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FUNCIONES INMUNOMODULADORAS DE LA GALECTINA-3 EN CONDICIONES DE NEURODEGENERACIÓN

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Recent findings suggest an important role of neuroinflammation in neurodegenerative diseases, such as Alzheimer's disease and Parkinson's disease, which are debilitating, expensive and largely untreatable conditions strongly linked with age. Microglia are the innate immune cells of the brain that, under homeostatic conditions, constantly survey their environment and prune neuronal synapses. Following an insult or injury, these cells become activated and can perform a wide array of functions by production of pro-inflammatory and neurotoxic factors, as well as protective, anti-inflammatory and phagocytic functions. In chronic neuroinflammatory states seen in neurodegenerative diseases a unique disease-associated phenotype is observed. Selective mutations in microglia/myeloid-specific genes, including the glycoprotein triggering receptor on myeloid cells 2 (TREM2), have been associated with Alzheimer's disease. Recent evidences have suggested TREM2 to be instrumental in neuroinflammation and linked to microglia neurodegenerative/disease-associated phenotype. One protein which induces this phenotype is galectin-3, which binds complex glycans in glycoproteins. Galectin-3 is expressed and released specifically by highly activated microglia in the injured brain and can propagate inflammation by binding glycoproteins such as TLR4, and also TREM2. In this presentation I will address whether galectin-3 also contributes to detrimental neuroinflammation in Alzheimer's pathology, and, hence, could be a therapeutic target.