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Schountz T1, Quackenbush S1, Rovnak J1, Haddock E2, Black WC 4th1, Feldmann H2, Prescott J2. **Differential Lymphocyte and Antibody Responses in Deer Mice Infected with Sin Nombre Hantavirus or Andes Hantavirus**

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Hantavirus cardiopulmonary syndrome (HCPS) is a rodent-borne disease with a high case-fatality rate that is caused by several New World hantaviruses. Each pathogenic hantavirus is naturally hosted by a principal rodent species without conspicuous disease and infection is persistent, perhaps for life. Deer mice (Peromyscus maniculatus) are the natural reservoirs of Sin Nombre virus (SNV), the etiologic agent of most HCPS cases in North America. Deer mice remain infected despite a helper T cell response that leads to high-titer neutralizing antibodies. Deer mice are also susceptible to Andes hantavirus (ANDV), which causes most HCPS cases in South America; however, deer mice clear ANDV. We infected deer mice with SNV or ANDV to identify differences in host responses that might account for this differential outcome. SNV RNA levels were higher in the lungs but not different in the heart, spleen, or kidneys. Most ANDV-infected deer mice had seroconverted 14 days after inoculation, but none of the SNV-infected deer mice had. Examination of lymph node cell antigen recall responses identified elevated immune gene expression in deer mice infected with ANDV and suggested maturation toward a Th2 or T follicular helper phenotype in some ANDV-infected deer mice, including activation of the interleukin 4 (IL-4) pathway in T cells and B cells. These data suggest that the rate of maturation of the immune response is substantially higher and of greater magnitude during ANDV infection, and these differences may account for clearance of ANDV and persistence of SNV.