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DA virus mutant H101 can alter t-cell mediated CNS pathogenesis through virus induced immunosuppression

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Viruses have immunomodulatory properties for the purpose of evading clearance by the host. We are investigating how a few changes in the Theiler's murine encephalomyelitis virus (TMEV) genome could provide a means to suppress T cell mediated central nervous system (CNS) disease. The mutant TMEV virus, H101, in its natural host (the mouse), causes immunomodulation when administered via a peripheral route (intraperitoneal – i.p.). In contrast, C57BL/6 mice infected with the wild-type DA strain of TMEV via the i.p. route develop an asymptomatic infection and clear the virus. C57BL/6 mice infected with the H101 mutant virus via the i.p. route become immunosuppressed through the depletion of T cells. Intracerebral infection of C57BL/6 mice with lymphocytic choreomeningitis virus (LCMV) leads to death around day 6 post infection due to an aggressive anti-viral CD8+ T cell response. Infection of mice with H101 prior to infection with LCMV results in a majority of mice surviving past day 7 post-LCMV-infection. Similarly, infection, with H101, of mice with relapsing-remitting experimental autoimmune encephalomyelitis results in elimination of the CD4+ T cell mediated disease exacerbations. This study provides experimental evidence that viruses can be used to treat T cell mediated CNS disease.

Biography

Robert S. Fujinami completed his PhD from Northwestern University and postdoctoral studies from the Scripps Research Institute. He is a Professor in the Department of Pathology at the University of Utah. He was the first recipient of the Harry Weaver Award from the National Multiple Sclerosis Society and he is a Jacob Javits Neuroscience recipient from NINDS, NIH. He has published more than 200 articles and has been serving as member on various editorial boards and NIH review panels.

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