I examined the biology behind and the mathematical modeling of the interactions between the Human Immunodeficiency Virus (HIV) and immune system cells known as CD4+ T-cells (helper T-cells). I give a basic explanation of viruses, the immune system, and how HIV affects and ultimately destroys the immune system. I examine four-equation system of differential equations and solve using Euler’s Method in MATLAB, allowing us to track the populations over time. I next examine a four-equation system of differential equations that distinguishes between latently and actively infected helper T-cells. This system is analyzed for steady-state solutions and their stability. Two steady-state solutions are found: an uninfected steady state and an endemically infected steady state with a constant level of virus. Finally, I model a mutating virus. The mutations of HIV create the greatest challenge to developing effective drug therapies, as the virus always seems to stay one step ahead of the body’s defense mechanisms. This causes the viral population to increase without bound, overwhelming the immune system and leading to the condition known as Acquired Immunodeficiency Syndrome (AIDS).

The equations for the populations are therefore as follows:

\[
\begin{align*}
\frac{dx}{dt} &= \lambda - d \cdot x - ay,
\frac{dy}{dt} &= k_x - dx - yv,
\frac{dv}{dt} &= ky - uv,
\frac{dw}{dt} &= d(vw - ay).
\end{align*}
\]

These are commonly known as helper T-cells. Helper T-cells play a key role in the immune response. Helper T-cells help both B-cells and killer T-cells to mount effective immune responses. Thus, if one helper T-cells are destroyed, the entire specific immune response fails. HIV kills the very cells that are required by our bodies to defend us from pathogens, including HIV itself. This is one reason that HIV is such a deadly virus. The infected person then contracts a variety of diseases to which uninfected individuals are resistant, and that person is then said to have AIDS.

I wrote a program in MATLAB to use Euler’s method to solve this system of differential equations. I used the parameter values given in Nowak and May’s book: \( \lambda = 105, d = 0.1, a = 0.5, \beta = 2^{10} - 0.7, k = 100, \delta = 5 \). Each is plotted as population vs. time.

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In the paper Dynamics of HIV Infection of CD4+ T Cells by Perelson et al., four differential equations were given. The four variables are: uninfected CD4+ T-cells, \( T_u \); latently infected CD4+ T-cells, \( T_l \); actively infected CD4+ T-cells, \( T_a \); and virus particles, \( V \). The equations are:

\[
\begin{align*}
\frac{dT_u}{dt} &= \lambda - d \cdot T_u - ay,
\frac{dT_l}{dt} &= k_x - dx - yv,
\frac{dT_a}{dt} &= ky - uv - d(T_a)w,
\frac{dV}{dt} &= dv - wV - dVv.
\end{align*}
\]

This model differentiates three kinds of T-cells: the normal variety of HIV T-cells, \( T_u \); T-cells that are infected with HIV but not producing free virus, \( T_l \); and T-cells that are infected with virus and are actively producing new virus, \( T_a \). In this model, only the active T-cells can produce new virus, and only the totally uninfected T-cells can become infected.

In summary, I have examined and analyzed the basic virus dynamics model. I have looked into current drug therapy strategies and how they change the model. I also examined a model that is tailored specifically to HIV. I analyzed this for the stability of the steady states. I analyzed the stability of the steady state solutions using Routh-Hurwitz criteria.