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Decreased adult neurogenesis due to innate immune signaling underlies virus-induced memory dysfunction

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Persistent cognitive sequelae occur following neuro-invasive infection with neurotropic flaviviruses, including Japanese encephalitis virus, Saint Louis encephalitis virus, and West Nile virus (WNV). We used an established murine model of recovery from WNV in which animals display spatial learning defects and loss of presynaptic termini within the hippocampal CA3. Transcriptional profiling of the hippocampi of mice with poor learning revealed decreased expression of genes involved in adult neurogenesis and increased expression of innate immune molecules known to inhibit this process, including interleukin (IL)-1. WNV-infected adult mice exhibited decreased numbers of proliferating neuroblasts, which are not directly targeted by virus, and increased generation of astroblasts, within neurogenic zones, with limited recovery of neurogenesis in the sub-granular zone at 30 days. Accordingly, IL-1R1-deficient, WNV-infected mice exhibited normal neurogenesis, rapid recovery of presynaptic termini, and resistance to WNV-mediated impairment in spatial learning and memory compared with wild type mice. Our results reveal that alterations to neuronal progenitor cell homeostasis during adult neurogenesis may underlie long-term cognitive consequences of WNV infection and provide a therapeutic intervention to prevent these deficits.

Biography

Robyn S Klein, is currently working as Professor, Departments of Medicine, Anatomy & Neurobiology, Pathology & Immunology. She has completed her BA: Barnard College, Columbia University, New York, NY (1985), MS, Neuroscience: Albert Einstein College of Medicine, Bronx, NY (1990), PhD, Neuroscience: Albert Einstein College of Medicine, Bronx, NY (1993). Her research interest focuses on the pathogenesis of neuroinflammatory diseases of the central nervous system (CNS). She has over 300 publications in reputed journals.

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