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Differential immunogenicity of myelin oligodendrocyte glycoprotein epitopes generates localized phenotypes of experimental autoimmune optic neuritis

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Multiple sclerosis affecting the optic nerve and related neuroinflammatory disorders such as autoimmune optic neuritis are characterized morphologically by significant immune cell infiltration and structural changes in nerve cells and myelinating oligodendrocytes in parallel to impaired visual performance. When functional deficits become permanent this is typically accompanied by damage to nerve cells such as the loss of axons in the optic nerve. Oligodendrocytes express myelin oligodendrocyte glycoprotein (MOG), a transmembrane protein of the immunoglobulin superfamily involved in intercellular communication at the myelin sheaths formed by oligodendrocyte around optic nerve axons. As MOG is expressed in several isoforms, the functional importance of the extracellular domain located at the very N-terminus shared by all splice variants and containing the site for N-linked glycosylation of the protein has become evident. Soluble peptides and recombinant proteins derived from the MOG N-terminus were used to induce autoimmune optic neuritis. Depending on sequence, length, dosing and timing of administration delivery of the MOG peptides and proteins differential effects were observed in disease models using functional markers of neuroinflammation such as demyelination and immune cell infiltration. Specifically, expression levels and distribution of proteins typically found in highly localized expression patterns within axons were significantly altered depending on the MOG-derived peptide constructs. This was paralleled by functional impairment at the cellular and organ level. Altogether, peptides and recombinant proteins derived from the MOG N-terminus can be used selectively in research models to recapitulate differential patterns and aspects of structural and functional damage of autoimmune optic neuritis.

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Metoclopramide-induced hyperprolactinemia changed on murine uterine

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Initially our group found out that the metoclopramide-induced hyperprolactinemia may negatively affect the endometrial morphology. And in later studies we found out that the hyperprolactinemia caused a decrease in pinopode numbers and embryo implantation in female mice thus interfering with the fertility and in ovarian function. In order to show the effectiveness of treatment with 200 µg metoclopramide for 50 consecutive days, we measured the serum prolactin levels and also analyzed the pituitary of animals. We proved that the hyperprolactinemia caused by metoclopramide in mice is due to an increase in the number and activity of lactotrophs. The deepening of the research led us to several questions. What are the biochemical changes that were occurring in the endometrial stroma (cells and extracellular matrix) of these animals? Finally, our results showed that the elevation of prolactin may lead to changes in the amounts of glycosaminoglycans which are important for embryo implantation in an animal model of hyperprolactinemia accompanied by a regular estrous cycle. Recently, we have researched the gene expression of small leucine-rich proteoglycans (SLRPs) on the murine uterus non pregnant and pregnant with hyperprolactinemia induced metoclopramide. The interactions between the production and degradation of these substances with steroids and hyperprolactinemia is complex and difficult to explain because the signaling pathways involving those hormones may influence the cell-cell and cell-extracellular matrix interactions in the endometrial stroma as well as they may interfere with the appropriate preparation of the endometrium to receive the embryo.

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