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Anti-CD52 therapy in multiple sclerosis

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Alemtuzumab is a humanized monoclonal antibody that selectively targets CD52 to deplete circulating T and B lymphocytes, thought to be critical mediators of MS inflammatory processes. Alemtuzumab has minimal impact on other immune cells. A distinctive pattern of T and B cell repopulation begins within weeks, potentially leading to a rebalancing of the immune system. Although alemtuzumab's exact mechanism of action is unknown, these pharmacodynamic changes may help explain its effect in MS Preclinical studies have focused on using a murine CD52 specific antibody to expand our understanding of anti-CD52 therapy in the C57BL/6 model of experimental autoimmune encephalomyelitis. These studies have demonstrated a profound impact of anti-muCD52 therapy on the course of disease. Specifically, therapeutic intervention inhibited disease development in animals prior to observable symptoms and reversed existing paralytic symptoms in more severely disabled animals. Longitudinal flow cytometric evaluation demonstrated a decrease in the number of lymphocytes in the CNS as well as circulating autoreactive T cells. Histological evaluation also showed a reduction in the level of lymphocytic infiltrate in the CNS of anti-muCD52 treated animals. Collectively, these results demonstrate the therapeutic efficacy of anti-muCD52 treatment in a murine model of EAE.

Biography

Turner received his Ph.D. from the University of Rochester in 2003, where he studied immune tolerance of CD8 T cell responses to prostate specific antigen. He continued his training as a postdoctoral fellow with Leo Lefrancois at the University of Connecticut Health Center where his studies focused on immune tolerance of memory T cells. Turner joined Genzyme Corporation in 2007 and is currently a Senior Scientist in Neuro-immunology. His research is currently focused on investigating the mechanism of action of alemtuzumab and novel agents in experimental models of multiple sclerosis.

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