

AbstractID: 12829 Title: Exploring the neuroprotective effects of bee venom in a mouse Parkinson model with immunohistochemistry, Magnetization Transfer Ratio imaging and spectroscopy

Purpose: Neuroprotective therapeutics stop or slowdown the degeneration process in animal models of Parkinson's disease (PD). In the present study, we investigated the anti-inflammatory effect of bee venom (BV) on lipopolysaccharide (LPS)-stimulated microglia.

Method and Materials: To investigate whether MR techniques have added value in neuroprotection research in the MPTP mouse model, both MRI and MRS were applied to investigate the neuroprotective effects of BV. BV is suggested to stimulate anti-inflammatory processing indirectly via DA-dependent mechanisms or γ -aminobutyric acid (GABA) and glutamate release. Magnetization transfer ratio (MTR) and ¹H-MRS studies were performed on control mice, MPTP-intoxicated mice and BV treatment mice. The successfully induced lesions were verified by tyrosine hydroxylase immunolabelling on the substantia nigra (SN) and striatum (ST) performed on perchloric extracts of mice brains, after the control mice and the MPTP-intoxicated mice were killed. All the MTR and ¹H-MRS experiments were performed on a 9.4 T MRI/MRS system using a standard head coil. Outer volume suppression combined with the ultra-short echo-time stimulated echo acquisition mode was used for the localized ¹H-MRS. The quantitative analysis of metabolites was determined using jMRUI from the ¹H spectra obtained in vivo on the striatum.

Results:

The peak height of the MTR histograms in the PD model group was significantly lower than that in the BV treatment group ($P < 0.05$). The present study revealed that the peak height of the MTR histogram was significantly decreased in the ST and SN, and a significant increase of the Glx/Cr ratio was found in the striatum of the PD group, as compared with that of the BV treatment group.

Conclusion: These results demonstrate that BV possess a potent suppressive effect on proinflammatory responses of microglia and suggest that these compounds may offer substantial therapeutic potential for treatment of neurodegenerative diseases that are accompanied by microglial activation.