**Mathematics Inspired by Biological Models**
**Mathématiques inspirées par des modèles biologiques**
(Org: **Fred Brauer** (UBC) and/et **Pauline van den Driessche** (Victoria))

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**JULIEN ARINO**, University of Manitoba
*An alternative formulation for a delayed logistic equation*

The logistic equation with time delay is closely linked to the evolution of the theory of delay differential equations (DDE). Known in the mathematical community as Wright's equation, it is a standard example of the richness of behaviors exhibited by DDE, but also of the problems that arise in their analysis. However, the delayed logistic equation is seldom used in theoretical ecology. After an introduction to the delayed form of the logistic equation, I will discuss the reasons that lead to its shunning by the theoretical ecology community. I will then propose an alternative formulation of the equation taking into account survival through the maturation process. This alternative form, of which a global analysis has been conducted, has a totally different behavior. I will describe this behavior, pointing out situations in which our equation seems better suited for the description of single species dynamics with delay than the classical DDE logistic.

This is joint work with Lin Wang and Gail Wolkowicz.

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**CAROLINE BAMPFYLDE**, University of Alberta, 632 Central Academic Building, Edmonton, Alberta T6G 2G1
*Perturbing a littoral-zone lake community to release a biological*

Rusty crayfish (*Orconectes rusticus*) are aggressive invaders of the Great Lakes ecosystem. When introduced into new lakes, they drive down native crayfish populations, disturb macrophytes, interfere with fish recruitment, and cause the overgrazing of algae and snails. Recently, the population density of rusty crayfish in some lakes has far exceeded previously recorded levels. Management of this nuisance species is necessary.

The interaction between rusty crayfish and indigenous smallmouth bass (*Micropterus dolomieu*) involves a mixture of competitive and predator-prey relationships. Juvenile smallmouth bass compete with all life stages of the invasive rusty crayfish. However, mature smallmouth bass are major predators of rusty crayfish. Intraspecific interactions for rusty crayfish also include cannibalism and resource competition.

We used mathematical and computer models to investigate the influence of biological control of rusty crayfish by smallmouth bass. The method is to apply perturbations to shift the dominance in a competitive bottleneck from rusty crayfish to smallmouth bass. The perturbations include crayfish trapping, trawling and changes to lake fishing regulations.

Our model was developed and parameterised using long term field data and laboratory experiments. The analysis suggests methods for effective control. Model validation will be carried out by use of a controlled experiment in Lake Ottawa, Michigan. We will test the hypothesis that trawling for crayfish is sufficient for control without changing fishing regulations. Our long term goal is to implement the control methods in selected lakes.

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**DANIEL COOMBS**, University of British Columbia, Dept. of Mathematics, Vancouver, BC V6T 1Z2
*Virus competition at multiple scales*

Viruses compete and are subject to natural selection at multiple levels: within-cell, within-host and within-population (of hosts). We looked at how viruses can optimally exploit their hosts and how this behaviour may influence the most successful strategy at the between-host, or epidemiological level. I will present a fairly general way to consistently combine models of disease process and disease spread with the goal of understanding the net selection pressure on a model virus. The method is illustrated using a popular model for HIV dynamics nested within a simple epidemiological model.

This is joint work with Mike Gilchrist (Tennessee).

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**ERIC CYTRYNBAUM**, University of British Columbia, Department of Mathematics
*Finding the center—how to solve simple geometry problems at the cellular scale*

Fragments of fish melanophore cells can form and center aggregates of pigment granules by dynein-motor-driven transport along a self-organized radial array of microtubules (MTs). In this talk, I will present a system of integro-differential equations that model pigment aggregation and MT-aster self-organization and the subsequent centering of both structures. The model is based on the observations that MTs are immobile and treadmill, while dynein-motor-covered granules have the ability to nucleate MTs. Scaling arguments and perturbation theory allow for analysis in limiting cases. This analysis explains the mechanism of aster self-organization as a positive feedback loop between motor aggregation at the MT minus ends and MT nucleation by motors. Furthermore, the centering mechanism is explained as a global geometric bias in the cell established by spontaneously nucleated microtubules. Numerical simulations lend additional supports to the analysis. The model sheds light on role of polymer dynamics and polymer-motor interactions in cytoskeletal organization.

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**LEAH EDELSTEIN-KESHET**, University of British Columbia, Dept. of Mathematics
*Models of actin dynamics and cell motility*

Actin is a biopolymer that forms a major part of the cytoskeleton—the structure that endows shape and motility to animal cells. I will describe the work of Ph.D. student, Adriana T. Dawes (joint with Bard Ermentrout, and Eric Cytrynbaum) on the dynamics of the actin cytoskeleton and its relationship to the speed of motion of a cell. We derive a 1D model describing the density of actin filaments and their tips. In this model, we assume that actin tips push out the leading edge of the cell by the polymerization thermal ratchet mechanism (proposed by Mogilner and Oster, 1996). We include the effects of nucleation of new filaments, capping of their tips, as well as polymerization and disassembly of the filaments to arrive at a set of PDEs. In 1D, the model can be partly analysed in closed form to determine when travelling wave solutions (depicting steady state motion of a cell) exist, and how their speed depends on rate constants and biochemical parameters. Numerical simulations extend the results where analysis is cumbersome. We use the model to investigate the effects of three types distinct mechanisms of filament nucleation, and conclude that side branching best describes experimentally observed actin density distributions.

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**ROD EDWARDS**, University of Victoria, Dept. of Math & Stats, P.O. Box 3045 STN CSC, Victoria, BC V8W 3P4
*Stochastic feedback and beats: a generic model for circadian rhythms*

Most organisms undergo circadian rhythms: at the cellular level, protein concentrations go through 24-hour cycles. These are intrinsic (they run in the absence of light) but respond to the diurnal cycle of sunlight. These cycles are thought to involve genetic regulatory processes—transcription and translation of proteins that affect the expression of other genes and produce oscillations through feedback. However, all such known 'transcriptional-translational oscillators' have periods of no more than 3 hours. So an important question is how such fast, 'ultradian' oscillations can produce slow 'circadian' ones. Another problem is that the particular genes and regulatory processes involved vary from organism to organism. This poses the theoretical question: How did circadian oscillations develop independently using different components in different organisms? We propose a biochemically realistic model that offers possible solutions to both of these questions as well as allowing entrainment by light. The mathematics is elementary but the mechanism is elegant, and some more difficult questions arise when the inherently stochastic nature of the gene regulation is taken into account.

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**MEREDITH GREER**, Bates College, Lewiston, Maine, USA
*Interaction of Infectious and Noninfectious Proteins in Prion Disease: Models, Simulations, and Steady State Study*

A prion is an infectious form of protein that differs from a naturally produced protein only in its folding. Prions are thought to cause several diseases, with BSE (Bovine Spongiform Encephalopathy) perhaps the most widely known example. Diseases associated with prions have very long incubation periods, are difficult to detect in all but the latest stages, and are highly fatal. These characteristics alone make study of prions interesting, but even more so, there is the question of prion replication. Proteins do not possess any nucleic acid. Without DNA or RNA, how does the structure copy itself and spread?

There is evidence that prions form polymers or aggregates, most likely with additional stability. Some or all of these polymers attach to the similar naturally produced protein and convert it to the infectious variety. Polymers also split. Altogether, both the overall quantity of infectious proteins, and the number of polymer strands, increase. To model these phenomena, we represent prion polymer length as a continuous structure variable. We obtain a system of two partial differential equations modeling interaction of the infectious and noninfectious conformations of prion protein within an infected individual. We use this system to create numerical simulations of disease progress within such an individual. Under some circumstances, we can simplify to a system of three ordinary differential equations. In the ODE case, we discuss steady states, their stability, and relative parameter changes that affect their viability.

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**ABBA GUMEL**, University of Manitoba, Dept. of Mathematics, Winnipeg, Manitoba, R3T 2N2
*Modeling the Impact of an Imperfect Vaccine and ART in Curtailing HIV Spread*

Since its emergence in the 1980s, the human immunodeficiency syndrome (HIV) continues to inflict major public health and socio-economic burdens globally. Currently, 34–46 million people live with HIV and over 20 million have so far died of the disease. Although the use of anti-retroviral therapy (ART) has been quite effective in slowing HIV spread in some nations, it is generally believed that the global control of HIV would require a vaccine. This talk aims at using mathematical modelling to assess the potential impact of using an imperfect anti-HIV vaccine and ART in combatting HIV. Deterministic models, which incorporate many of the essential biological features of HIV (such as staged-progression and differential infectivity) and anticipated vaccine characteristics (*e.g.*, "take", "degree", "duration" and offering some therapeutic benefits) as well as the ART-induced evolution and transmission of drug-resistant HIV strain, would be presented and analyzed to determine thresholds conditions for effective control of HIV within a community.

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**THOMAS HILLEN**, University of Alberta
*Mathematical Models for Mesenchymal Motion*

Mesenchymal motion is a form of cellular movement that occurs in three-dimensions through tissues formed from fibre networks, for example the invasion of tumor metastases through collagen networks. The movement of cells is guided by the directionality of the network and in addition, the network is degraded by proteases. I derive mathematical models for mesenchymal motion in a timely varying network tissue. The models are based on transport equations and their drift-diffusion limits. It turns out that the mean drift velocity is given by the mean orientation of the tissue and the diffusion tensor is given by the variance-covariance matrix of the tissue orientations. I will discuss relations to existing models and future applications.

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**LEV IDELS**, Malaspina University-College
*Time delays in periodic harvesting*

To model a fish population in periodic environment we introduce a Getz delay differential equation with a parameter which describes population outbreaks.

This dynamical system then analyzed and the global existence of a solution is established.

We study some harvesting problems that were posed by F. Brauer, *e.g.*, whether periodic variations in the model transform a stable equilibrium to the stable periodic solution, will the stability of equilibrium be preserved and result in stable periodic solution?

We illustrate numerically that the resulting system has a very rich dynamic.

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**MARK LEWIS**, University of Alberta
*Spread and persistence of competitive species in advective flows*

My talk will focus on mathematics inspired by biological problems involving multispecies competitive spread and persistence in advective flows. The biological problems are: movement of vegetation in response to climate change, and persistence of populations in rivers. In the analysis of these problems, I will connect the classical critical domain size problem with the theory of spread rates and travelling waves. I will finish with some recent work on the role of disease in the historical spread of competing species.

Some of the research is in collaboration with Frithjof Lutscher, Paul Moorcroft and Alex Potapov.

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**CONNELL MCCLUSKEY**, Wilfrid Laurier University
*Stability for a Class of Epidemic Models with Mass Action Incidence*

Many epidemic models with mass action incidence can be written as a sum of constant, linear and quadratic terms:

$$x' = K + Nx + Qx_1x_2$$

where $K$ and $Q$ are constant vectors and $N$ is a matrix. Under very reasonable assumptions on $K$, $N$ and $Q$, it will be shown that this $n$-dimensional system has a globally asymptotically stable equilibrium. The result resolves the asymptotic behaviour of several models in the literature for which the global dynamics had not been determined. The main results are obtained by the use of a Lyapunov function.

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**REBECCA TYSON**, University of British Columbia Okanagan, 3333 University Way, Kelowna, BC V1V 1V7
*Recolonization of Harvested Forest stands by Tamiasciurus hudsonicus*

We present a model for the population dynamics of *Tamiasciurus hudsonicus* within a forest environment subject to harvesting and regrowth. The forest is represented as a mosaic of patches at various levels of development from harvested to mature forest. Each patch grows over time, eventually becoming mature forest unless it is harvested again. At the same time, each harvested patch is gradually recolonized by squirrels. We find that the time it takes for a second growth forest patch to be recolonized at the mature forest level is much longer than expected. In particular, it is much longer than the time it takes for the second growth patch to produce as many cones as an equivalent mature forest patch. We also report that recolonization pressure decreases squirrel populations in neighbouring patches. We discuss reasons for these behaviours and predict how squirrel populations are affected by different harvesting geometries.

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**LIN WANG**, University of Victoria
*Transient Oscillations in Chemostat Models*

Despite the fact that the competitive exclusion principle holds in most classical chemostat models, transient oscillations are frequently observed in actual experiments, which cannot be explained from those classical models. What could be the source hiding behind this phenomenon? In this talk we give two possible sources, namely, non-negligible species-specific death rate and delay in growth. We show how each source can give rise mathematically to transient oscillations.

This talk is based on joint work with G. S. K. Wolkowicz and H. Xia.

**JAMES WATMOUGH**, University of New Brunswick, Fredericton, NB
*Disease transmission at home and abroad*

Most models of disease transmission make very simple assumptions about the incidence of infection. In differential equation models these are usually bilinear and assume a well-mixed population. Many models have been proposed to study heterogeneities arising from age structure, behavioural groups, stages of infection and spatial variation. More recently, network models, in various forms, have been used to model heterogeneities in the transmission setting. For example, transmission may occur in a household, a hospital, a workplace, or on a transit system. In this talk I present a simple ordinary differential equation model for disease transmission with multiple groups and multiple settings and formulate conditions for the spread of the disease through a population. The assumptions lead to a model that has no explicit spatial variable, yet still account for spatial variation through the various transmission settings.

**JIANHONG WU**, York University
*Modeling Eradicating Vector-borne Diseases via Structured Culling*

We derive appropriate mathematical models to assess the effectiveness of culling as a tool to eradicate vector-borne diseases. The model, focused on the culling strategies determined by the stages during the development of the vector, becomes either a system of autonomous delay differential equations with impulses (in the case where the adult vector is subject to culling) or a system of non-autonomous delay differential equations where the time-varying coefficients are determined by the culling times and rates (in the case where only the immature vector is subject to culling). Sufficient conditions are derived to ensure eradication of the disease, and simulations are provided to compare the effectiveness of larvicides and insecticide sprays for the control of West Nile virus. We show that eradication of vector-borne diseases is possible by culling the vector at either the immature or the mature phase, even though the size of the vector is oscillating and above a certain level.

This is a joint work with S. A. Gourley and R. S. Liu.

**XINGFU ZOU**, University of Western Ontario
*Existence and global attractivity of posititve periodic solution in Lotka–Volterra competition systems with deviating argument*

In this talk, we consider the periodic Lotka–Volterra competition systems with deviating arguments. We present some sufficient conditions, and sufficent and neccessary conditions for a special case, for existence of a positive periodic solution. We also establish some 3/2 type criteria for the global attractivity of the positive periodic solution.

This is joint work with Xianhua Tang.