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Anti-inflammatory effects of benfotiamine in LPS stimulated microglia: role of NF-κB signaling

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Microglial cells are immune cells of the central nervous system (CNS) that play a major role in its surveillance. Changes in CNS homeostasis, invading pathogens or neuron impairment, lead to activation of microglial cells that quickly proliferate, acquire amoeboid morphology and produce toxic mediators such as nitric oxide (NO) and pro-inflammatory cytokines. These changes are regulated by transcription factors, most importantly NF-κB. Although microglial activation is important for maintaining tissue homeostasis, excessive activation leads to chronic inflammation that can damage healthy neurons. Substances that suppress microglial activation are potential therapeutics for neurodegenerative diseases. Benfotiamine (S-benzoylthiamine-O-monophosphate) is a synthetic derivative of vitamin B1 that has anti-inflammatory properties. In this study we investigated anti-inflammatory properties of benfotiamine on activated microglia in vitro.

BV-2 microglia were pre-treated with benfotiamine, stimulated with LPS and their viability and morphology were evaluated. LPS induced prominent alterations in cell morphology, enlargement of cell bodies and spreading of multiple processes. Pre-treatment with benfotiamine before LPS stimulation suppressed these morphological changes. Additionally, benfotiamine diminished LPS induced NO production, without altering cell viability. Furthermore, benfotiamine decreased LPS induced production of pro-inflammatory cytokines TNF- α and IL-6, while increasing production of anti-inflammatory cytokine IL-10. Analysis of NF-κB activation revealed that benfotiamine's effects were mediated by this transcription factor.

In conclusion, benfotiamine exerts anti-inflammatory properties in LPS activated microglia *in vitro* and should be further investigated as a potential therapeutic for neurodegenerative diseases.