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Draft Abstract/ Summary

## Introduction

Neurological cancer and other neurodegenerative diseases are amongst the most crippling illnesses with the greater portion of infected individuals not projected to survive past five years of initial diagnosis depending on the severity, stage, and form of the disease; the research in this project will identify the neurological molecular mechanisms of specific biological markers increasingly found to be identified in instances of these diseases, specifically c Jun kinase, which upon identification will be inhibited by utilizing SAB antibodies. Consequently, further damage such as mutation in the mitochondrial DNA and stress including cascades of apoptotic signaling pathways will be entirely halted, providing a highly effective treatment to the aforementioned neurodegenerative diseases.

## Hypothesis

I propose that upon further research with the interaction of SAB and c Jun kinase, that there will be a highly interdependent molecular reaction revealed that links the inhibition of JNK kinase to the presence of the SAB, or SH3BP5, gene.

## Methods

The major methods that I will use throughout the course of this research includes the use of the Western Blot Protocol in conjunction with the extensive cell culture of brain cancer cell lines including JNK and wild-type. This protocol will utilize specific proteins from these cell lines and blot them with antibodies of common mitochondrial outer membrane mediators. The proteins will first be ran through a selective membrane via gel electrophoresis that allows for progressive movement based on molecular weight; increased weight produces greater facility in movement on the membrane. Under specific radiation, these will fluoresce in order to provide a correlation or lack thereof between the protein and antibody.

## Significance

My project is innovative in that it will provide further insight into the very nature of the interaction of JNK and SAB, seeking to develop a SAB isotope that will effectively inhibit JNK kinase. This may provide an effective treatment to terminate neurodegenerative disease by inhibiting a major stressor in order to prevent further mutation or dysfunction in molecular mechanisms associated with numerous neurodegenerative diseases.