Acute inflammation occurs in response to cell damage resulting from infection or injury. During this process, cellular and plasma-derived factors encourage extravasation, which is the recruitment of circulating immune cells into the affected tissue. These immune cells increase the expression of inflammatory cytokines that recruit additional immune cells. These cells mount an immune response to any invading organisms and further promote, and eventually resolve, the inflammatory response. Chronic inflammation, or expression of these cytokines and receptors at low levels over long periods of time, promotes various pathological conditions including allergies and asthma, cardiovascular system disorders (atherosclerosis), central nervous system disorders (Alzheimer’s disease), fibrosis, and rheumatoid arthritis. The majority of cytokines are inflammatory, including classes such as chemokines, interferons, and interleukins. These cytokines bind and activate their respective receptors to promote inflammatory responses.

Acute inflammation occurs in response to cell damage resulting from infection or injury. During this process, cellular and plasma-derived factors encourage extravasation, the recruitment of circulating immune cells into the affected tissue. These immune cells increase their expression of inflammatory cytokines, recruiting additional immune cells to resolve the infection. The immune response is precisely controlled, and dysregulation of any aspect of the process can result in disease. Chronic inflammation contributes to various pathological and autoimmune conditions including allergy, cardiovascular system disorders, central nervous system disorders, and rheumatoid arthritis. Autoimmunity occurs when immune cells attack the other cells in the same organism. Some immune cell types, such as Th2 cells and alternatively-activated macrophages, promote the allergic response. Recent studies show that inappropriate levels of these cell types can occur during pathological conditions, potentially causing autoimmunity. Analysis of Th2 cell and alternatively-activated macrophage functions during autoimmune responses may identify novel cellular mechanisms that cause these pathophysiological conditions.  
  
The tumor necrosis factor (TNF) superfamily includes 29 receptors in humans that interact with a variety of ligands. These receptors fall into 3 major groups, depending on their cytoplasmic domains: death domain containing, TRAF (TNF receptor associated factor)-interacting motif containing, and “decoy” receptors that have neither of these motifs. The death domain containing receptors initiate cell death programs such as apoptosis and necrosis. The TNF receptors with TRAF-interacting motifs bind with one of the 6 TRAF adaptor proteins to propagate signal transduction. These receptors are involved in inflammatory and immune responses. The final TNF receptor family, the decoy receptors, has no known function but is capable of binding TNF ligands. TNF receptor signaling dysfunction contributes to diseases such as chronic inflammation, Crohn’s disease, and rheumatoid arthritis. Analyzing the expression, regulation, and sequence of TNF signaling genes can help determine their relative importance to the biology of the cellular or disease processes under study.