



cordially invites you to an

## Interdisciplinary Seminar

with

**Dr. Ruian Ke**

on

### ***“Superinfection and cure of infected cells as novel mechanisms for hepatitis C virus expansion and persistence”***

**Tuesday, October 10, 2017**

3:30-5 p.m.

*Reception & refreshments at 3 p.m.*

Hallam Auditorium, Room 206  
1122 Volunteer Boulevard



Dr. Ruian Ke joined NC State in January 2015 as a Chancellor’s Faculty Excellence Program cluster hire in Personalized Medicine. He is an assistant professor in the Department of Mathematics and a member of the Comparative Medicine Institute. His research focuses on developing mathematical theories and tools to understand viral dynamics, treatments and immune responses across multiple scales of biological organization, including at intracellular, cellular and population scales. An overarching theme of his research is to elucidate emerging principles at higher scales arising from viral-host interactions at lower scales. He aims to use the theories and principles to address pertinent biological, clinical and epidemiological problems.

**Abstract:** RNA viruses exist as a genetically diverse quasispecies with extraordinary ability to adapt to abrupt changes in the host environment. However, the mechanisms that contribute to their rapid adaptation and persistence are not well studied. Here we probe hepatitis C virus persistence by analyzing clinical samples taken from subjects who were treated with a novel protease inhibitor. Frequent longitudinal viral load determinations and single genome sequence analyses revealed rapid antiviral resistance development, and surprisingly, dynamic turnover of dominant drug resistant mutant populations long after treatment cessation. We fitted mathematical models to the data, and the results provided strong support for the critical roles that superinfection and cure of infected cells play in facilitating the rapid turnover and persistence of viral populations. Thus, we propose a new theoretical framework integrating viral and molecular mechanisms to explain rapid viral evolution, resistance and persistence despite antiviral treatment and host immune responses.