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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-$\alpha/\beta^{-/-}$; lower left). The mice were infected by aerosol with 10^2 *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-$\alpha/\beta^{-/-}$ mice failed to control infection and died much earlier. See related article in this issue by Mogues et al., pp. 271–280.

Brief Definitive Reports

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*The online version of this article contains supplemental material.