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In vivo Animal Models

Neuroinflammation

Lipopolysaccharide (LPS) Induced Mouse Model

Neuroinflammation is a common feature of different neurodegenerative diseases, such as Alzheimer's disease, Parkinson's disease, Frontotemporal dementia, Amyotrophic lateral sclerosis and several others. Recent research suggests that targeting neuroinflammation might present a valid method to treat neurodegenerative diseases. In order to model neuroinflammation independent from other disease-relevant pathologies, wildtype mice can be peripherally injected with lipopolysaccharide (LPS). Here we present the effect of daily 0.5 mg/kg LPS injections on four consecutive days. Other treatment protocols can be performed according to your needs.

Figure 1:
Immunofluorescent labeling of the medial and lateral hippocampus of LPS-treated mice. Animals were intraperitoneally injected with vehicle **(A, B)** or 0.5 mg/kg LPS **(C, D)** on 4 consecutive days. Tissue was sampled 24 h after last injection and labeled with CD68 antibody (red) for macrophages, IBA1 antibody (green) for microglia and DAPI (blue) for nuclear staining.

medial hippocampus, Vehicle 4 x

Figure 1: A

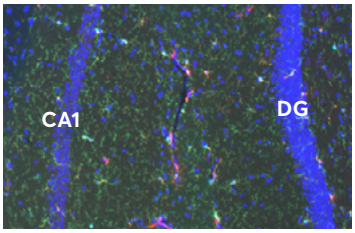
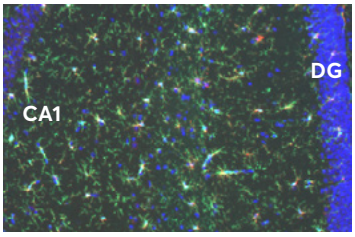


Figure 1: C, CD68, IBA1, DAPI



lateral hippocampus, Vehicle 4 x

Figure 1: B

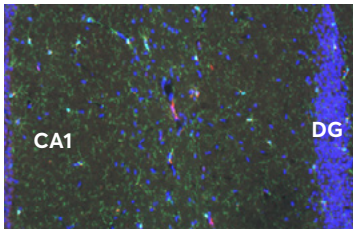


Figure 1: D, CD68, IBA1, DAPI

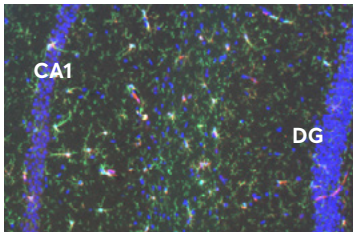
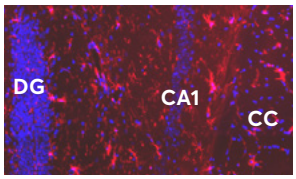


Figure 2:
Representative images of IBA1 immunofluorescent labeling. Arrows indicate specific staining of microglia in the dentate gyrus (DG) and CA1 region of the hippocampal formation. Treatment with LPS increased the number and size of labeled cell bodies (arrows in **A, B**). Background labeling was generally higher in mice sacrificed 7 days after treatment start (TS 7, **B**). **C:** absence of immunofluorescent labeling when the primary antibody was omitted (negative control). cc = Corpus callosum.

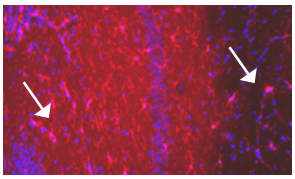
LPS 4x 0.5mg/kg, TS 4

Figure 2: A



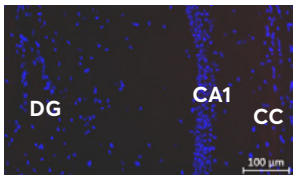
LPS 4x 0.5mg/kg, TS 7

Figure 2: B



Negative control

Figure 2: C



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