

Neuroimmune control of interleukin-6 secretion in the murine spleen. Differential β -adrenergic effects of electrically released endogenous norepinephrine under various endotoxin conditions

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Abstract:

In a previous study we demonstrated a superfusion technique which allows for investigation of nerve-immune cell interaction in murine spleen. We demonstrated that under septic-like conditions in the presence of bacteria and lipopolysaccharide (LPS), electrically induced inhibition of interleukin 6 (IL-6) secretion was attenuated by the β -adrenergic antagonist propranolol. This effect was now investigated more closely under various endotoxin conditions in order to dissect effects of bacteria and endotoxin: (A) bacteria-rich conditions (without penicillin/streptomycin [P/S] and without LPS), (B) LPS-enriched conditions (with P/S and with LPS), and (C) bacteria-free conditions (with P/S and without LPS). Under bacteria-rich conditions, norepinephrine ($E_{max} = 10^{-6}$ M, $p = 0.012$) and isoproterenol ($E_{max} = 10^{-6}$ M, $p = 0.048$) concentration-dependently inhibited IL-6 secretion from murine spleen slices in contrast to bacteria-free conditions. In a bacteria-free environment the β -adrenergic antagonist propranolol did not attenuate the electrically induced inhibition of splenic IL-6 secretion. The insertion of bacterial filters in front of the superfusion chambers to avoid direct contact between bacteria and cells increased the electrically-induced inhibition of IL-6 secretion ($p = 0.0036$). Added LPS did not change the electrically-induced release of norepinephrine from presynaptic nerve terminals in murine spleen. The study demonstrates two different β -adrenergic effects on IL-6 secretion of murine spleen slices under bacteria-rich or bacteria-free conditions.

Keywords: Superfusion technique; Murine spleen slice; Norepinephrine; Interleukin-6 secretion; Bacteria

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