



**CALIFORNIA STATE SCIENCE FAIR  
2015 PROJECT SUMMARY**

<b>Name(s)</b> <b>Emily A. Kim</b>	<b>Project Number</b> <b>S0516</b>
<b>Project Title</b> <b>Electrophysiology-Based Screen to Discover Genes Involved in Synaptic Homeostasis</b>	
<div><div><b>Objectives/Goals</b> Homeostatic feedback systems are omnipresent forms of biological regulation which play crucial roles in the development of the nervous system, regulation of synaptic strength, and the establishment of the proper balance of excitation and inhibition. Dysfunction in these systems may contribute to the onset of neurological diseases such as schizophrenia, autism, epilepsy, and other complex neurological diseases. This experiment is intended to identify genes which are involved in the maintenance of the stability of neural function in regards to synaptic plasticity.</div><div><b>Methods/Materials</b> Seven different mutated Drosophila stocks were crossed against a T15 cross which knocked down the targeted genes and a C15 cross, the control. The resulting 3rd instar larvae were dissected to reveal the neuromuscular junction. Electrophysiology was performed to gather mEJP (miniature excitatory junction potentials) and EJP (excitatory junction potentials) values. If a gene appeared to have a role in homeostatic plasticity, imaging was also conducted to see if there were any morphological changes. The NMJ of the larvae was dissected and was fixed in fixative, PFA, and then stained with primary and secondary antibodies. A confocal microscope was used to take pictures of the synapses of the larvae.</div><div><b>Results</b> Results showed that one of the seven tested genes, unc104, may potentially be involved in homeostatic plasticity as the average size of the mEJP and EJP values when unc104 was knocked down were significantly smaller than in the larva expressing the gene. Imaging revealed that knocked-down unc104 resulted in postsynaptic densities defects.</div><div><b>Conclusions/Discussion</b> This experiment proved my hypothesis that if a gene is essential in synaptic homeostasis, then there will be no compensation for the synaptic challenge to restore proper physiological excitation as a retrograde signal will not be sent from the post synaptic neuron to the presynaptic neuron to release more vesicles. A future experiment could be to overexpress the genes and observe the effects on homeostatic plasticity.</div></div>	
<b>Summary Statement</b> This project uses the D. melanogaster neuromuscular junction as a model to discover genes involved in synaptic plasticity by using electrophysiology.	
<b>Help Received</b> Received help from Dr. Dion Dickman and Koto Kikuma.	