

## Researching the Role of Endothelial cells in the HIV Infection of CD4+ T-cells

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The World Health Organization estimates that over 36.9 million people are infected with HIC worldwide and HIV contributes to roughly 1.2 million deaths annually. Domestically, over one million people are infected with HIV, and according to the Center for Disease Control, 50,000 patients are diagnosed with HIV every year. Thanks to organizations like the Red Project, public awareness of the virus is high, but much has yet to be known about how it infects the immune system and how a cure can be found.

Previous studies have shown that the cells that line your blood vessels –endothelial cells- play a role in HIV's infection process. Shen's lab has begun discovering the differences that lymphatic endothelial cells play in the infection of HIV. Additionally, last year's work unveiled that high levels of a specific cytokine IL6 correlate with high infection rates in CD4+ T cells. Our lab's goal this summer was to expand on this work and explore the roles that different cytokines may play in the infection of HIV in the CD4+ T cells.

My daily work generally focuses on data collection and analysis. My fellow lab-mates will add CD4+ cells to wells with different variables added to them. For example, one triplicate of wells may contain endothelial cells and the IL6 cytokine while another triplicate of wells will contain endothelial cells and a different cytokine. These cells are all infected with a HIV retrovirus we create in our lab. After a few days of incubation, I collect these samples and run them through a flow-cytometer. This machine is able to indicate what cells are infected. I then use software to analyze which sets of variables correspond to higher infection rates. From this data, I can then conclude what cells or cytokines contribute to the infectibility of HIV in these CD4+ T cells.

This summer our experiments have revealed that in addition to IL6, the cell surface molecule CD2 correlates with high infection rates in CD4+ T cells. We discovered this when we blocked the CD2 molecules on the CD4+ T cell's surface and observed that infection rates decreased. Theoretically, if the receptors for both CD2 and IL6 were blocked, infection would cease. However, when we tried this, there was still infection in the CD4+ T cells, suggesting that there is a third – or possibly more- cytokine at play.

I've known that the reputation of research is that things almost never go exactly as planned, and that one must adapt on the fly. But this summer I was able to experience that first hand and work with Dr. Shen to find solutions to these bumps in the road. This ability to adapt, as well as the physical experience of lab work will hopefully assist me later in my educational and professional career.