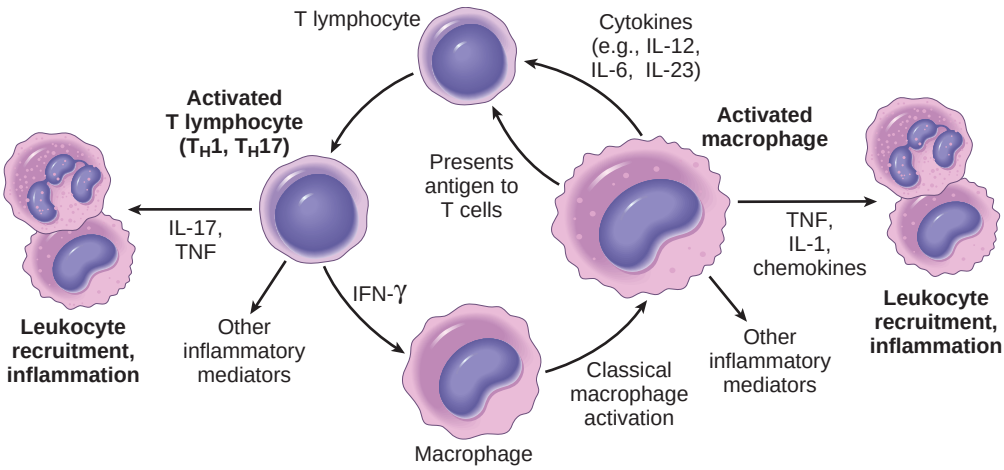

Figure 3-21 Macrophage-lymphocyte interactions in chronic inflammation. Activated T cells produce cytokines that recruit macrophages (TNF, IL-17, chemokines) and others that activate macrophages (IFN-γ). Activated macrophages in turn stimulate T cells by presenting antigens and via cytokines such as IL-12.

is why eosinophils are of benefit in controlling parasitic infections, yet they also contribute to tissue damage in immune reactions such as allergies (Chapter 6).

- **Mast cells** are widely distributed in connective tissues and participate in both acute and chronic inflammatory reactions. Mast cells express on their surface the receptor (FcεRI) that binds the Fc portion of IgE antibody. In immediate hypersensitivity reactions, IgE antibodies bound to the cells' Fc receptors specifically recognize antigen, and the cells degranulate and release mediators, such as histamine and prostaglandins (Chapter 6). This type of response occurs during allergic reactions to foods, insect venom, or drugs, sometimes with catastrophic results (e.g., anaphylactic shock). Mast cells are also present in chronic inflammatory reactions, and because they secrete a plethora of cytokines, they may promote inflammatory reactions in different situations.
- Although **neutrophils** are characteristic of acute inflammation, many forms of chronic inflammation, lasting for months, continue to show large numbers of neutrophils, induced either by persistent microbes or by mediators produced by activated macrophages and T lymphocytes. In chronic bacterial infection of bone (osteomyelitis), a neutrophilic exudate can persist for many months. Neutrophils are also important in the chronic damage induced in lungs by smoking and other irritant stimuli (Chapter 15). This pattern of inflammation has been called *acute on chronic*.

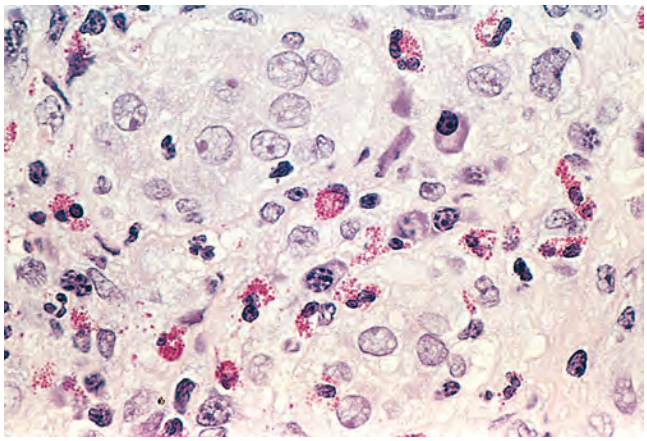

Figure 3-22 A focus of inflammation containing numerous eosinophils.

Granulomatous Inflammation

Granulomatous inflammation is a form of chronic inflammation characterized by collections of activated macrophages, often with T lymphocytes, and sometimes associated with central necrosis. Granuloma formation is a cellular attempt to contain an offending agent that is difficult to eradicate. In this attempt there is often strong activation of T lymphocytes leading to macrophage activation, which can cause injury to normal tissues. The activated macrophages may develop abundant cytoplasm and begin to resemble epithelial cells, and are called *epithelioid cells*. Some activated macrophages may fuse, forming multinucleate *giant cells*.

There are two types of granulomas, which differ in their pathogenesis.

- **Foreign body granulomas** are incited by relatively inert foreign bodies, in the absence of T cell–mediated immune responses. Typically, foreign body granulomas form around materials such as talc (associated with intravenous drug abuse) (Chapter 9), sutures, or other fibers that are large enough to preclude phagocytosis by a macrophage and do not incite any specific inflammatory or immune response. Epithelioid cells and giant cells are apposed to the surface of the foreign body. The foreign material can usually be identified in the center of the granuloma, particularly if viewed with polarized light, in which it appears refractile.
- **Immune granulomas** are caused by a variety of agents that are capable of inducing a persistent T cell–mediated immune response. This type of immune response produces granulomas usually when the inciting agent is difficult to eradicate, such as a persistent microbe or a self antigen. In such responses, macrophages activate