

Color plates

Plate 1. In B6-+/+ mice, clearance of the virus from the lung was associated with resolution of the inflammatory process. In AICD-deficient B6-*lpr/lpr* mice, clearance of the virus from the lung was not associated with clearance of inflammation. The authors suggest that an AICD defect that occurs in middle-aged humans and mice, combined with an environmental exposure, such as a viral infection, triggers a chronic inflammatory or autoimmune disease that does not resolve. (See also Fig. 5B, in article by Hsu and Mountz.) (*Adapted from* Fleck M, Kern ER, Zhou T, Podlech J, Wintersberger W, Edwards 3rd CK, Mountz JD. Apoptosis mediated by Fas but not tumor necrosis factor receptor 1 prevents chronic disease in mice infected with murine cytomegalovirus. *J Clin Invest* 1998;102:1431–43; with permission.)

Plate 2. Early atherosclerotic lesion in a paraffin-section of a specimen collected by von Rokitansky in 1840. Immunohistological demonstration of T cells by a monoclonal antibody against CD3 visualized by an alkaline phosphatase labeled rabbit–antimouse immunoglobulin conjugate (original magnification $\times 200$). (See also Fig. 1, in article by Knoflach, Mayrl, Mayerl, Sedivy, and Wick.)

Plate 3. Unfixed frozen section of a venous bypass replacing the common carotid artery of a mouse with severe restenosis 6 weeks after the operation. Demonstration of activated T cells in the thickened intima by a hamster–antimouse-interleukin-2 receptor antibody visualized with a biotinylated rabbit–antihamster antibody and a streptavidin alkaline phosphatase conjugate (original magnification $\times 600$). (See also Fig. 2, in article by Knoflach, Mayrl, Mayerl, Sedivy, and Wick.)

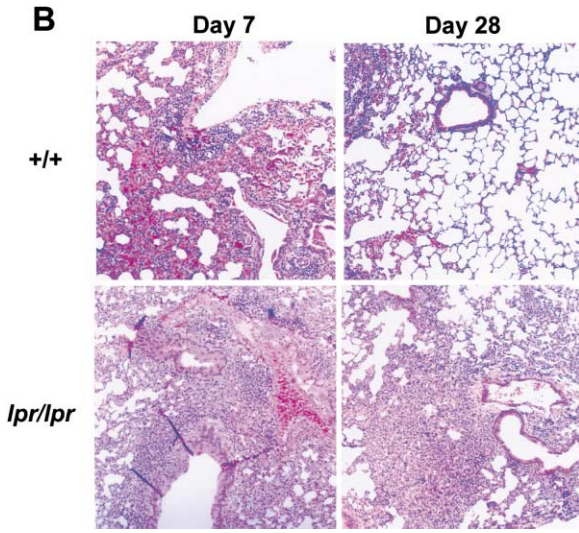


Plate 1

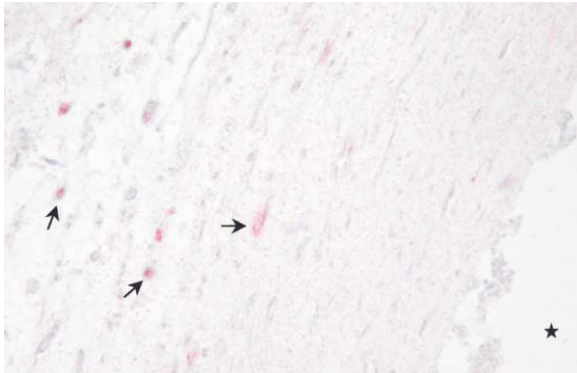


Plate 2

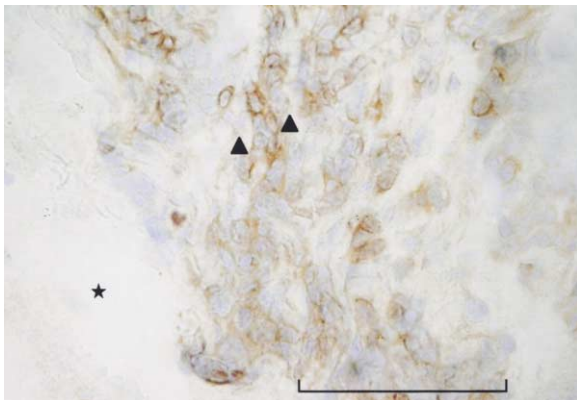


Plate 3