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Lipocalin as a novel marker for early onset alzheimer?

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Pro-inflammatory pathways are involved in the pathogenesis of Alzheimer's disease. We have identified a TNF-α-induced proinflammatory agent, lipocalin 2 (Lcn2) via gene array in murine primary cortical neurons. Lcn2 protein production and secretion were activated solely upon TNFR1 stimulation when primary murine neurons, astrocytes, and microglia were treated with TNFR1 and TNFR2 agonistic antibodies. In addition, Lcn2 was found to be decreased in CSF of human patients with mild cognitive impairment (MCI) and AD and increased in brain regions associated with AD pathology in human postmortem brain tissue. In the second study we investigated the functional roles of TNFR1 and TNFR2 in learning and memory, motor performance and anxiety-like behavior via several behavioral and cognitive assessments in young and aged mice, deficient of either TNFR1 or TNFR2. Results from this study show that deletion of TNFR2 impairs novel object recognition, spatial memory recognition, contextual fear conditioning, motor performance and can increase anxiety-like behavior in young adult mice.

In a subsequent study in a clinical cohort we examined the determinants of plasma Lipocalin in late-life depressive disorder (N=350). Depressed patients had significantly higher Lipocalin plasma levels compared to non-depressed comparison group. Subjects with a recurrent depression had higher plasma NGAL levels compared to those with a first episode. Lipocalin could be a marker for late onset Depression / MCI and this research provides an example of successful translational research in which similar findings were obtained from animal studies and clinical samples.

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