**Figure 41.1.** Type I Hypersensitivity. Immediate hypersensitivity is mediated by IgE. The primary cellular component in this hypersensitivity is the mast cell (as shown in this figure) or basophil. The mechanism of reaction involves preferential production of IgE, in response to certain antigens (allergens). IgE has very high affinity for its receptor on mast cells. A subsequent exposure to the same allergen crosslinks the cell-bound IgE and triggers the release of various pharmacologically active substances. Figure courtesy of University of Indiana.

**Figure 41.2.** Type IV Hypersensitivity. Delayed type hypersensitivity results when an antigen presenting cell (typically a tissue dendritic cell that has picked up an antigen, processed it, and displayed appropriate peptide fragments bound to Class II MHC) is contacted by an antigen-specific Th1 cell patrolling the tissue. The resulting activation of the T-cell produces cytokines such as chemokines (for macrophages, other T-cells and, to a lesser extent, neutrophils), TNF-beta, and IFN-gamma. The consequences are a cellular infiltrate in which mononuclear cells (T-cells and macrophages) tend to predominate. It is usually maximal in 48–72 hours. Figure courtesy of University of Cambridge, Department of Pathology.
Figure 41.3. Intestinal Barrier Function and Loss of Oral Tolerance. Microbial antigens are escorted across the intestinal epithelial cells (IEC) by dendritic cells that sense and sample via toll-like receptors (TLRs) or M cell dependent antigen uptake. Alternatively, antigens can “leak” across the epithelium if there are breaches in the integrity of the intestinal lining. The unregulated transport of microbial antigens can then trigger an immune response beyond the mucosal immune system’s capacity to attenuate. Once the regulatory pathways of oral tolerance are deranged, then inflammatory pathways dominate, and chronic intestinal inflammation can result in those who are genetically susceptible.

Figure courtesy of www.science-autism.org.
Figure 41-4 Reduction in the IBS Symptom severity index improves with higher levels of adherence. Mean change in symptom severity scores at 12 weeks according to degree of adherence. Difference between the groups with high adherence: 101 (95% confidence interval 54, 147).


REFERENCES


