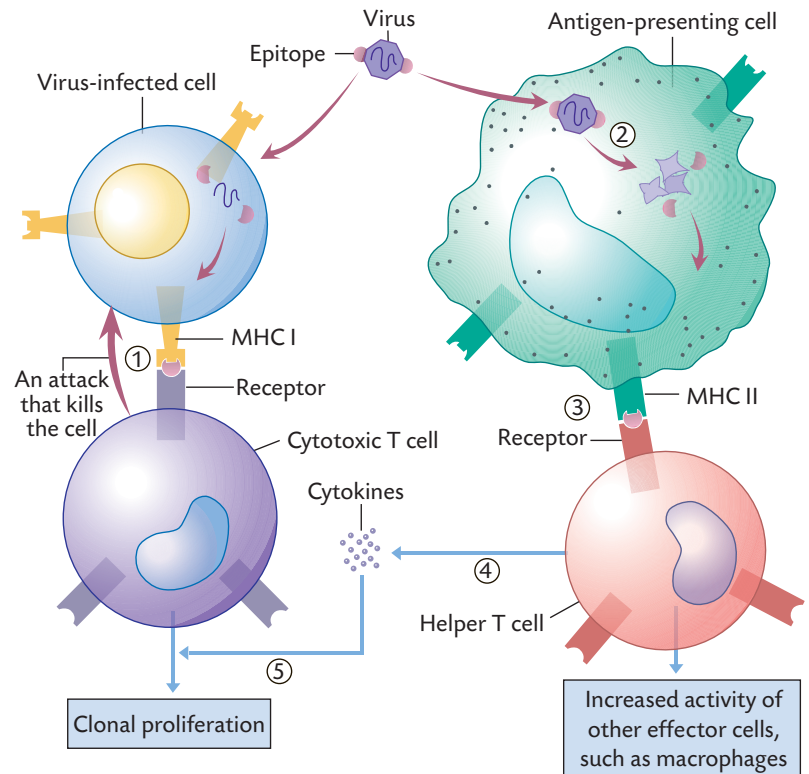


**Figure 10.10** The T lymphocyte reaction. In cells infected with a virus, cell-produced fragments of viral proteins (viral epitopes) are presented on the cell surface bound to MHC class I molecules (1). In this form, the viral epitopes are recognized by cytotoxic T cells. In order for expansion of the particular clone of the cytotoxic T cells to occur, helper T cells must also be activated. This occurs by phagocytosis of the virus by a professional antigen-presenting cell (2), for example, a dendritic cell. In antigen-presenting cells, the viral epitopes are presented on the cell surface together with MHC class I proteins (not shown) as well as MHC class II proteins (3). When the epitopes are presented together with MHC class II molecules, they are recognized by the helper T cells. The activated helper T cells produce cytokines (4) that stimulate differentiation and proliferation of both the helper T cells themselves (not shown) and the activated clone of cytotoxic T cells (5).



**Mode of action of cytotoxic T cells.** When a virus is killed, the virus-infected cell also has to be destroyed. Cytotoxic T cells identify and bind to the complex formed by the viral antigen and MHC class I proteins on the surface of the infected cells. When a cytotoxic T cell has been activated in this manner, it releases proteins that kill the virus-infected cell. Some of the proteins that are released are inserted into the cell membrane of the infected cell as transmembrane pores (perforins). Killing cells by perforating their cell membranes is analogous to the manner in which some of the factors in the complement cascade kill invading microorganisms (p. 339). When the infected cell has been destroyed, virus production from this cell stops. Released viruses may be neutralized by antibodies or destroyed by phagocytes. The cytotoxic T cells are not injured during this process, and therefore can continue to kill more virus-infected cells. Usually, only a few of the body's cells are destroyed in the effort to stop a viral infection.

**Self-tolerance.** When the immune system functions satisfactorily, it distinguishes the body's own normal molecules ("self") from foreign antigens. Self-tolerance is acquired during the maturation process in late fetal life and in the

early postnatal period, when the lymphocytes expressing receptors that bind the body's own molecules, primarily proteins, are destroyed. After birth, the body is left with millions of lymphocytes that can identify and attack foreign antigens without mounting an attack on the body's own antigens.

**Immune Prophylaxis.** Injection of antigens or antibodies into a body to achieve protection against infectious diseases is referred to as immune prophylaxis. By *vaccination*, disease-causing microorganisms, or toxins from a microorganism, are injected into the body. Prior to injection, the microorganism, or its toxin, has been treated in such a way that it loses its pathogenicity, but retains its antigenic properties. After injection, the body launches an immune response directed against the epitopes in the vaccine. A clonal expansion of the lymphocytes within the clone that recognizes the antigen epitopes in the vaccine then starts. Memory cells are also formed, and they enable the immune system to recognize the epitopes and mount a faster and stronger immune response should the vaccinated individual later be infected by the same microorganism. Thus, vaccination leads to *active immunization*.

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**49** Compare elimination of antigens by B lymphocytes and by cytotoxic T cells.

**50** How must an antigen be presented in order to be detected by a cytotoxic T cell?

Cytotoxic T cells must be in direct contact with the antigen in order to destroy it

Cytotoxic T cells secrete proteins that insert pores into the cell membrane of target cells

Lymphocytes that react against the body's own molecules are destroyed