1035-92-1169 **Angela Reynolds*** (amr31@pitt.edu), 301 Thackeray Hall, Department of Mathematics, Pittsburgh, PA 15260, and **Gilles Clermont**, **Jonathan Rubin** and **G Bard Ermentrout**. *A model of the acute inflammatory response with immune mediators in both the blood and tissue and the effects of linking multiple tissue units to the same blood compartment.*

During an inflammatory response to an infection within the tissue, local macrophages are activated. These macrophages produce cytokines that diffuse into the blood and trigger a local influx of activated neutrophils. The interplay between compartments is essential in eliminating infection and restoring homeostasis. Therefore, we expanded our previous acute inflammation model to incorporate a distinction between immune components that are in the blood and those in the tissue. Our model for the acute inflammatory response consists of ODE's for immune components in both the tissue and/or blood. We track overall inflammation to determine changes in diffusion and tissue integrity. During the development of the model multiple subsystems were analyzed. This approach allowed us to develop a model in which known dynamics are present, such as bistability and excitability. Combining subsystems we formed our compartmental model. Using one blood compartment we linked multiple tissue units creating a multi-tissue model. Linking identical tissue units allows the acute immune response to overcome a larger pathogen insult than that seen in a single tissue unit. Adding diversity to the tissue will allow us to explore the spread of infection between organs. (Received September 18, 2007)