CALIFORNIA STATE SCIENCE FAIR
2012 PROJECT SUMMARY

Name(s)

Vaishnavi L. Rao

Project Number

32429

Project Title

Activity-Dependent Regulation of Nitric Oxide Expression: Novel Form of Neurotransmitter Plasticity

Abstract

Objectives/Goals

As a gaseous neurotransmitter, Nitric Oxide (NO) plays a key role in several physiological functions including sleep, feeding, sensory and motor functions. Imbalances in the levels of NO lead to neurotoxicity, implicated in multiple neurological disorders such as stroke, Alzheimer#s disease and Parkinson#s. Activating NO plasticity as a means of regulating NO levels has never been explored. Therefore, my novel study focused on unraveling the plastic properties of NO in the regions of the hindbrain via alterations in electrical activity. This could aid in the development of effective clinical therapies.

Methods/Materials

Fixed tissues of the embryonic tadpole X. laevis, previously injected with mRNA encoding for decreased and increased electrical activity through overexpression of potassium (Kir) and sodium (Nav) ion channels respectively, and cascade blue dye (control) were obtained. By means of cryostat sectioning followed by immunohistochemistry and observations under the confocal microcope, I obtained layer-by-layer count of neurons in each of the three hindbrain regions. Similarly, I examined the plasticity of classical neurotransmitters serotonin and GABA in relation to NO expression. This required significant enhancements to previously established protocols.

Results

Electrical activity does allow for NO regulation at a localized level. Under Nav, statistically significant increase in NO expression was observed in the reticulospinal region, whereas under Kir, there was a statistically significant decrease across all three regions. Furthermore, coexpression of NO with Serotonin and GABA was observed under decreased electrical activity.

Conclusions/Discussion

This research is the first to establish a successful model for regulation of gaseous neurotransmitter NO at a localized level using electrical activity. The results suggest the activation of reserve pools of neurons, which gain the ability to respecify neurotransmitter phenotypes. This holds promise for the restoration of broken neuronal circuitry that occurs as a result of neurotoxic conditions. The protocol developed here can be implemented for future studies on localized plasticity of NO. As opposed to pharmacological means, electrical activity offers a more immediate, efficient, localized and rapid response to be elicited by the nervous system.

Summary Statement

Electrical activity offers a novel and promising means of Nitric Oxide neurotransmitter plasticity in the form of localized regulation and recruitment of reserve pools of neurons, with enormous applications to neurodegenerative disorders.

Help Received

Staff at Spitzer Lab (UCSD) for providing supervision during independent experimentation.