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Chapter 9 The Humoral Immune Response

Many of the bacteria that cause infectious disease in humans multiply in the extracellular spaces of the body, and most intracellular pathogens spread by moving from cell to cell through the extracellular fluids. The extracellular spaces are protected by the **humoral immune response**, in which antibodies produced by B cells cause the destruction of extracellular microorganisms and prevent the spread of intracellular infections. The activation of B cells and their differentiation into antibody-secreting plasma cells (Fig. 9.1) is triggered by antigen and usually requires helper T cells. The term 'helper T cell' is often used to mean a cell from the T_H2 class of CD4 T cells (see Chapter 8), but a subset of T_H1 cells can also help in B-cell activation. In this chapter we will therefore use the term **helper T cell** to mean any armed effector CD4 T cell that can activate a B cell. Helper T cells also control isotype switching and have a role in initiating somatic hypermutation of antibody variable V-region genes, molecular processes that were described in Chapter 4.

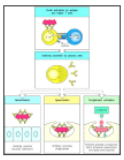

Figure 9.1

The humoral immune response is mediated by antibody molecules that are secreted by plasma cells. Antigen that binds to the B-cell antigen receptor signals B cells and is, at the same time, internalized and processed into peptides that activate armed helper (more...)

Antibodies contribute to immunity in three main ways (see Fig. 9.1). To enter cells, viruses and intracellular bacteria bind to specific molecules on the target cell surface. Antibodies that bind to the pathogen can prevent this and are said to neutralize the pathogen. Neutralization by antibodies is also important in preventing bacterial toxins from entering cells. Antibodies protect against bacteria that multiply outside cells mainly by facilitating uptake of the pathogen by phagocytic cells that are specialized to destroy ingested bacteria. Antibodies do this in either of two ways. In the first, bound antibodies coating the pathogen are recognized by Fc receptors on phagocytic cells that bind to the antibody constant C region (see Section 4-18). Coating the surface of a pathogen to enhance phagocytosis is called opsonization. Alternatively, antibodies binding to the surface of a pathogen can activate the proteins of the complement system, which was described in Chapter 2. **Complement activation** results in complement proteins being bound to the pathogen surface, and these opsonize the pathogen by binding complement receptors on phagocytes. Other complement components recruit phagocytic cells to the site of infection, and the terminal components of complement can lyse certain microorganisms directly by forming pores in their membranes. Which effector mechanisms are engaged in a particular response is determined by the isotype or class of the antibodies produced.

In the first part of this chapter we will describe the interactions of B cells with helper T cells that lead to the production of antibodies, the affinity maturation of this antibody response, the isotype switching that confers functional diversity, and the generation of memory B cells that provide long-lasting immunity to reinfection. In the rest of the chapter we will discuss in detail the mechanisms whereby antibodies contain and eliminate infections.

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