type of white blood cell, is especially important since it helps other cells fight the virus. However, the HIV-1 virus use the CD4-positive T-helper cells to create more virions, destroying the CD4-positive T-helper cells in the process.

We can develop a system of differential equations to better understand the dynamics of the HIV-1 virus [20]. Let $V = V(t)$ be the population of the HIV-1 virus at time t. We will assume that the virus concentration is governed by the following differential equation,

$$\frac{dV}{dt} = P - cV.$$

The first term, P is some function of t that determines the rate at which new viral particles are created. The term $-cV$ is the death rate for the virions. If someone discovers a drug that blocks the creation of new HIV-1 virions, then P would be zero and the virions would clear the body at the following rate,

$$\frac{dV}{dt} = -cV,$$

and $V(t) = V_0 e^{-ct}$, where V_0 is the initial viral population.

Now let us consider a model for the concentration $T = T(t)$ of (uninfected) CD4-positive T-helper cells,

$$\frac{dT}{dt} = s + pT\left(1 - \frac{T}{T_{\max}}\right) - d_T T.$$

The constant s represents the rate at which T-cells are created from sources in the body, such as the thymus. New CD4-positive T-helper cells can also be created from the proliferation of existing CD4-positive T-helper cells, and the second term in the equation represents the logistic growth of the T-cells, where p is the maximum proliferation rate and $T_{\max}$ is the T-cell population density where proliferation ceases. Finally, d_T is the death rate of the T cells.

Like the influenza virus, the HIV-1 virus is an RNA virus. An RNA virus cannot reproduce on its own and must use the DNA from a host cell. To do this, the virus attaches itself to a CD4-positive T-helper cell and injects its RNA into the cell. This way the virus can use the T-cell's DNA to replicate itself using a process called reverse transcription, where a DNA copy of the virus's RNA is made. New virus particles are created, and the T-cell eventually bursts releasing the virions into the body. If we let T^* be the concentration of infected T-cells, we can model this process with the following system of equations,

$$\begin{aligned}\frac{dT^*}{dt} &= kTV - \delta T^* \\ \frac{dV}{dt} &= N\delta T^* - cV,\end{aligned}$$

where δ is the rate of loss of the virus producing T-cells and N is the number of virions produced per infected T-cell during its lifetime. The term kTV tells us the rate at which the HIV-1 virus infects T-cells. This is the same idea as modeling how predators interact with prey in a predator-prey model. Thus, our complete model becomes

$$\begin{aligned}\frac{dT}{dt} &= s + pT\left(1 - \frac{T}{T_{\max}}\right) - d_T T - kTV \\ \frac{dT^*}{dt} &= kTV - \delta T^* \\ \frac{dV}{dt} &= N\delta T^* - cV.\end{aligned}$$