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Uncontrollable stimulation inhibits plasticity within the rat spinal cord: Role of the group I metabotropic glutamate receptors

A.R.Ferguson^{1,2*}; M.A.Hook¹; J.W.Grau¹

1. Psychol Dept, Texas A & M Univ, College Station, TX, USA; 2. Dept of Neurosci., Ohio State Univ., Columbus, OH, USA

Prior work has shown that the spinal cord is capable of learning a simple instrumental (response–outcome) task. Subjects are spinally transected at the second thoracic vertebra (T2) and tested 24 h later. During testing, subjects are given shock when the hind limb is in an extended position. Rats given controllable shock quickly learn to maintain the hind limb in a flexed position that minimizes net shock exposure. Rats that receive an equivalent amount of shock independent of leg position (uncontrollable shock) fail to learn. Uncontrollably shocked rats also fail to learn when tested with controllable shock, a learning deficit that lasts up to 48 hrs. Evidence suggests that uncontrollable stimulation interferes with later learning by saturating NMDA–mediated plasticity. The present study examines whether this effect depends on the group I metabotropic glutamate receptors (subtypes mGluR1 and mGluR5). Spinally transected rats were treated with the noncompetitive antagonists for the mGluR1 (CPCCOEt) or mGluR5 (MPEP) receptors (0, 1, 10, or 100 nmol, i.t.) prior to uncontrollable shock and tested 24 hrs later with controllable shock. Uncontrollable shock inhibited learning in the vehicle treated rats ($p < .05$). Both CPCCOEt and MPEP attenuated the deficit in a dose dependent fashion ($p < .05$). In Experiment 2 rats received the general group I mGluR agonist DHPG alone or in combination with a small amount of uncontrollable shock that was insufficient to induce a robust deficit. Pretreatment with the agonist inhibited instrumental learning and enhanced the destructive effect of uncontrollable stimulation ($p < .05$). The results suggest that the induction of the learning deficit depends on group 1 mGluRs.

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