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BALB/c mice that show a genetically predetermined susceptibility to severe experimental rhinosinusitis also demonstrate a broad CD8+ T lymphocyte repertoire and altered nasal mucosal lymphocyte homing patterns. The majority of patients with acute rhinosinusitis (ARS) have a Th1 phenotype based on CD4(+) T lymphocyte profiles and enhanced levels of interferon-gamma (IFN-gamma). These same Th1 profiles have also been described in animal models of ARS. However, many patients with ARS also have a concurrent infection with a virus that induces a Th2-type reaction. The most common virus associated with ARS is respiratory syncytial virus (RSV), but human beings commonly have concurrent rhinovirus infections. To study the response of a susceptible mouse to experimental rhinosinusitis, BALB/c mice were inoculated with RSV or PBS intranasally. Nasal mucosal responses were examined in RSV-infected and noninfected mice, and corresponding draining lymph node (DLN) responses were examined ex vivo. Bacterial, fungal, and viral infections were excluded by use of serologic analysis and bacterial culture. Mice were administered FTY720, a potent molecule known to block lymphocyte egress from peripheral tissues. Survival was assessed after high-dose RSV infection (1×10^7 pfu). DLN responses were quantified with CytoSpot assays. Susceptible BALB/c mice showed heightened levels of RSV-specific lymphocyte proliferation in the nasal mucosa and in draining DLN as compared with partially resistant C3H mice. RSV infection induced a Th1-like response in susceptible animals with enhanced interferon-gamma production. However, RSV-infected susceptible animals also had a robust population of CD8(+) T lymphocytes in the nasal mucosa

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