1048-34-147 Ian Price* (imp5@pitt.edu), 301 Thackeray, Pittsburgh, PA 15260, David Swigon (swigon@pitt.edu), 301 Thackeray, Pittsburgh, PA 15260, G Bard Ermentrout (bard@math.pitt.edu), 301 Thackeray, Pittsburgh, PA 15260, and Gilles Clermont (cler@pitt.edu), 3350 Terrace St, Pittsburgh, PA 15261. MATHEMATICAL MODEL OF THE MAMMALIAN IMMUNE RESPONSE TO INFLUENZA A.

Influenza A virus triggers innate before adaptive immunity; but, an exaggerated response harms tissue and does not further viral elimination. This project models the immune response, and identify methods of preventing lung failure and improving recovery. The model introduces a dynamic innate immunity with relevant biology, expanding upon existing models of adaptive immune response to Influenza A virus. The inflammatory process begins with macrophage mediated production of cytokines and chemotaxis of immune cells. Viral immune responses such as interferon I and NK cells are introduced. Interferon II, CTLs and antibodies are produced to complete virus removal. The project employs an ODE–based model to study the dynamics of regulation between virus, immune and respiratory cells, and signaling macromolecules. The system allows for stable health and death, with initial viral load leading to each. For some choices of key parameters unstable health and a stable chronic state with viral clearance can be attained. The model gives us a metric relating initial infection, strength of various immune responses, and total lung damage. Decoding the pathways of immunity allows us to measure their effect on damage and recovery, and motivates experimental studies. (Received February 04, 2009)