Research Question- Can our affibody molecule, Z_{SYM73}-ABD, be able to be used to reverse amyloid beta burden within APP/PS1 double transgenic mice while not affecting inflammation levels.

Hypothesis- I hypothesize that amyloid beta levels will successfully decrease while close to no change will occur in levels of microgliosis or astriogliosis.

Engineering Goal- The engineering goals of this project are to validate the role of this engineered affibody in Alzheimer's mouse models. In order to make sure that this specific affibody can properly function and decrease amyloid burden. This will in turn also engineer a vaccine that can be administered at any stage of Alzheimer's and reverse the amyloid beta pathology.

Expected Outcome- The expected outcomes of our procedures is to see a decrease in amyloid beta plaques within the cortex and hippocampus of aged Alzheimer mouse models. Levels of microglia and astroglia should also remain constant and not increase.

Procedure- In order to characterize the role of affibody Z_{SYM73}-ABD, brain sections will be stained immunohistochemically. This is done in order to see if amyloid beta plaques visibly decreased which would indicate that the affibody was effective. In order to stain for amyloid beta we will use a mixture of antibody 6E10 and antibody 4G8. Brain sections will also be stained for microglia and astroglia by using anti-IBA1 and anti-GFAP respectively.

Risk and Safety- Proper procedures like wearing gloves, safety goggles, and a lab coat will be used while handling certain substances like DAB-nickel, hydrogen peroxide, and ethidium brodium under the supervision of my mentor.

Data Analysis- Brain sections will be analyzed visually under the microscope first in order to see a visual decrease in amyloid beta plaques. Subsequently, a graphlab software was used to semi-quantify the levels of amyloid beta throughout the sections.

No changes were made to this research plan.