Z503 **The Role of Protein Synthesis in Activity-Dependent Inactivation of Ca Channel**

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Protein synthesis are generally involved in a long-lasting synaptic changes in physiology and morphology. Previous studies have shown that an increase in impulse activity of a previously inactive crayfish motoneuron F3, produces a long-lasting reduction in Ca current. To examine the effect of protein synthesis on this activity-dependent reduction in Ca current, protein synthesis was blocked by applying cycloheximide (CHX) to the bath 4-5 hr before the onset of stimulation. The axon was stimulated at 5 Hz for 1 hr and the peak Ca currents were recorded from the cell body 6-7 hr after stimulation. Electrical stimulation in normal saline produced a reduction in the peak Ca current. The peak Ca current density was significantly less in stimulated cells (-46.3 ± 4.5 nA/nF) compared to contralateral controls (-61.6 ± 6.2 nA/nF, n=7; $p<0.05$). However, in the presence of protein synthesis inhibitor (0.6 mM CHX), the mean density of the peak Ca current in the stimulated cells (-55.2 ± 3.7 nA/nF) was not significantly different from the contralateral control cells (-59.4 ± 4.8 nA/nF, n=7, $p>0.05$). In addition, the reduction in Ca current ($7.6 \pm 2.8\%$) observed when protein synthesis was blocked is significantly different from that observed in control cells ($33.1 \pm 6.3\%$, n=7, $p>0.05$). These results demonstrate that protein synthesis is required for full expression of this activity-dependent reduction in Ca currents.

Z504 **The Effect of Cerebral Ischemia-Reperfusion Injury on Serum Nitrites and Isoprostane-F2α Levels in the Mongolian Gerbil**

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The neurodegeneration by ischemia-reperfusion(IR) injury is caused by the integrated results of excitotoxicity, free fatty acid release, free radical injury during & after ischemia, and nitric oxide. To evaluate the role of free radical injury, the serum 8-isoprostane F2α was assayed and the effects of the systemically administered nitric oxide synthase(NOS)inhibitor, NG-nitro-L-arginine methyl ester (L-NAME), on the changes in extracellular glutamate. After injection of saline or L-NAME, forebrain IR was performed by bilateral carotid artery occlusion with controlled hypotension for 7 minutes and reperfusion. 8-isoprostane F2α and nitrite levels in saline treated groups during the cerebral IR injury were increased up to 3 hrs and 1 hr of reperfusion respectively and returned to control levels as the time passed. Those in L-NAME treated groups showed the same trend as in saline treated groups. IR brain damage was common in the frontoparietal cortex, the hippocampus, and to a varying extent in the medial segment of the striatum, depending on the duration of the occlusion. The results showed that IR brain injury may be related to increase the production of reactive oxygen species, and therefore the damage of macromolecules in the tissue leading to increase of cell damages and induction of apoptosis which can cause brain injury.