3.T.1         Virus diffuses in the microenvironment with low diffusion coefficient  
3.T.2         Virus adhesion to a cell stops its diffusion (acts as an uptake term)  
3.T.3         Pro-inflammatory cytokine diffuses in the microenvironment  
3.T.4         Pro-inflammatory cytokine is taken up by recruited immune cells  
3.T.5         Pro-inflammatory cytokine is eliminated or cleared  
3.T.6         Chemokine diffuses in the microenvironment  
3.T.7         Chemokine is taken up by immune cells during chemotaxis  
3.T.8         Chemokine is eliminated or cleared  
3.T.9         Debris diffuses in the microenvironment  
3.T.10       Debris is taken up by macrophages and neutrophils during chemotaxis  
3.T.11       Debris is eliminated or cleared

3.RT.1      Virus adheres to unbound external ACE2 receptor to become external (virus)-bound ACE2 receptor  
3.RT.2      Bound external ACE2 receptor is internalized (endocytosed) to become internal bound ACE2 receptor  
3.RT.3      Internalized bound ACE2 receptor releases its virion and becomes unbound internalized receptor. The; the released virus is available for use by the viral lifecycle model **V**  
3.RT.4      Internalized unbound ACE2 receptor is returned to the cell surface to become external unbound receptor  
3.RT.5      Each receptor can bind to at most one virus particle.

3.V.1        Internalized virus (previously released in 2.RT.3) is uncoated  
3.V.2        Uncoated virus (viral contents) lead to release of functioning RNA  
3.V.3        RNA creates viral protein forever[[1]](https://app.slack.com/client/T011202KRL7/D010ZFL4NP6/thread/C013Y43H7H6-1591228841.026000#_msocom_1) , unless it degrades  
3.V.4        Viral protein is transformed to an assembled virus state  
3.V.5        Assembled virus is released by the cell (exocytosis)

3.VR.1      After infection, cells begin to secrete chemokine  
3.VR.2      As a proxy for viral disruption of the cell, the probability of cell death increases with the total number of assembled virions  
3.VR.3      Apoptosed cells lyse and release some or all of their contents

3.D.1        Dead cells secrete debris

3.MPhi.1   Macrophages phagocytose dead cells  
3.Mphi.2   After phagocytosing dead material, macrophages secrete pro-inflammatory cytokines  
3.Mphi.3   Macrophages are recruited into tissue by pro-inflammatory cytokines.  
3.Mphi.4  Macrophages move locally in the tissue along chemokine and debris gradients  
3.MPhi.5   Macrophages die naturally and become dead cells.  
3.MPhi.6   Macrophages also die after collecting sufficient dead cell material.

3.N.1        Neutrophils are recruited into the tissue by pro-inflammatory cytokines  
3.N.2        Neutrophils die naturally and become dead cells  
3.N.3        Neutrophils move locally in the tissue along chemokine and debris gradients  
3.N.4        Neutrophils phagocytose dead cells  
3.N.5        Neutrophils uptake virus

3.CD8.1    CD8+ T cells are recruited into the tissue by pro-inflammatory cytokines  
3.CD8.2    CD8+ T cells apoptose naturally and become dead cells  
3.CD8.3    CD8+ T cells move locally in the tissue along chemokine gradients  
3.CD8.4    CD8+ T cells induce apoptosis in infected cells with some probability