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Abstract

Amyotrophic Lateral sclerosis (ALS) is a fatal neurodegenerative disease characterized by a selective loss of motoneuron in the cortex and spinal cord. Mitochondrial dysfunctions are increasingly recognized as key factors in the pathophysiology of ALS (Le Masson and al., 2014). To restore mitochondrial functions is therefore a potential new therapeutic strategy. In this framework, it has recently been shown that the X protein of the Borna Virus has powerful neuroprotective properties in a toxic model of Parkinsonism. We propose to explore the protective potential of this same protein in a murine model of ALS (SOD1 mice). This protein will be delivered using a CAV2 viral vector in vivo, with muscle injection first and then intra-thecally. The progression of the disease will be monitored using behavioral studies, electromyography measurement and immunochemistry. An ineffective mutated version of the X protein will be used as control.

Qualification required

Master2 in Neurosciences. A knowledge in viral transfection or in murine electrophysiology would be a plus