Lipocalin-2, an astrocytic protein in the context of multiple sclerosis

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Abstract

Lipocalin-2 (LCN2), an acute phase protein, has gained increased attention in the central nervous system, in the context of disorders like multiple sclerosis (MS). Specifically, we previously found increased LCN2 levels in the cerebrospinal fluid of MS patients. However, its role for MS establishment and progression is highly contradictory. To contribute for the better understanding of LCN2 role in MS, in this work we induced experimental autoimmune encephalomyelitis (EAE) in LCN2-null and wild-type (Wt) littermate controls. Contrary to previous works, we did not found alterations in the clinical score of the animals when comparing both genotypes. However, in the cerebellum, LCN2-null EAE mice presented decreased expression of pro-inflammatory cytokines, namely *interferon gamma*, and decreased percentage of demyelination and inflammatory infiltrates. Moreover, astrocytes from LCN2-null EAE mice seemed to become more reactive later in the disease, compared to Wt EAE animals. At the periphery, both genotypes presented similar alterations in the thymocyte and splenocyte populations. In patients, higher LCN2 levels seemed to associate with a faster progression of the disease. Overall, our results support a harmful role for LCN2 in the disease context.