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Effects of bromelain on striatal neuro inflammation following a 6-OHDA lesion in rat model of Parkinsonism

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Neuro inflammation alters the microenvironment in certain areas of the brain resulting in the development of Parkinson's disease (PD). Mechanisms involving cytokine and non-cytokine induced pathways are activated in the inflammatory response following injury. The proteolytic activity of bromelain appears to eliminate receptors on immune cells that respond to pro-inflammatory signals. This raises the question of whether manipulation of the inflammatory response pathways could lead to a beneficial intervention for PD. In this study, we investigated the effect of bromelain exposure on pro-inflammatory cytokine concentration and microglial activation in a parkinsonian rat model. Male Sprague-Dawley rats were lesioned stereotaxically with the neurotoxin, 6-OHDA. The anti-inflammatory drug bromelain was used to treat a subset of the rats before or 24 hrs post 6-OHDA lesion. The systemic blood concentration of leukocytes and platelets was measured along with plasma and striatal concentrations of pro-inflammatory cytokines IL-1β, IL-6 and TNF-α. In addition, cd11b/cd86 was quantified as a measure of glial cell activation. Intramedial forebrain bundle injection of 6-OHDA resulted in a marked increase in the systemic concentration of leukocytes and platelets which was inhibited by pre-treatment with bromelain. Pre-surgical bromelain treatment also resulted in the suppression of both systemic pro-inflammatory cytokines and gliosis. Early treatment with bromelain may slow the progression of PD by attenuating the inflammatory response associated with the disease. The present results suggest that bromelain may be considered for further clinical study and perhaps use as prophylactic treatment for patients with PD.

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