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Cover picture: Low power micrographs of individual *Mycobacterium tuberculosis*-induced lung lesions in wild-type mice (upper right) and mice deficient in a capacity to generate CD8 T cell-mediated immunity (class I^{-/-}; upper left), CD4 T cell-mediated immunity (class II^{-/-}; lower right), or either type of immunity (TCR- α/β ^{-/-}; lower left). The mice were infected by aerosol with 10² *M. tuberculosis* 50 d earlier. The sections are stained for inducible nitric oxide synthase (NOS2), an enzyme necessary for successful expression of immunity and an indicator of IFN- γ -mediated macrophage activation. The brown NOS2 reaction product is located in cells in macrophage-dominated regions of the lesions, but not in lymphoid regions (dark blue). Similarities between the lesions of wild-type and class I^{-/-} mice are in keeping with the ability of both types of mice to eventually control infection and to survive beyond 230 d. Class II^{-/-} and TCR- α/β ^{-/-} mice failed to control infection and died much earlier. See related article in this issue by Mogues et al., pp. 271–280.

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