Involvement of c-Abl kinase in activation of NLRP3 inflammasome via cathepsin B: Implications for Parkinson's Disease

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Abstract:

Parkinson’s Disease (PD) is a progressive neurodegenerative disease characterized by the pronounced loss of dopaminergic neurons. Studies from past decade demonstrate accumulation of activated microglia as pathological hallmark of PD. In this context, we investigated the signaling mechanisms by which minimally toxic concentration of LPS, an inflammogen, and rotenone (ROT), a mitochondrial complex I inhibitor, elicited microglial activation response. Our findings reveled a novel signaling axis connecting c-Abl/cathepsin B/NLRP3 is responsible for aberrant microglial activation. This study suggests targeting c-Abl kinase for modulating microglia-dependent inflammation as therapeutic strategy for PD.