

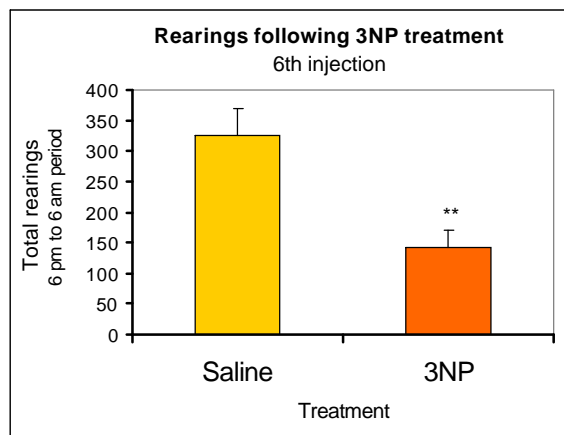
INTRODUCTION

Systemic injections of 3-nitropropionic acid (3-NP) in rodents cause striatal neuropathy similar to that observed in clinical Huntington disease. This treatment produces a variety of neurobehavioral abnormalities and motor deficit such as bradykinesia, muscles weaknesses and rigidity. These observations lead to consider this protocol as a relevant model of Huntington disease (Brouillet et al., 1999). 3-NP is an irreversible inhibitor of succinate dehydrogenase and induces a severe failure of mitochondrial oxidative phosphorylation process. Such energetic lesion process made this model relevant to study in vivo anti-oxidant drugs (Kasparova et al., 2006).

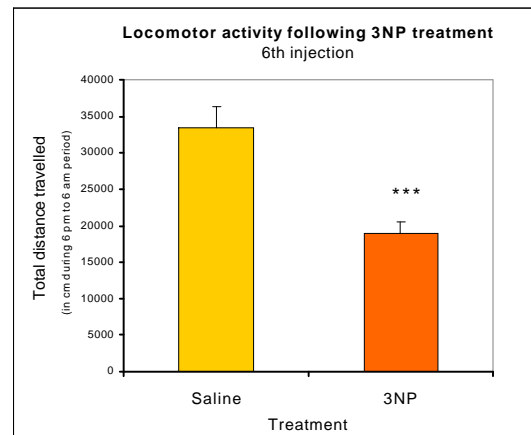
PROTOCOL

Rats received repeated daily injections of 3-NP. Following each administration, animals are placed in the actimeter and activity is recorded overnight.

Results



** $p < 0.01$ vs. Saline



*** $p < 0.001$ vs. Saline

Figures show that 3-NP treatment induces a large decrease in the nocturnal locomotor activity after 6 injections.

References

Brouillet et al., Replicating Huntington' disease phenotype in experimental animals. Progress in Neurobiology, 1999, 59, 427-468.
 Kasparova et al., Effect of coenzyme Q10 and vitamin E on brain energy metabolism in the animal model of Huntington's disease Neurochem Int., 2006, 48, 93-99.