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Opinion Article

Role of T-Lymphocytes in Models of Amyotrophic Lateral Sclerosis

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DESCRIPTION

T lymphocytes are immune cells that develop from stem cells in the bone marrow. They help to protect the body from infection and may aid in the fight against cancer. These are also known as T cell or thymocyte. Amyotrophic Lateral Sclerosis (ALS) is a multifactorial neurodegenerative condition characterized by progressive degradation of upper and lower motor neurons resulting in muscular weakness, paralysis and death within 2–5 years after diagnosis. ALS is also known as Lou Gehrig's Disease.

In ALS, the inflammatory process involves T cell infiltration and activation of antigen-presenting cells, which co-localize with motor neuron loss in the brain and spinal cord. T cells' function in the pathogenic process is unknown. T cells can harm motor neurons directly through cell-cell interaction or cytokine production, or they can contribute indirectly through activation of microglia and macrophages. T cell infiltration might also be an epiphenomenon associated to the clearing of dead motor neurons.

$CD4^+CD25^+FOXP3^+$ regulatory T lymphocytes (Tregs) are an immunosuppressive subset of T lymphocytes that maintain tolerance to self-antigens with their malfunction playing a critical role in the development of autoimmune diseases. Treg infusions delay disease progression and prolong longevity in mice with Amyotrophic Lateral Sclerosis (ALS), and Tregs inhibit the multiplication of responder T cells and the activation of microglia. The expression of the Treg master transcription factor FOXP3 is decreased in fast advancing ALS patients, 7 resulting in impaired Treg suppressive functions; FOXP3 expression and Treg suppressive functions correlate with the degree and velocity of illness development. T-lymphocytes have a role in the etiology of motor neuron damage and death as well. Endogenous T-lymphocytes, especially Tregs are elevated in the slow-progressing mSOD1 model of ALS, boosting IL-4 expression and protective M2 microglia. Tregs are reduced in the early stages of illness predominantly *via* downregulation of FoxP3 expression. The overall number of Tregs and their FoxP3 expression are enhanced in ALS animals during the stable disease phase compared to wild-type mice; during the fast advancing phase, the number of mSOD1 Tregs and FoxP3 expression falls while the proliferation of mSOD1 Teffs rises.

If T-lymphocytes influence the microglial-motor neuron dialogue in mSOD1 mice, the mice were crossed with RAG2 knockout mice and $CD4^+$ knockout mice, which lack functional T-lymphocytes or $CD4^+$ T-lymphocytes. The surprise finding was that motor neuron illness manifested itself at a considerably younger age, implying that a lack of $CD4^+$ T-lymphocytes reflects a lack of a neuroprotective T-lymphocyte population. This finding contradicts the findings of MPTP tests in Parkinson's disease models, in which the lack of $CD4^+$ T-lymphocytes dramatically slows disease progression, implying that $CD4^+$ T-lymphocytes are cytotoxic. Additionally, the comparatively sluggish period of illness development is removed in mSOD1/CD4/ transgenic mice. Anti-inflammatory and neurotrophic factors rise, whereas proinflammatory and cytotoxic factors decrease.

Cytotoxicity is inhibited after BMT and the restoration of $CD4^+$ T-lymphocytes; the slowly progressing phase of illness is resumed, and life is prolonged. The M2-activated microglial phenotype is promoted by $CD4^+$ neuroprotective T-lymphocytes, which increase the production of anti-inflammatory and neurotrophic substances.

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