can modulate various functions of the gut, as well as the perception of gut stimuli, via a set of parallel outflow systems that are referred to as the EMS, which include the sympathetic and parasympathetic branches of the ANS, the HPA axis, and endogenous pain-modulation systems. (Mayer, 2000) Reprinted by permission from Macmillan Publishers Ltd: Nature Reviews Gastroenterology and Hepatology (Rhee et al., Nat Rev Gastroenterol Hepatol. 2009 May;6(5):306–314) Copyright 2009.

Figure 3.6. Enterochromaffin cells as bidirectional signal transducers between host and enteric microbiota. Reprinted with permission from Furness, J. B. & Clerc, N. Prog. Brain Res. 1999; 122:159–172.
REFERENCES


