



Virginia Commonwealth University  
VCU Scholars Compass

---

Biology and Medicine Through Mathematics  
Conference

---

## In silico mouse model of infection and immunity

Daniel Jonas

*Colorado State University, [djonas25@gmail.com](mailto:djonas25@gmail.com)*

Michael Kirby

*Colorado State University, [kirby@math.colostate.edu](mailto:kirby@math.colostate.edu)*

Alan R. Schenkel

*Colorado State University, [Alan.Schenkel@colostate.edu](mailto:Alan.Schenkel@colostate.edu)*

Follow this and additional works at: <https://scholarscompass.vcu.edu/bamm>



Part of the [Immunity Commons](#), [Immunology of Infectious Disease Commons](#), [Medicine and Health Sciences Commons](#), and the [Physical Sciences and Mathematics Commons](#)

---

<https://scholarscompass.vcu.edu/bamm/2020/talk/5>

This Event is brought to you for free and open access by the Dept. of Mathematics and Applied Mathematics at VCU Scholars Compass. It has been accepted for inclusion in Biology and Medicine Through Mathematics Conference by an authorized administrator of VCU Scholars Compass. For more information, please contact [libcompass@vcu.edu](mailto:libcompass@vcu.edu).

An organism's immune system tries to protect it by identifying the presence of pathogens and attempting to eliminate them. The defense is twofold: innate immune cells mobilize rapidly, while acquired immune cells slowly develop into pathogen-killing specialists. These responses incur collateral tissue damage, which anti-inflammatory mediators seek to control. This system of checks and balances is responsible for host survival. Experimental research has demonstrated how vastly complex these interactions are, indicating a place for theoretical and computational study. In this work we develop a comprehensive differential equation model of the immune system by considering interactions between immune system components in the presence of pathogen or tissue trauma. Through this step-by-step construction we explore the dependence of the anti-inflammatory mediators on pathogen levels, and also how they temper the immune response at the end of infection. We then challenge the "virtual mouse" with typical pathogens of varying virulence and observe the outcomes via model simulation. We find that anti-inflammation can downregulate the activation and proliferation of immune cells or promote apoptosis as cessation mechanisms, suggesting the need for *in vivo* experiments. Bifurcation theory describes how the outcomes of infection depend on model parameters, from which we conclude that initial insult and pathogen growth rate allow us to predict whether or not the *in silico* mouse overcomes the disease in a deterministic framework.