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below to download.Immunologic memory has been
demonstrated in reovirus- or adenovirus-infected mice
and humans. However, the cellular processes by which
memory is generated and maintained have not been
completely elucidated. We generated a mouse deficient
in a major histocompatibility complex class I antigen
(H-2Db), which otherwise are ubiquitous expressed
molecules critical to function of natural killer cells and

dendritic cells. The phenotype of the mice is similar to that of inbred recombination activation gene (RAG) knockout mice, indicating that natural killer cells and dendritic cells are the principal cell types expressing H-2Db. Since these cells are critical to immunity, we have examined the consequences of the H-2Db deficiency on the ability of mice to respond to viral infections. Infections with reovirus or adenovirus indicated that immunocompetence was reduced, although the basis for this was more difficult to define. However, after immunization with influenza virus, IgG responses in the absence of H-2Db were similar to normal immunized animals, suggesting that there are qualitative differences between primary and secondary responses that remain relevant in the absence of H-2Db. Interestingly, analysis of T cell responses indicated that there was a differential requirement for H-2Db for memory cells and for effector cells. These studies will have implications for understanding the process of