



CALIFORNIA STATE SCIENCE FAIR 2011 PROJECT SUMMARY

Name(s) Vaishnavi L. Rao	Project Number 31276
Project Title A Novel Study of Neurotransmitter Plasticity in the Embryonic Brain	
Objectives/Goals Neurotransmitter respecification is a recently discovered form of neuronal plasticity with enormous applications to disorders including stroke, Alzheimer's and Parkinson's disease. While respecification behavior of classical neurotransmitters is well understood, plasticity of nitric oxide (NO), a gaseous neurotransmitter that regulates various physiological functions, is yet to be established. Therefore, the objective of my study was to determine whether nitric oxide expression could be respecified when electrical activity was altered through over-expression of potassium (K ⁺) and sodium (Na ⁺) ion channels. I hypothesized that over-expression of K ⁺ channels would decrease NO expression while over-expression of Na ⁺ channels would display opposite effects.	
Abstract Neurotransmitter respecification is a recently discovered form of neuronal plasticity with enormous applications to disorders including stroke, Alzheimer's and Parkinson's disease. While respecification behavior of classical neurotransmitters is well understood, plasticity of nitric oxide (NO), a gaseous neurotransmitter that regulates various physiological functions, is yet to be established. Therefore, the objective of my study was to determine whether nitric oxide expression could be respecified when electrical activity was altered through over-expression of potassium (K ⁺) and sodium (Na ⁺) ion channels. I hypothesized that over-expression of K ⁺ channels would decrease NO expression while over-expression of Na ⁺ channels would display opposite effects.	
Methods/Materials Since plasticity is most clearly manifested in the embryonic brain, larvae of the vertebrate <i>Xenopus Laevis</i> (in stage 42 of development) were used for experimentation and nitric oxide synthase (NOS), the enzyme that catalyzes the synthesis of NO, was used as marker of NO expression. Expression of serotonin via tryptophan hydroxylase (TPH) was used as reference for validation of NO results. Fixed larvae tissue, injected with mRNA and encoding for Kir and Nav were first obtained. I then dissected the brains of the samples, immunostained them with fluorescently tagged anti-NOS antibody and imaged using confocal microscope and counted the number of neurons.	
Results In brains obtained from K ⁺ channel over-expressing larvae, I observed that NOS expression was downregulated compared to control. Brains with enhanced Na ⁺ channels exhibited upregulation of NOS. Interestingly, under decreased electrical activity, TPH and NOS were coexpressed, suggesting phenotype respecification in the embryonic brain.	
Conclusions/Discussion This research has established the procedure for studying the neuronal plasticity of gaseous neurotransmitter Nitric Oxide. The data from this study establishes a successful model of gaseous neurotransmitter behavior under different conditions of electrical activity, which will allow for further research related to the ability of the brain to adapt, as well as treatments for neurodegenerative disorders.	
Summary Statement Nitric oxide can be regulated in the brain via electrical activity, demonstrating a form of neurotransmitter plasticity, which has enormous applications to neurodegenerative disorders.	
Help Received Staff at Spitzer lab (UCSD) for teaching me how to use the equipment and providing supervision while I was conducting experiment.	