CALIFORNIA STATE SCIENCE FAIR
2003 PROJECT SUMMARY

Name(s)
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Project Number
S1329

Project Title
The Apoptotic Trends in Immune Cells

Abstract

Objectives/Goals
The purpose of my project is to investigate the life and death of Varicella Zoster Virus infected immune cells and possible mechanism(s) that maintain immune cell survival after VZV infection. I hypothesized that CD4 T cells are the carriers of the virus to our skin. Also, I believed that the virus takes over a pathway in a cell for replication or survival purposes.

Methods/Materials
I used flow cytometry and microscopy to determine which specific sub-population of immune cells are the most viable after VZV infection. I started out processing human tonsils to obtain the immune cells. I then purified and separated the cells using magnetic beads and conjugated monoclonal antibodies over a steel mesh column. The co-cultured VZV infected monolayer was stained with anti VZV immune serum and anti Annexin V. I counted various fields of vision to get the percentage of cells that were infected and had gone through apoptosis. To verify my results, I compared it with the results from flow cytometry. I then used column purified cells and incubated them with MAP Kinase inhibitors (U0126 and SB203580) or capase inhibitors in concentrations of 25ìM and 100ìM before adding them to a VZV infected monolayer. I analyzed these cells also through microscopy and flow cytometry.

Results
The majority, 73-100%, of CD8 and CD19 infected cells died within 48 hours post infection which eliminates them for carrying Varicella Zoster Virus to our skin layer. The percentages of T cells that were VZV infected was reduced with MAP kinase inhibitors from 30.9% (untreated) to 2.7% (U0126 treated) or 6.5% (SB203580 treated). However, the percentages of VZV infected T cells that expressed AnnexinV did not change. Inhibition of VZV infected T cells with caspases inhibitor did not rescue the cells from apoptosis.

Conclusions/Discussion
I concluded that all CD4+, CD8+ T cells, and CD19+ B cells can be infected with VZV. Although 50% of infected CD4 cells died within 48 hours, there are enough infected cells and time to infect our skin layer making them the primary carrier. After analyzing the results from the MAP kinase pathway,it can be seen that this pathway may be involved in viral replication but not in cell death. Also, I discovered that the virus does not use caspase pathways for survival or replication.

Summary Statement
To discover which specific subpopulation of immune cells are the most likely carriers of Varicella Zoster Virus and which cell pathway the virus uses to survive.

Help Received
Used lab equipment at Stanford University under the supervision of Dr. Chia-Chi Ku