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Role of Endothelial Cells in HIV Infection

Human Immunodeficiency Virus (HIV) is a retrovirus that causes Acquired Immunodeficiency Syndrome (AIDS). In recent years, scientists have learned to use Highly Active Antiretroviral Therapy (HAART) to suppress the virus to minimal level in the majority of patients. However, eradication of HIV is still unachievable because latent viral reservoirs persist. HIV latent infection is when the virus infects T cell, the viral RNA is integrated to its host cell’s chromosome but doesn’t express viral genetic identity. Consequently, the latent infected cells show no sign of infection and HAART is not curative. Thus, Latent reservoirs become a major barrier for curing HIV infection. A better understanding of HIV latent reservoir establishment will enhance the chance of eradicating the virus in patients. However, how the virus preferentially choose latent infection or productive infection is still puzzling. Knowing that resting T cells are not easily infected by HIV, Dr. Shen observed that when resting T cells co-culture with endothelial cells, they become much more susceptible to viral infection. Is it possible that latent infection established in the endothelial cell co-cultured resting T cells? Does the endothelial cells have anything to do with latent infection? This summer, we explored endothelial cell co-cultured resting T cells.

In my experiment, I used a “reformed” virus to infect resting T cell, endothelial cells co-cultured T cell, and activated T cell. A “reformed” virus is engineered to capable of infecting human T cells but not threaten life. After infection, I can see how the infected cells are different from uninfected cells in the resting T cell culture, endothelial cells co-cultured T cell culture, and activated T cell culture. One interesting difference is the expression of activation markers (such as CD38 and PD1 markers) on the cell surface. To observe if a cell has expressed an activation marker, I can use its complementary antibody to stain the cell and then see if the antibody stained successfully.

As a result, I found that both CD38 and PD1 expression increased from resting T cell culture to endothelial cells co-cultured T cell culture to activated T cell culture. Now, the questions are “Does HIV infected cells express higher CD38 and PD1 marker?” and “what is the co-relation between CD38/PD1 expression and infection?” I compared the uninfected with infected cultures. Infected cell cultures only express a slightly higher activation than the uninfected cells. What do this mean to endothelial cells’ role to resting T cells when co-cultured? It means that CD38 and PD1 markers probably don’t play a significant role in increasing resting T cell’s susceptibility to HIV.

Not to boast, this research experience is life changing to me. I encountered many obstacles in the past summer. In the beginning, I had great troubles to carry out consistent experiments. Thankfully I’m learning to pay attention to details now. I also found building a healthy relationship with lab partners difficult to me. Overall, if I’m planning to do anything profitable (or significant) in the area of biomedical research, I need to constantly shape my personality and share my obstacles.