IMMUNOLOGY AT THE CONFLUENCE OF MULTIDISCIPLINARY APPROACHES

ABSTRACT BOOK

Institute for Biological Research "Siniša Stanković" National Institute of Republic of Serbia
University of Belgrade

Immunological Society of Serbia

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Poster presentation

PROPRANOLOL REDUCED SEVERITY OF EAE BY INCREASING THE EXPRESSION OF Nrf2 IN MICROGLIA

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Sympathetic dysfunction was proposed to participate in development of multiple sclerosis and its animal model, experimental autoimmune encephalomyelitis (EAE). This may be linked with findings indicating that noradrenaline, the key sympathetic end-point mediator, through β-adrenoceptor exerts immunomodulatory action. Considering importance of the target tissue for the clinical outcome of EAE, the study investigated the effects of propranolol, a non-selective β-adrenoceptor blocker, on the disease severity in Dark Agouti rats. Administration of propranolol over the effector phase of EAE substantially moderated neurological symptoms of the disease. This correlated with the increased proportion of spinal cord microglia expressing CX3CR1, the crucial neuroinflammation-limiting molecule, and upregulated expression of Nrf2, the key CX3CR1 downstream target gene. Additionally, in spinal cord of propranolol-administered rats the expression of heme-oxigenase 1, Nrf2 target gene, was upregulated. Consequently, microglia from propranolol-administered rats, exhibited increased proportion of IL-10–expressing cells, but decreased those of IL-1β– and IL-23–expressing ones. Propranolol also downregulated the IL-6 and MCP-1/CCL2 expression in spinal cord. Furthermore, propranolol affecting CXCR1/Nrf2 signaling pathway enhanced microglial phagocytic/endocytic capacity and surface expression of anti-inflammatory CD163/CD83 markers. Results from *in vitro* pharmacological study examining influence of noradrenaline/propranolol on functional properties of microglia showed that microglia synthesize noradrenaline, which, in turn, through β-adrenoceptor, downregulated their Nrf2 expression, in a CX3CR1-independent manner. In accordance with microglial shift towards a more anti-inflammatory profile, in spinal cord of propranolol-administered rats was found: i) decreased infiltration with blood-borne myeloid and CD4+ T cells, and ii) reduced CD4+ T-cell reactivation/proliferation and differentiation into highly pathogenic IL-17+ cells co-producing IFN-γ and GM-CSF. The study suggests a neuroinflammation-promoting role for central noradrenaline in EAE, via β-adrenoceptor–mediated modulation of microglial Nrf2 expression. Thus, it points out to a putative target for future translational pharmacological research to optimize multiple sclerosis therapy. Funding: MPNTR RS (grant number 175050).