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Changes in ion channel expression levels following axonal injury in the crustacean stomatogastric ganglion

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Ion channels play an essential part in carrying the electrical impulses that control motor and sensory functions. The regulation of these channels is fundamental in establishing and maintaining appropriate neuronal output. Therefore, mechanisms may exist that alter the expression of these ion channels as a result of changes in neuronal activity. To explore this possibility, we hypothesized that a decrease in sodium channel activity would result in a lower level of expression of sodium channel mRNA. We tested this hypothesis by determining whether injury to axons causes a decrease in the abundance of sodium channel mRNA. We performed this experiment in motor neurons of the crab (*Cancer borealis*) stomatogastric ganglion (STG) by measuring sodium channel mRNA in control and in preparations where axons of STG neurons were cut. Using quantitative PCR, we determined that sodium channel mRNA levels were twofold higher in STG cells taken from the control preps as compared to injured preps. The data was inconsistent with our hypothesis that mRNA expression levels would decrease after an injury that lowers sodium channel activity. These results suggest a balance between ion channel abundance and neuronal activity may be dependant not only on the nerves themselves, but the environment they are contained in due to the variance in technique used between the first and second sets of preparations. Even though little "homeostatic plasticity" was exhibited, these results may shed light on changes in cellular properties that could influence therapeutic treatments when dealing with damage to the nervous system.