



MECHANISTIC REFINEMENT OF THE COMMON MARMOSET MODEL FOR MULTIPLE SCLEROSIS

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Multiple sclerosis (MS) is a chronic neurological disease that affects the brain and spinal cord. It is thought that MS is an autoimmune disease, where a person's immune system reacts to its own body, namely the myelin sheath that surrounds the nerve cells. Disease symptoms include vision problems, imbalance and paralysis of the limbs. The exact cause of MS is still unknown, and therapies without adverse effects are not available. Substantial evidence suggests that the disease arises as a result of a certain combination of environmental, genetic and infectious circumstances. We hypothesize that herpesviruses create a repertoire of autoreactive T-cells, which play a crucial role in the demyelination process. To unravel the contribution of these autoreactive T-cells to the pathogenesis of MS, the common marmoset is used as an animal model for MS. The common marmoset is a relevant model with a close genetic and immunological proximity to humans, and it resembles the disease in its clinical and pathological presentation. This thesis describes how the common marmoset model for MS is refined ethically, mechanistically, and conceptually to better understand the underlying pathogenic processes, as well as to develop and improve new targets for MS therapy.

