Dissemination of mycobacteria from a primary lung infection to other organs can occur when alveolar macrophages become infected with bacteria following phagocytosis. Migration of activated macrophages to secondary lymphoid tissue for antigen presentation to CD4+ helper T cells can facilitate spread of mycobacteria to other tissues such as liver, lymph nodes, spleen, gut, bone marrow and the urogenital tract. In immunocompetent hosts, mycobacteria disseminated to other tissues is usually controlled, however, the testis is an immune privileged site where pro-inflammatory immune responses are suppressed and it is likely that intracellular mycobacteria may have a survival advantage in this tissue.
Mycobacterial infection of macrophages in the interstitial space of the testis results in granuloma formation that sequesters infected macrophages at the centre, surrounded by immune cells, predominantly CD4+ helper T cells. Fusion of infected macrophages to form foamy macrophage giant cells is thought to result from the release of mycolic acid products from infected cells. A sheath of collagen fibres produced by fibroblasts surrounds the cells. In the immune privileged testis, Th1 immune responses are suppressed which may promote mycobacterial survival since CD8+ T cells are also required to kill intracellular bacteria. Recruitment of macrophages and T cells result in testicular swelling and disruption of sperm production.