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Neuro-endocrine immune interactions in response to bacterial infection in mice

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A reciprocal regulation exists between the central nervous and immune systems where the CNS signals the immune system *via* hormonal and neuronal pathways and the immune system signals the CNS through cytokines. The hypothalamic pituitary adrenal (HPA) axis regulates the immune system *via* glucocorticoid hormones (GC). Neuroendocrine regulation of immune function is essential for survival during stress or infection and to modulate inflammatory disease. Glucocorticoids have multiple effects on immune cells but insensitivity of immune cells to this hormone during chronic stress and persistent inflammation has been the cause of several neuroimmunological disorders. In a mouse model of restraint stress and infection with Gram negative (*E. coli*) or Gram positive (*S. aureus*) or injection of bacterial endotoxin (LPS) several inflammatory parameters and behavioral changes were studied. Increased resistance of mice to infections that correlated with their behavioral changes in an open field test (OFT) and on an elevated plus maze (EPM) has been observed. Reduced acetyl cholinesterase (AChE) activity in the hypothalamus indirectly suggested that more acetylcholine molecules must be playing their anti-inflammatory role to modulate the activity of the HPA axis. Exogenous administration of pro-inflammatory cytokine (IL-6) or an anti-inflammatory cytokine (IL-10) along with ciprofloxacin (an antibiotic having secondary anti-inflammatory role apart from its primary anti-bacterial effect) to stressed and infected mice, down regulated the inflammatory mediators and also effectively helped in bacterial killing. Thus this work provides new insight into the therapeutic strategy that can be adopted when considering treatment against chronic stress and infection induced neuroimmunological disorders.

Biography

Biswadev Bishayi has completed his BSc in Physiology in 1990 from Midnapore College and MSc in Human Physiology from Vidyasagar University West Bengal, India in 1992. After qualifying NET-CSIR examination he joined Indian Institute of Chemical Biology as JRF and completed his PhD from Jadavpore University, West Bengal, India in 1999. He has joined as Lecturer in the Department of Physiology, University of Calcutta in 1997 and promoted to Professor in 2012. After being awarded the Biotechnology Overseas Associateship from the DBT he did his Postdoctoral research in Boston University Medical School, USA. The main focus of his research is host-pathogen interaction, role of cytokines in inflammatory diseases as well as neuro-endocrine immune interactions in relation to *Staphylococcus aureus* infection. He has regularly published papers in national and international reputed journals.

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