Investigation of Inflammatory Molecules during the Disease Progression of Multiple Sclerosis in Mouse, Mus musculus, Tissue

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Multiple sclerosis (MS) affects millions of young adults worldwide. The disease is caused by the immune system mistakenly attacking the central nervous system (CNS), which consists of the brain and spinal cord, and results in a range of neurological symptoms. Currently, there are only a few disease-modifying drugs on the market, and there is a need for more effective treatments. The purpose of this project was to investigate a mouse model of MS called experimental autoimmune encephalomyelitis (EAE) to better understand the disease course of human MS and contribute to the basis for future studies on new treatments. It was hypothesized that levels of inflammatory molecules interferon-gamma (IFN-gamma), tumor necrosis factor-alpha (TNF-alpha), and interleukin-12 (IL-12) would vary (increase/decrease) in the brains of mice, Mus musculus, as the disease progressed to a chronic phase. First, RNA from normal (sham-induced) and EAE-induced mouse brain tissue was isolated and converted to cDNA. This was amplified with primers specific to each inflammatory molecule through quantitative Polymerase Chain Reaction (qPCR). Finally, gel electrophoresis was performed to verify the size of amplified samples. Data indicated that levels of both IL-12 and IFN-gamma were highest at time point 4, mid-stage disease, while the level of TNF-alpha was highest at time point 6, study endpoint. It was concluded that levels of inflammatory molecules were highest in the final stages of the disease, and the original hypothesis was accepted.