



**CALIFORNIA STATE SCIENCE FAIR  
2011 PROJECT SUMMARY**

<b>Name(s)</b> <b>Alexander J. Lu</b>	<b>Project Number</b> <b>S1713</b>
<b>Project Title</b> <b>The Role of the Parabrachial Nucleus in Regulation of Cardiac Sympathoexcitatory Reflexes Evoked by Bradykinin</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> The objective of this study is to characterize the role of the parabrachial nucleus (PBN) in regulating sympathetic cardiac reflexes during myocardial ischemia.</p> <p><b>Methods/Materials</b> In fifteen sinoaortic-denervated, vagotomized, and anesthetized cats, 0.1-3 µg/ml of bradykinin (BK) was applied to the epicardium of the heart to evoke the reflex responses. Then, 50 nL of non-specific glutamate receptor-antagonist Kynurenic acid (Kyn) was microinjected into the PBN followed by three repeated BK applications. Blood pressure and renal sympathetic nerve activity (RSNA) are recorded throughout each experiment. Chicago Sky Blue was microinjected at the Kyn injection site and the brain is removed for histological analysis to confirm our results.</p> <p><b>Results</b> The BK-evoked reflex responses were attenuated by an integrated mean of 48% in mean arterial blood pressure (MAP) and 56% in RSNA 25 minutes after microinjection of Kyn into the PBN. The changes in RSNA confirm our changes in blood pressure because the renal sympathetic nerve innervates the renal artery and kidney, which strongly influences blood pressure. All microinjections were accurately placed into the PBN.</p> <p><b>Conclusions/Discussion</b> The strong correlation between attenuation in RSNA and MAP confirms the significance of the non-specific glutamate blockade in the PBN with relation to regulating cardiac sympathetic response during myocardial ischemia. I concluded that the BK-evoked sympathoexcitatory reflexes are regulated by PBN neurons through the glutamate receptor mechanism.</p>	
<b>Summary Statement</b> My project has found a new pathway regulating cardiac sympathetic reflexes, which will eventually be used to create new drugs to counter life threatening cardiac reflexes during ischemic episodes.	
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