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Therapy of experimental allergic encephalomyelitis under circumstances relevant to human multiple sclerosis - Part II

Experimental allergic encephalomyelitis (EAE) is an inflammatory disease of the central nervous system (CNS) that resembles human multiple sclerosis (MS). EAE and MS develop when proteins of the myelin sheath that covers axons are released and encounter cells of the immune system such as T lymphocytes. Activation of these lymphocytes will trigger inflammation that destroys the myelin leading to clinical signs that manifest mostly in the form of motion impairment and muscle paralysis. Inactivation of myelin specific lymphocytes is currently viewed as a means to halt immune attack against the brain and reverse the course of disease. Previous research in our lab has shown that peptide delivery on immunoglobulin (Ig) is effective against EAE. This method of treatment presents a clinical challenge for use in humans, however, because it involves intraperitoneal injection of the chimeric Ig (Ig-MOG). We have determined that the oral route, which is more practical, yields comparable results against the disease. This model is also useful for investigation of mechanisms of oral tolerance.