Publication

D-JNKi a peptide inhibitor of c-Jun N-terminal kinase promotes functional recovery after transient focal cerebral ischemia in rats.

E. Esneault, V. Castagné, P. Moser, C. Bonny and M. Bernaudin.
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Abstract

The c-Jun-N-terminal kinase (JNK) pathway has been shown to play an important role in excitotoxic neuronal death and several studies have demonstrated a neuroprotective effect of D-JNKi, a peptide inhibitor of JNK, in various models of cerebral ischemia. We have now investigated the effect of D-JNKi in a model of transient focal cerebral ischemia (90 min) induced by middle cerebral artery occlusion (MCAo) in adult male rats. D-JNKi (0.1 mg/kg), significantly decreased the volume of infarct, 3 days after cerebral ischemia. Sensorimotor and cognitive deficits were then evaluated over a period of 6 or 10 days after ischemia and infarct volumes were measured after behavioral testing. In behavioral studies, D-JNKi improved the general state of the animals as demonstrated by the attenuation of body weight loss and improvement in neurological score, as compared with animals receiving the vehicle. Moreover, D-JNKi decreased sensorimotor deficits in the adhesive removal test and improved cognitive function in the object recognition test. In contrast, D-JNKi did not significantly affect the infarct volume at day 6 and at day 10. This study shows that D-JNKi can improve functional recovery after transient focal cerebral ischemia in the rat and therefore supports the use of this molecule as a potential therapy for stroke.