

## Abstracts

**Mohammadkheer Al-Jararha (Memorial):** *Age-structured population dynamics: age-dependent diffusion and death rates, and their applications to the cell population*

This talk is divided into two parts. In the first part, we consider the standard age-structured model with diffusion

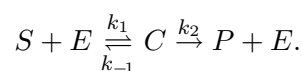
$$\frac{\partial u}{\partial t} + \frac{\partial u}{\partial a} = D(a) \frac{\partial^2 u}{\partial x^2} - d(a)u,$$

where  $u(t, a, x)$  is the population density of the species at time  $t$ , age  $a$ , and location  $x$  in  $\Omega$  ( $\Omega = \mathbb{R}$  or  $\Omega = [a, b] \subseteq \mathbb{R}$ ). The age-functions  $D(a)$  and  $d(a)$  are the age-dependent diffusion and death rates, respectively. Using this model, we constructed a completely new model in terms of an integral equation. To investigate the new model, we consider two cases, the unbounded spatial domain case as well as the bounded domain case. For the unbounded domain case, we investigate the existence of traveling wave solutions for different choices of birth functions. For the bounded spatial domain case, we investigate the existence and stability of a strongly positive steady state solution for different choices of birth functions. As a by-product for this case, we also prove rigorously the existence of a real principal eigenvalue with a corresponding strongly positive eigenfunctions.

In the second part of this talk, we present an age-size structured model to represent the dynamics of cell population in two comparative phases  $G_0$  – Phase (the resting phase) and  $P$  – Phase (the proliferation phase), during the cell-division cycle. Using this model, we derived a non-linear delay differential equation with a non-local term to represent the density of cell population in the resting phase ( $G_0$ –Phase). We also investigate the local stability of the zero solution of this delay differential equation. To do this, we investigate the analogous variational linear delay differential equation around the zero solution. Conclusively, we show that the linear delay differential equation admits a real eigenvalue, as well as a strongly positive real eigenfunction. For each case of study, we present a numerical simulation.

**Matt Calder (Western Ontario):** *Estimates for solutions of the planar and scalar reductions of the Michaelis-Menten mechanism*

The Michaelis-Menten mechanism is the simplest chemical network modeling the formation of a product from a substrate with the aid of an enzyme. Specifically, an enzyme  $E$  reacts with the substrate  $S$  and reversibly forms an intermediate complex  $C$ , which decays into the product  $P$  and original enzyme:



Although relatively simple, many results and techniques for this mechanism are easily adapted to more complicated networks. The Law of Mass Action, conservation laws, and a rescaling yield a dimensionless planar reduction, consisting of a system of two ordinary differential equations for the scaled substrate concentration  $x(t)$  and scaled complex concentration  $y(t)$ , where  $t$  is time. By suppressing the dependence on  $t$ , we can also form the scalar reduction, consisting of an ordinary differential equation

for the complex concentration  $y(x)$  as a function of the substrate concentration. In this talk, I will present results on the phase portrait which are independent of the size of the parameters. Furthermore, I will develop estimates for  $x(t)$  and  $y(t)$  (for large  $t$ ) and  $y(x)$  (both for small and large  $x$ ). Using these estimates, I will comment on the validity of the quasi-steady-state assumption, which results in a hyperbolic rate law, and present a better hyperbolic rate law. Finally, I will estimate the length of the pre-steady-state period, after which the quasi-steady-state assumption is valid.

**Andy Foster (Memorial):** *Effects of caffeine on the human sleep-wake cycle*

A recent model of Rempe et al. (J. Math. Biol. 60, 2010) effectively predicts the characteristics of the human sleep-wake cycle, including transition into and out of sleep, as well as the REM-NREM stages of sleep. We modify this model to more closely reflect current literature on homeostatic component decay during sleep, and use our modified model to predict the effects of caffeine intake on the human sleep-wake cycle and the REM-NREM sleep cycle. Joined work with Nicole Stockley.

**Xi Hu (New Brunswick):** *Mathematical model with diatom, corophium and snail*

Diatom, snails and corophium are three important species in the Bay of Fundy. In this talk, I will present a two-dimensional ODE model to demonstrate their interactions. More precisely, it is shown that if there are snails, then there will be a limit cycle and both diatom and corophium coexist in the environment. We also investigate all other possible dynamic behavior as the snail's population changes.

**David Iron (Dalhousie):** *Roll of cellular geometry in the formations of morphogen patterns*

We examine a specific reaction diffusion system responsible for the formations of cellular polarity in the development of a nematode. We demonstrate that simple reaction-diffusion dynamics cannot account for the observed pattern formation. However if the effects of the domain geometry is included, robust stable patterns result.

**Chris Levy (Dalhousie):** *Model of cell signal transduction in a 3-dimensional domain*

Intracellular signalling molecules form pathways inside the cell. These pathways carry a signal to target proteins which results in cellular responses. We consider a spherical cell with two internal compartments containing localized activating enzymes where as deactivating enzymes are spread uniformly through out the cytosol. Two diffusible signalling molecules are activated at the compartments and later deactivated in the cytosol due to deactivating enzymes. The two signalling molecules interact through a simple cascade reaction and form a self regulated dynamical system involving positive and negative feedback. Using matched asymptotic expansions we obtain three dimensional concentration profiles for the signalling molecules.

**Xin Li (Western Ontario):** *A diffusion model for sterile insect release method with release only on the boundary of the habitat*

The sterile insect release method (SIRM), originally suggested by E.F. Knippling, is one way of pest control. By releasing the same kind but sterile insects into the habitat of the pest, one can dilute the

reproductive capacity of fertile pests, thus controlling the density of pests, which, in particular, can be used for eradication. In this talk, we consider a SIRM space model with release only on boundary of the habitat. Choosing the release amount as the discussing parameter, we give the conditions determining the existence and non-existence of the coexistence steady states. We show the existence of steady states by using upper and lower solutions.

**Rui Peng (Memorial):** *A reaction-diffusion SIS epidemic model in a periodic environment*

In this talk, we consider a susceptible-infected-susceptible (SIS) reaction-diffusion model, where the rates of disease transmission and recovery are assumed to be spatially heterogeneous and temporally periodic and the total population number is constant. We introduce a basic reproduction number  $R_0$  and establish the threshold type results on the global dynamics in terms of  $R_0$ . In particular, we obtain the asymptotic properties of  $R_0$  with respect to the diffusion rate  $d_I$  of the infected individuals, which exhibit the delicate influence of the time-periodic heterogeneous environment on the extinction and persistence of the infectious disease. Our analytic results suggest that the combination of spatial heterogeneity and temporal periodicity tends to enhance the persistence of the disease. This is a joint work with Dr. Xiaoqiang Zhao.

**Hongying Shu (New Brunswick):** *Impact of mitosis and intracellular delay on viral dynamics: stability, and bifurcation analysis*

The dynamics of an in-host model with general form of target-cell dynamics, nonlinear incidence and distributed delay are investigated. We derive the basic reproduction number  $R_0$  for the viral infection, and establish that the global dynamics are completely determined by the values of  $R_0$ . An implication is that intracellular delays will lead to periodic oscillations in in-host models only with the right kind of target-cell dynamics. To understand joint effects of logistic growth in target cells and intracellular delay on viral dynamics in vivo, we carry out two-parameter bifurcation analysis of an in-host model that describes infections of many viruses including HIV-I, HBV and HTLV-I. The bifurcation parameters are the mitosis rate  $r$  of the target cells and an intracellular delay  $\tau$  in the incidence of viral infection. We describe the stability region of the chronic-infection equilibrium  $E^*$  in the two-dimensional  $(r, \tau)$  parameter space, as well as the global Hopf bifurcation curves as each of  $\tau$  and  $r$  varies. Our analysis shows that, while both  $\tau$  and  $r$  can destabilize  $E^*$  and cause Hopf bifurcations, they do behave differently. The intracellular delay  $\tau$  can cause Hopf bifurcations only when  $r$  is positive and sufficiently large, while  $r$  can cause Hopf bifurcations even when  $\tau = 0$ . Intracellular delay  $\tau$  can cause stability switches in  $E^*$  while  $r$  does not.

**Lin Wang (New Brunswick):** *Backward bifurcation induced by proliferation of T cells in an HIV model*

For HIV patients, lifelong therapy is generally required to control their viral replication and it is very difficult to effectively eradicate HIV infection from the host. In this paper, using a mathematical model, we show that the stimulation of T cells to proliferate in the presence of HIV is one possible

factor causing such difficulties. This is based on the fact that if the proliferation rate is non-negligible, then there exists a bistability region over which the virus can either be cleared or persist depending on the initial state. The model exhibits a backward bifurcation and hysteresis. Outside of the bistability region, the infection persists if the basic reproduction number  $R_0 > 1$ , otherwise, the infection is cleared. This is derived from the global stability of infection free equilibrium and infection persistent equilibrium established by the method of Liapunov function and the geometric approach. The model is then extended to the case where the loss of free infectious virions during attacking the target cells is taken into consideration. Our analysis shows that a backward bifurcation is still possible. This is joint work with Dr. Hongying Shu.

**Wendi Wang (Southwest University, China):** *Adaptive dispersals of populations between two patches*

I will talk about influences of adaptive migrations of populations between patches. First, we consider the case where emigration rates of populations are adjusted by local survival conditions. Then we suppose that populations can get global information to decide their dispersal strategies.

**Xiang-Sheng Wang (York):** *Asymptotic analysis in migration ecology and simple SIR epidemiology*

I will use a seasonal bird migration model and a SIR model to illustrate the connection between asymptotic analysis and biological dynamics.

**James Watmough (New Brunswick):** *Switching-type neural network models for the task-regulation in honey bee colonies*

Honey Bees show a remarkable ability to coordinate activities in a colony. It is thought that this coordination is accomplished through the production of and response to pheromones produced by the queen, brood and workers. This talk presents a model for the regulation of the ratio of forager bees to hive bees in a colony based on the hypothesis that foragers produce a pheromone that represses worker development. The model is cast as a system of differential equations with discontinuous right-hand-sides similar to models used for switching-type neural networks. It is shown that with some reasonable restrictions the model has a set of equilibria that are globally stable up to permutations of the indices, representing a stable equilibrium ratio of workers to foragers. This research is funded by the Human Frontier Science Program ([www.hfsp.org](http://www.hfsp.org)). This is joint work with PhD student Fang Yu.

**Yuan Yuan (Memorial):** *A coupled plankton system with instantaneous and delayed predation*

We present two simple plankton population models, one has instantaneous predation, another has delayed predation. The models consist of two coupled differential equations representing the interaction between phytoplankton and herbivorous zooplankton with additional effect of zooplankton predation by a constant fish population. We study the dynamical behavior and investigate the conditions to guarantee the coexistence of two species, address the stability and bifurcation under different density of fish, with or without the maturation time delay. Analytical methods and numerical simulations are

used to obtain information about the qualitative behavior of the models.

**Yuxiang Zhang (Memorial):** *Bistable travelling waves in competitive recursion systems*

In this talk, I will report our recent research on the existence and global stability of bistable travelling wave solutions for a class of discrete-time recursion systems, which describe the spatial propagation of two competitive invaders. The methods involve the upper and lower solutions, spreading speeds of monostable systems, and the monotone semiflows approach. The numerical simulations are shown for the analytic results. This talk is based on the joint work with Dr. Xiaoqiang Zhao.

**Xiaoqiang Zhao (Memorial):** *Global dynamics of a reaction and diffusion model for Lyme disease*

In this talk, I will report my recent research on a reaction and diffusion model for Lyme disease, which was proposed in [Caraco et al., *The American Naturalist*, 160(2002), 348-359]. In the case of a bounded spatial habitat, we obtain the global stability of either disease-free or endemic steady state in terms of the basic reproduction number. In the case of an unbounded spatial habitat, we establish the existence of the spreading speed of the disease and its coincidence with the minimal wave speed for traveling fronts. Our analytic results show that the basic reproduction number is a threshold value for the global dynamics and that the spreading speed is linearly determinate.