DNA REPAIR AND NEUROPROTECTION IN CEREBRAL ISCHEMIA

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Research Summary

Dr. Chen's laboratory is interested in molecular mechanisms of neuronal cell death associated with cerebral ischemia or Parkinson's disease. The work focuses on determining the role of programmed cell death and mitochondrial dysfunction in cellular and animal models. The main theme of this research is that elucidation of the signaling mechanisms underlying the pathologic neurodegenerative processes in the brain may help identify new targets for future therapeutic intervention of the disease. The lab is currently investigating the specific signaling molecules and pathways that trigger mitochondrial apoptosis and downstream caspase-dependent or caspase-independent cell death-execution cascades in neurons.

Another main area of interest of Dr. Chen's laboratory is to elucidate the role of oxidative DNA damage and repair in ischemic brain injury, focusing on the mechanisms associated with the base-exCISION repair (BER) pathway in neuronal injury. The evolution of oxidative DNA damage, such as single-strand breaks and AP sites, is an early determinant of neuronal cell death or survival in cerebral ischemia. As a highly inducible process, BER is the key mechanism responsible for the repair of various oxidative DNA damage in the brain. This line of research is based on the belief that manipulation of cellular BER activity may markedly impact the vulnerability of neurons to ischemic challenges.

Recent Publications


Figure on the left:
The molecular pathway of DNA repair in neurons

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