**Abstract**

Programmed cell death is essential to the continuation of life. Apoptosis is a naturally occurring process that is vital to normal life and development. It is the genetically controlled form of cell suicide that is initiated by normally inactive enzymes. This cellular pathway eliminates damaged, dangerous or unwanted cells in organisms. Dangerous cells being those that are virally infected or even cancer transformed.

Caspase is one of these inactive enzymes that when activated begins the cascade of cell death or apoptosis. Located in all cells, whether eukaryotic or prokaryotic, caspase is highly conserved and therefore shows little variation between species. When apoptosis is initiated prematurely a number of diseases and organ failures occur. Therefore, preventing the early onset of apoptosis through the control of caspase release could create many potential therapeutic treatments for diseases such as liver diseases, inflammatory diseases, cardiovascular diseases, and central nervous system diseases.

Our research focuses on caspase and its inhibition. Should a protein such as P35 inhibit caspase, it would be unable to initiate programmed cell death. (Baculovirus P35 is a potent substrate inhibitor, which affects caspases found in many organisms.) This would mean that if a virus would infect a cell, it would be able to use this cell to reproduce and form more viruses.

**Conclusion**

The caspases are a class of conserved proteins responsible for programmed cell death. This multi-step process, called apoptosis, is important to all living things to protect against viruses and diseases that would otherwise take over the cell. Our researcher particularly looked at caspase and P35 inhibitor in insect cells. P35 is an inhibitor that has the capacity to completely or partially stop cell suicide, allowing the virus to replicate and spread. In addition, we found this process is important in development of the lens of the eye and removing fetal webbing from between fingers and toes. Our model shows caspase-1 and the binding site for the inhibitor P35.