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Niacin attenuates inflammatory cytokine upregulation in PD mediated through GPR109A

Neuroinflammation is central in Parkinson's disease (PD) pathology. Microglia derived inflammatory cytokines are known to be involved in progressive degeneration of substantia nigra (SN) neurons. We have demonstrated upregulation of anti-inflammatory receptor GPR109A in blood (PD patients) as well as in SN (post-mortem PD patient samples). Up-regulation of GPR109A may be a part of body's defense mechanism. Niacin (vitamin B3) acts on GPR109A to reduce the inflammation in PD. To understand the cellular mechanisms involved in the anti-inflammatory action of niacin here we utilize lipopolysaccharide (LPS) induced inflammatory cascade in RAW 267.4 cells. These cells are macrophages that resemble microglial lineage. LPS is known to trigger inflammatory cytokines production such as IL1-b, IL-6 and TNF-a via NF-kB pathway. NF-kB is the transcription factor and the translocation of its p65 unit to nucleus is an essential step in the inflammatory cascade. Here we demonstrate inhibition of pNF-kB translocation by niacin in RAW 267.4 cells via GPR109A. However, in the absence of GPR109A, niacin failed to block the translocation of pNF-kB and the subsequent production of inflammatory cytokines in RAW 267.4 cells. This anti-inflammatory action of niacin via GPR109A might be beneficial in PD to alleviate motor and non-motor symptoms.

Biography

Chandramohan Wakade has been engaged in the field of CNS injury and its amelioration for number of years. The focus of his research includes trauma to the nervous system and neural repair. He has worked on various animal stroke models including MCAo, SAH and TBI models in rats and mice. His recent work focuses on studying role of inflammation in CNS injury and neurodegenerative diseases in patients.

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