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Neuroprotective and Neurotherapeutic Effects of Bee Venom on Neurodegenerative Diseases

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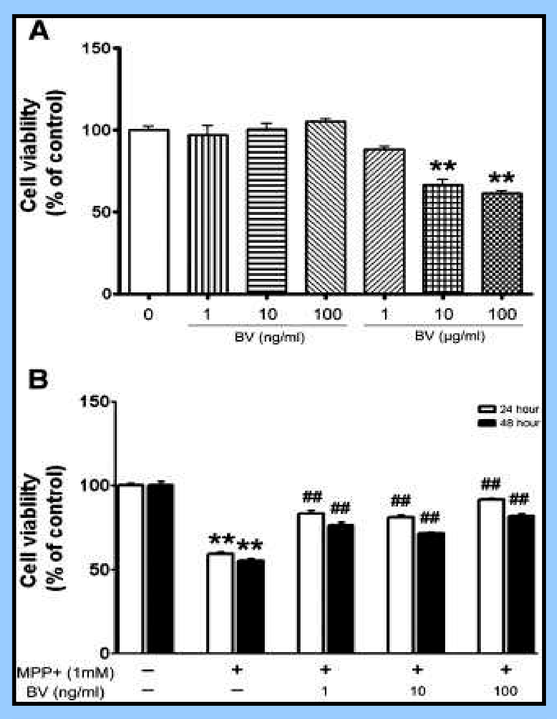
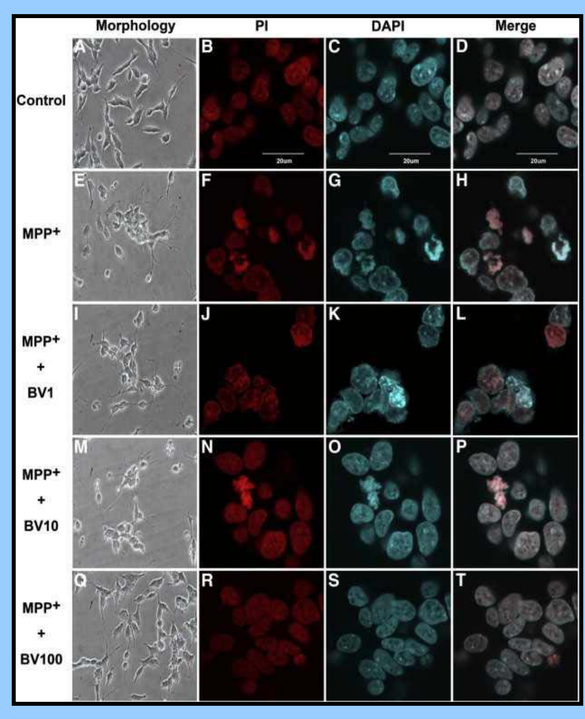
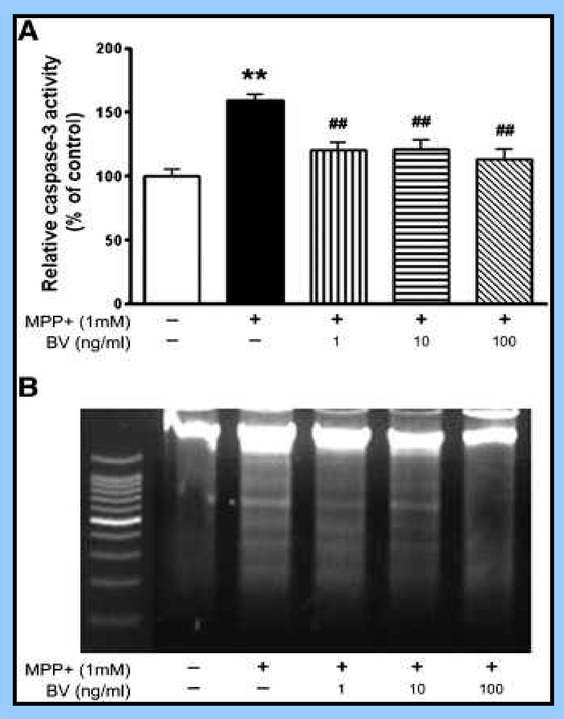
Introduction

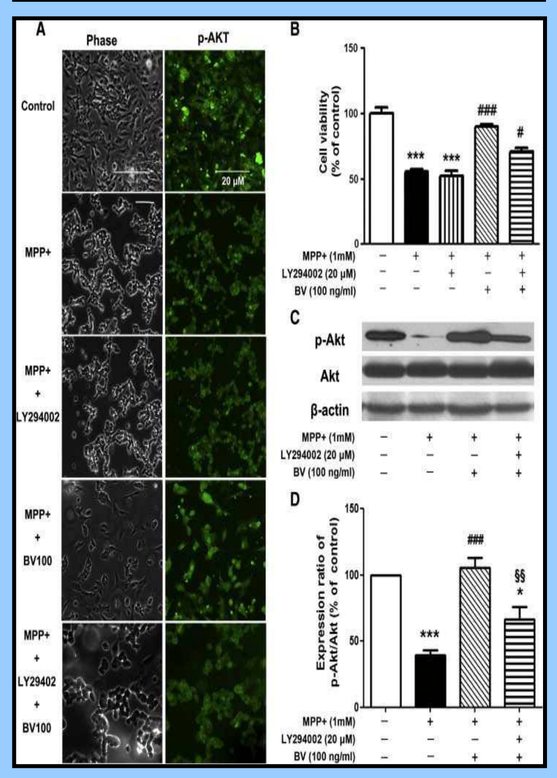
Acute and chronic neurodegenerative diseases are illnesses associated with high morbidity and mortality, and few or no effective options are available for their treatment. A characteristic of many neurodegenerative diseases — which include stroke, brain trauma, spinal cord injury, amyotrophic lateral sclerosis, Huntington's disease, Alzheimer's disease, and Parkinson's disease — is neuronal cell death. Given that central nervous system tissue has very limited, if any, regenerative capacity, it is of utmost importance to limit the damage caused by neuronal death. Bee venom, which is also known as apitoxin, consists of several biologically active peptides, including melittin, adolapin, mast cell degranulating peptide and phospholipase A2. Furthermore, bee venom contains a variety of bioamines, such as apamin, histamine, procamine, serotonin, and norepinephrine, which facilitate nerve transmission and healing in a variety of nerve disorders. This gives bee venom the ability to travel along the neural pathways from the spine to various trigger points and injured areas to help repair nerve damage and restore mobility. This review article overviews; (1) causes and mechanisms of neurodegenerative diseases which pertains to neuronal cell death, (2) evidence linking composition comprising bee venom to its substantial potential for preventing and treating of neurodegenerative diseases associated with neuronal cell death, and (3) how improving our knowledge of the various mechanisms mediating neuroprotective and neurotherapeutic activities of bee venom against neuronal cell death may led to novel therapeutic strategies for the treatment of neurodegenerative diseases.

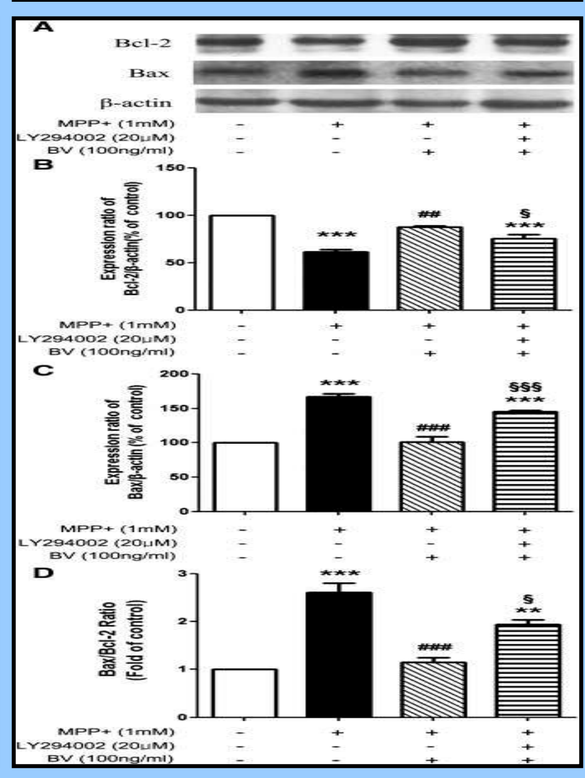
Anti-Apoptotic Effect of Bee Venom against Neuronal Cell Death

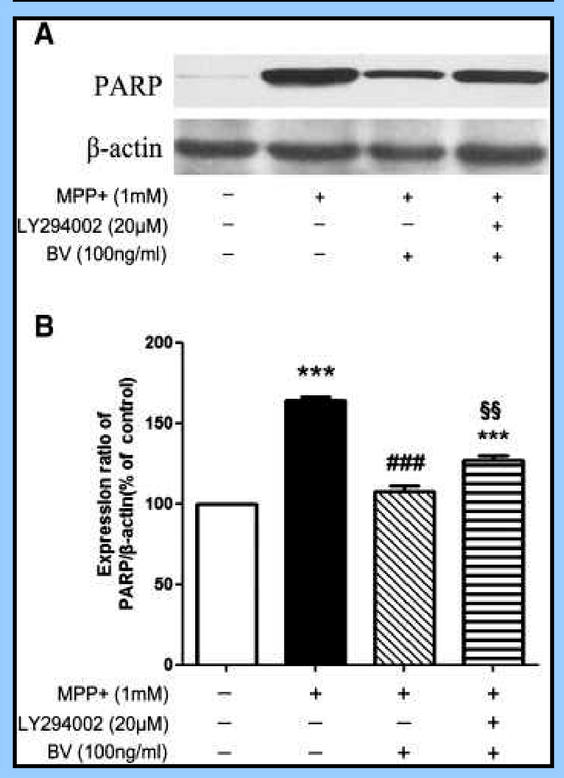












Conclusion

During the past several years, our understanding of the mechanisms mediating neuronal cell death has improved considerably. The fact that activation of these pathways is a feature of a broad range of neurodegenerative diseases makes them important and attractive therapeutic targets. Bee venom inhibits neuronal cell death and activation of proapoptotic signaling in neurons. These findings emphasize the clinical importance of bee venom for treating neurodegenerative diseases. Further investigation of bee venom activity *in vivo* is necessary to elaborate the mechanisms involved and to permit the full exploitation of the therapeutic potential of bee venom. In addition, bee venom contains a variety of peptides (e.g., melittin and apamin), enzymes (e.g., phospholipase A2, histamine, and epinephrine), non-peptide components including lipids and carbohydrates, and free amino acids. Therefore, further research is required to determine bioactive single element of bee venom. Future challenges remaining will be to elucidate signaling responses activated by bee venom in neurons.

References

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