RYERSON UNIVERSITY

DEPARTMENT OF MATHEMATICS
BIOMATHEMATICS & FLUIDS SEMINAR

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Date: Monday, February 22, 2016
Time: 9:30am
Location: ENG 162

Mathematically Modelling the Body's Response to Cancer

Abstract:

In this talk, I will discuss separately two mathematical frameworks constructed to capture and analyze aspects of cancer as it interacts with the whole body. This work arises from interdisciplinary collaborative projects at the Center of Cancer Systems Biology in Boston, Massachusetts.

Cancer progression is modulated by interactions with healthy host cells. Nonlinear growth dynamics result from immune interference, causing periods of growth stimulation, inhibition, or forestallment (known as tumor dormancy). Cancer presence can also deteriorate host health by causing irreversible loss of body mass (known as cancer cachexia).

The first framework is a dynamical systems approach to model the stimulatory and inhibitory nature of the immune response to cancer. Simulations and phase-plane analysis demonstrate how this approach, which can predict the transient nature of immune-induced tumor dormancy, is an improvement over existing models, which cannot. I will discuss how dormancy is captured by the model, the process of parameterization, and the significant implications of this mathematical analylsis to our understanding of tumor dynamics and disease treatment.

The second framework is a multi-scale approach to investigate systemic tumor-host communication in cachexia. We hypothesize that mesenchymal stem cells in host muscle and adipose tissues are susceptible to recruitment and reprogramming by tumor-derived systemic factors in blood, leading to the development of cachexia. Specifically, we compare 2 signaling methods: soluble factors (proteins) and microvesicles (proteins protected by a lipid bilayer). I will briefly present our tri-scale analysis, including genetic-level alterations, the physical diffusive properties of the signaling molecules, and host-level mechanisms for tissue loss. Computational biology, pathway analysis, and protein interaction networks are used at the smallest scale. The diffusion PDE is used at the tissue-scale. And a system of ODEs is used at the host scale. I will conclude by discussing the biological implications of this work, as well as possible future directions, open problems, and challenges in cancer systems biology and mathematical multi-scale modeling.

All Faculty, staff, students and guests are welcome to attend