



**CALIFORNIA STATE SCIENCE FAIR  
2013 PROJECT SUMMARY**

<b>Name(s)</b> <b>Jonathan F. Fung</b>	<b>Project Number</b> <b>S1713</b>
<b>Project Title</b> <b>Monosodium Glutamate: A Ligand for mGluRs One and Five Inducing Microtubule Depolymerization in Alzheimer's Disease?</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> Alzheimer's disease is a neurological degenerative disease projected to affect 13.2 million people by 2050. The many observed causes of Alzheimer's, varying from tau aggregations to aluminum, have a common link: microtubule depolymerization. Glutamate (GLU) has been experimentally verified to activate mGluRs (metabotropic G-protein coupled Glutamate Receptors) one and five, which then phosphorylates MAP2 protein, leading to EB3 protein accumulation and microtubule depolymerization. The primary objective of my investigation is to determine if Monosodium Glutamate (MSG) is a ligand to mGluRs one and five leading to microtubule depolymerization and Alzheimer's disease.</p> <p><b>Methods/Materials</b> Due to limitations in school laboratory equipment, I adopted the flowering plant Arabidopsis Thaliana, a versatile model organism with homologous processes of the human neuronal microtubule cytoskeleton. Arabidopsis was grown in custom-poured agar plates with concentrations (10, 1, 0.1, and 0.01 mM) of the positive control GLU and experimental variable MSG. Trichomes (extensions of the plant proportional to microtubule length) were measured in microns.</p> <p><b>Results</b> MSG elicited a decrease in microtubule length with increasing concentration. In 10mM MSG, 10mM GLU, and 1mM GLU, no trichome growth was observed. 0.01 and 0.1 mM GLU had average trichome lengths of 17.87 and 16.47 <math>\mu\text{m}</math>, respectively. 0.01, 0.1, and 1mM MSG had average trichome lengths of 19.83, 18.92, 14.03 <math>\mu\text{m}</math> respectively, as compared to control lengths of 51.78 <math>\mu\text{m}</math>. Exponential regression showed the correlation between MSG concentration (C) and microtubule length (L) to be <math>L = \log_2(852747e^{-3.944C})</math>. P-values computed using Welch's T-test between MSG microtubule length and control microtubule length were all less than 0.05, thus proving the decrease in microtubule length MSG has elicited is statistically significant.</p> <p><b>Conclusions/Discussion</b> This study provided statistically significant evidence to support my hypothesis: MSG acts as a ligand for mGluRs one and five, leading to microtubule depolymerization. If MSG passes the blood-brain-barrier, it will activate mGluRs 1 and 5, leading to microtubule depolymerization and Alzheimer's. Newborns and infants are especially susceptible, as their blood brain barriers are not fully developed. Further research would involve serotonin as a potential therapeutic for Alzheimer's, as it increases MAP2 levels leading to stable microtubules.</p>	
<b>Summary Statement</b> I determined that MSG, one of the most commonly consumed food additives, is a ligand for mGluRs one and five, leading to microtubule depolymerization and Alzheimer's Disease when diffused through the blood brain barrier.	
<b>Help Received</b> I would like to thank my parents for paying for materials and providing support, and my advisor Ms. Fallon for providing extra materials from the STEM class and helping with statistics.	