To determine the viral titer, we used retroviruses produced from PT67 packaging cell lines. The retroviruses produced from the same PT67 cells used in the viral titer experiment were K was the only term that required iteration to obtain. When k is increased, the model cell staining with propidium iodide was carried out to determine live and dead cell counts. Rather than use the ordinary differential equation solver on Matlab, we opted to use Euler’s when the infected host cells (Jurkats) express this protein after the gene has been integrated Cell staining with propidium iodide was carried out to determine live and dead cell counts. Finding Number of Infected Cells When the infected host cells (Jurkats) express this protein after the gene has been integrated into their genome by using the reverse transcriptase material also carried in the virus. The GFP gene produces fluorescence light, which can be detected under a fluorescence microscope or using flow cytometry (Mitra et al. 1996). In this manner, successfully infected cells can be measured and hence, the number of infected cells can be measured. It is important to note that due to laboratory safety precautions, our retroviruses do not produce additional viral particles through bursting of the host cell — in other words, the initial viral particles introduced to the system are the only viral particles present in the system. We chose to use Jurkat cells as the host cells, which are immortalized human T lymphocyte cells, because as cancer cells, they will proliferate constantly at a predictable rate. In this paper, we present a group of differential equations that models the factors that influence initial viral infection in a group of cells. We were able to determine a specific rate of infection for all experimental groups, a consistent growth rate of the infected population and uninfected population, and a model of viral decay. We found that the rate of infection is dependent on the initial number of viral particles present to a certain extent, and that there is a significant difference in the growth rate of cells that are uninfected and infected.

### CONCLUSIONS
- We found that in the two highest dilutions we used, the rate of infection was relatively similar, but a decrease in the rate of infection could be seen in the lowest dilution. In addition, we found in our experiments that the highest viral titer surprisingly did not produce the highest infection efficiency. Rather, a two-fold dilution lower generated the highest percentage of infected cells.
- We did, however, find a correlation between the number of viral particles present at initial infection and the resulting rate of cell growth. As expected, the more virus particles present, the slower the cell are able to bounce back from the infection and resume normal growth rates.
- We noticed that the infected cells had a slower growth rate after infection, despite being the exact same cells as the uninfected cells. This suggests that something in the infection and genome integration process hinders the cell proliferation ability and rate.
- While the model for uninfected cells holds in the short run.

### METHODS

#### Viral Titer
- To determine the viral titer, we used retroviruses produced from PT67 packaging cell lines, which are capable of infecting both human and mice cells to infect mouse NIH3T3 cells. The virus was infected at various dilutions (1:10, 1:100, 1:1,000, and 1:10,000).
- To determine the viral titer, we used the following equation:

\[
\%\text{GFP} = \frac{\text{starting cell #}}{\text{dilution} \times \text{total volume}}
\]

The titer was determined to be 455,500 infecting units/mL.

#### Viral Infection Protocol
- The retroviruses produced from the same PT67 cells used in the viral titer experiment were transduced into Jurkat cells, an immortalized line of human T lymphocyte cells. The cells were infected with varying titers of virus using a spinfection protocol, and polybrene was added to aid in infection efficiency.

#### Finding Number of Infected Cells
- When the infected host cells (Jurkats) express this protein after the gene has been integrated into their genome, the cells will emit fluorescence in the FITC channel when excited with a 488nm laser. Thus, infected cells can be determined through positive expression of GFP, as determined by a flow cytometer.

#### Finding Number of Live and Dead Cells
- Cell staining with propidium iodide was carried out to determine live and dead cell counts. Propidium iodide is a non-membrane permeable intercalating agent that stains DNA in the PERCP channel, and is useful for staining dead cells because the DNA will only be stained if the cell is dead and leaking DNA into the environment.

### EQUATIONS

#### BOX MODEL

**EQUATIONS**

\[
\begin{align*}
&\frac{dU}{dt} = a - \frac{U}{V}M \\
&\frac{dV}{dt} = N - \frac{V}{K}
\end{align*}
\]

**Figure 1.** Box model of a viral infection model in which viral infection occurs only at time 0 and cells are able to continuously replicated, either infected or uninfected, without constraint.

**MATHEMATICAL MODELS**

**Figure 2.** Mathematical modeling of different viral infections over time for a) no virus or b) a 1:2 initial viral titer

**Table 2.** Correlation Coefficients Between Model and Actual Data

<table>
<thead>
<tr>
<th>Titer</th>
<th>Uninfected (100hrs)</th>
<th>Infected (100hrs)</th>
<th>Uninfected (1000hrs)</th>
<th>Infected (1000hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:1</td>
<td>0.9984</td>
<td>0.9505</td>
<td>0.9953</td>
<td>0.5196</td>
</tr>
<tr>
<td>1:2</td>
<td>0.9995</td>
<td>0.9080</td>
<td>0.9889</td>
<td>0.5196</td>
</tr>
<tr>
<td>1:4</td>
<td>1</td>
<td>0.9521</td>
<td>0.9999</td>
<td>0.5196</td>
</tr>
</tbody>
</table>

**CONCLUSIONS**

- We found that in the two highest dilutions we used, the rate of infection was relatively similar, but a decrease in the rate of infection could be seen in the lowest dilution.
- In addition, we found in our experiments that the highest viral titer surprisingly did not produce the highest infection efficiency. Rather, a two-fold dilution lower generated the highest percentage of infected cells.
- We did, however, find a correlation between the number of viral particles present at initial infection and the resulting rate of cell growth. As expected, the more virus particles present, the slower the cell are able to bounce back from the infection and resume normal growth rates.
- We noticed that the infected cells had a slower growth rate after infection, despite being the exact same cells as the uninfected cells. This suggests that something in the infection and genome integration process hinders the cell proliferation ability and rate.
- While the model for uninfected cells holds in the short run (under 100 hours) and long run (under 1000 hours), but the model for infected cells only holds in the short run.