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Intravenous injection of lipopolysaccharide increases expression of inflammatory cytokines in murine brain: Possible the role of norepinephrine

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Evidence suggests that noradrenaline has a tonic anti-inflammatory activity in the central nervous system (CNS) owing to its ability to suppress microglial and astrocytic activation, as well as inhibiting production of inflammatory mediators. Therefore it is proposed that noradrenaline may play an endogenous neuroprotective role in CNS disorders where inflammatory events contribute to pathology.

Here we survey anti-inflammatory effects of norepinephrine on tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) gene expression following a systemic challenge with bacterial lipopolysaccharide (LPS) in the mouse.

In the present study twenty one male NMRI mice (25 ± 5 g) used and divided into three groups (n = 7). They were injected through the mouse tail vein with saline, 100 nM norepinephrine 30 min before the intravenous injection of LPS (5 mg/kg). Thus, inflammation induced by the intraperitoneal injection of LPS. The control animals received sterile saline in the first and second injections. Changes in the expression TNF-α and IL-6 genes studied in the mice brain by a semi quantitative RT-PCR method.

The results of this study showed that norepinephrine makes alteration to the expression of IL-6 genes in brain 2 h before the injection of LPS. Norepinephrine markedly reduced the expression of IL-6 genes. The data suggest that norepinephrine protective effect in brain inflammation induced by LPS through inhibition IL-6. Overall, this study has yielded significant insights into the ability augmentation of noradrenaline strategies to limit neuroinflammation.

Defining the signaling mechanisms of lymphocyte activation gene-3 (LAG-3) in T cells

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Background: Lymphocyte activation gene-3 (LAG-3) is an inhibitory receptor expressed on activated T cells, B cell, and Natural Killer cells. LAG-3 is engaged by MHC class II molecules expressed by antigen presenting cells (such as dendritic cells) which lead to T cell inhibition. It is one of the main targets in immune therapy. Several studies are currently exploring the enhancement T cell proliferation via the blockage of LAG3 or combining with other receptor blockage. However, the molecular mechanism of this is yet unclear.

Aim: The broad aim of our research is to define the signalling pathways that are modulated by LAG-3. The specific aim for the initial part of our research is to establish a DC-T cell antigen specific experimental system that will enable analysis of key T cell signalling pathways.

Results: We demonstrate that a mouse transgenic T cell receptor model (F5 TCR transgenic mouse) when coupled with dendritic cells that express the correct antigen is an appropriate model to examine LAG-3 signalling. Our results reveal induction of tyrosine phosphorylation in several T cell proteins and identify a potential candidate target of LAG-3 modulation.

Conclusion: The F5 TCR transgenic and dendritic cell mouse experimental system is useful for signalling studies on LAG-3.

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