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**Mathematical Biology**  
**Biologie mathématique**  
(Org: **Gail Wolkowicz** (McMaster))

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**JULIEN ARINO**, University of Manitoba, Winnipeg, Manitoba  
*Effect of the introduction of refractory vectors in a vector-borne disease*

A model for a vector-borne disease is considered, in which some vectors are refractory to infection by the pathogen. The model describes two populations of vectors: wild vectors can be infected by the pathogen, while refractory vectors cannot. However, refractoriness comes at the cost of reduced fitness. Interbreeding between wild and refractory vectors can produce both types of vectors, as described in the model by a somewhat complicated demography. This model for vectors is then coupled to a simple SIR model for hosts, and the dynamics of the whole system is studied. We show that in the absence of disease, refractory vectors can become established in the population. When the disease is present, this has the effect of lowering the basic reproduction number, rendering the disease easier to control.

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**ELENA BRAVERMAN**, University of Calgary, 2500 University Drive NW, Calgary, AB, T2N 1N4  
*On Nicholson's Blowflies and Other Models with a Distributed Delay*

We consider the Nicholson's blowflies equation with a distributed delay

$$\dot{N}(t) = -\delta N(t) + p \int_{h(t)}^t N(s) e^{-aN(s)} d_s R(t, s), \quad t \geq 0,$$

and obtain existence, positiveness and permanence results for solutions with positive initial conditions. In the range of parameters  $p, \delta$ , where the relevant equation with a constant delay is locally asymptotically stable (see the paper by Michael Li *et al.*, we prove that the solution is globally stable, as far as  $h(t)$  tends to infinity for  $t \rightarrow \infty$ . We also consider the general equation with several distributed delays

$$\dot{x}(t) + \sum_{k=1}^m r_k(t) \int_{-\infty}^t f_k(x(s)) d_s R_k(t, s) = 0,$$

which includes equations with several delays and integrodifferential equations as special cases and obtain some additional results for this equation, like linearized oscillation theorems. The results are applied to logistic, Lasota–Ważewska and Nicholson's blowflies equations with a distributed delay.

In addition, the "Mean Value Theorem" is proved which claims that any solution of an equation with a distributed delay also satisfies the linear equation with a variable concentrated delay.

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**SUE ANN CAMPBELL**, University of Waterloo  
*Synchronization, Multistability and Clustering: how useful are predictions from phase models?*

We consider a model of a network of hippocampal interneurons based on the work of Wang and Buzsáki. We construct a phase model representation of the network, and show that this model can give reasonably accurate quantitative information, such as the size of basins of attraction and the maximum heterogeneity permissible in the inherent frequencies of the neurons before synchrony is lost. We show that predictions of existence and stability of the synchronous solution from a two cell network carry over to  $N$ -cell networks, either exactly or in the limit of large  $N$ .

This is joint work with Jeff Chadwick.

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**YUMING CHEN**, Wilfrid Laurier University

*Global Attractivity of a Positive Periodic Solution of a Delayed Periodic Respiration Model*

In this talk we consider a delayed periodic respiration model. First, using the method of coincidence degree, we establish the existence of a periodic solution. Then, we give several sufficient conditions for the global attractivity of this periodic solution.

This is a joint work with L. Huang.

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**TROY DAY**, Queen's University, Jeffery Hall, Kingston, ON, K7L 3N6, Canada

*Modeling the emergence and control of infectious diseases*

Mathematical models are increasingly being used to understand and predict the spread of infectious diseases. Often deterministic models suffice for tracking the dynamics of well-established diseases, but the initial stages in the emergence of new infectious diseases are often marked by considerable stochasticity. This stochasticity enters in at least two important ways. First, many new diseases arise from cross-species transmission, and some degree of evolutionary adaptation is often required before the pathogen can spread. Stochasticity in the evolutionary process in terms of which mutations arise and reach fixation plays a key role is whether or not such diseases take hold. Second, once a pathogen has adapted and is beginning to spread, medical interventions (e.g., quarantine) will typically be imposed. The relatively small initial number of cases during this phase also means, however, that stochasticity will play an important role in the extent to which such interventions are effective. I will present some recent modeling results treating both of these phenomena, with reference to the emergence of pandemic influenza as well as the 2003 outbreak of SARS.

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**HERB FREEDMAN**, University of Alberta, Department of Mathematical & Statistical Sciences, Edmonton, AB, T6G 2G1

*Mathematical Models of Cancer Treatment Using Competition*

This talk contains a brief survey of modelling the growth and treatment of cancer using the competition paradigm for interaction with normal cells. The models consist of systems of ordinary differential equations. Treatment techniques include radiotherapy, chemotherapy and immunotherapy.

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**MICHAEL LI**, University of Alberta, Department of Mathematical and Statistical Sciences, Edmonton, AB, T6G 2G1

*Global Stability in Multigroup Epidemic Models*

For a class of  $n$ -group epidemic models of SEIR type, the uniqueness and global stability of the endemic equilibrium, when the basic reproduction number is greater than 1, is proved using a global Lyapunov function.

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**XINZHI LIU**, University of Waterloo, Department of Applied Mathematics, Waterloo, Ontario, N2L 3G1, Canada

*Study on HIV transmission among intravenous drug users*

The intravenous-drug-using community has been strongly affected by the HIV virus, and can further contribute to the transmission of AIDS in the non-drug-using community. It has also been indicated that IV-drug users provide a major link between the heterosexual and the homosexual population, thus affecting HIV prevalence in many different population classes. As a result, methods to reduce or remove HIV infections among IV-drug users need to be considered. In this talk, a model for HIV transmission among intravenous drug users is studied. Several control methods are introduced and analyzed. In each case, a stability analysis of the disease-free equilibrium point may provide an estimation of the effectiveness of the control method.

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**CONNELL MCCLUSKEY**, Wilfrid Laurier University  
*A Study of Mimicry in Poison Arrow Frogs*

In the Ecuadorean rainforest, there is an interesting case of mimicry among three species of frogs; one species (*E. parvulus*) is quite poisonous, one is slightly poisonous (*E. bilineatus*), and the other is non-poisonous (*A. zaparo*). The two poisonous species look similar (though not identical), and have overlapping habitats. Throughout the same geographical region, the non-poisonous frog species mimics the poisonous frogs, displaying colouring that is practically indistinguishable from whichever poisonous species happens to occupy the region. In the overlap zone, where both poisonous species are present, the non-poisonous species accurately mimics only one of the poisonous species (and therefore is an imperfect mimic for the other poisonous species).

One would expect the mimic to copy either the more abundant or more noxious of the two poisonous frogs. In this case, the more abundant poisonous frog is also the more noxious. Surprisingly, the mimic copies the less frequent and less poisonous species. It was previously hypothesized that the mechanism driving this unexpected result is differential generalization of learning among the predators. In particular, the more intensely negative the experience of capturing a poisonous frog, the more likely a predator is to avoid not just that species of frog, but also frogs that look similar. We have modelled this species interaction using a predator-prey system of ordinary differential equations, varying the degree to which the predators generalize their learned avoidance to include the imperfect mimics.

This is joint work with Jeff Orchard at the University of Waterloo.

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**STEPHANIE PORTET**, University of Manitoba, Winnipeg, Manitoba  
*Dynamics of in vivo intermediate filament organization*

The cytoskeleton is a complex arrangement of structural proteins organized in networks: microfilaments, intermediate filaments and microtubules. Each network has specific properties and organization as well as particular roles in the cell. The organization of a cytoskeletal network is the main determinant of its cellular function.

In this work, the organization of the intermediate filament network is studied. The model describes the dynamics of four structural states of the intermediate filament material: soluble proteins, particles (precursors of filaments), and short and long filaments. Assembly processes are considered, taking into account the formation and growth of particles, the elongation of particles into short filaments, and the integration and solubilization of filaments. Different hypotheses are tested by mathematical and numerical analysis.

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**ROBERT SMITH**, University of Ottawa, 585 King Edward, Ottawa, ON

*Predicting the potential impact of a cytotoxic T-lymphocyte HIV vaccine: how often should you vaccinate and how strong should the vaccine be?*

To stimulate the immune system's natural defences, a HIV vaccination program consisting of regular boosts of cytotoxic T-lymphocytes (CTLs) has been proposed. We develop a mathematical model to describe such a vaccination program, where the strength of the vaccine and the vaccination intervals are constant. We apply the theory of impulsive differential equations to show that the model has an orbitally asymptotically stable periodic orbit. We show that, on this orbit, it is possible to determine vaccine strength and vaccination intervals so that the number of infected CD4<sup>+</sup> T cells remains below a maximal threshold. We also show that the outcome is more sensitive to changes in the vaccine strength than the vaccination interval and illustrate the results with numerical simulations.

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**JAMES WATMOUGH**, University of New Brunswick  
*The final size of an epidemic*

The early disease transmission model of Kermack and McKendrick established two main results that are still at the core of most disease transmission models today: the basic reproduction number,  $\mathcal{R}_o$ , as a threshold for disease spread in a population; and the final size of an epidemic. As models become more complex, the relationship between disease spread, final size and  $\mathcal{R}_o$  are not as clear; yet  $\mathcal{R}_o$  remains the main object of study when comparing control measures.

In this talk I review the final size relation for a simple epidemic model and discuss its form in more complex models for treatment and control of influenza and HIV.

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**JIANHONG WU**, York

*Progress in modeling pandemic influenza and bird flu*

We summarize our recent progress in modeling pandemic influenza and bird flu, using a variety of deterministic dynamical models. We show how model analysis and simulations are useful to evaluate different control strategies and to understand the mechanisms for the current spread patterns of the H5N1 avian flu.

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**HUAIPING ZHU**, York

*Stability and Oscillations for SIR Epidemiological Models*

SIR compartmental epidemiological models have been widely used to study the transmission dynamics of certain infectious diseases. In this talk, I shall discuss the stability and oscillations of such three dimensional models with a general nonlinear incidence function.

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**XINGFU ZOU**, University of Western Ontario

*Can the cut-burn strategy eradicate a wood-boring beetle infestation?*

We propose a mathematical model for an infestation of a wooded area by a beetle species in which the larva develop deep in the wood of living trees. Due to the difficulties of detection, we presume that only a certain proportion of infested trees will be detected and that detection, if it happens, will occur only after some delay which could be long. An infested tree once detected is immediately cut down and burned. The model is stage structured and contains a second time delay, the development time of the beetle from egg to adult. There is a delicate interplay between the two time delays due to the possibility in one case for a larva to mature even in a tree destined for destruction. We present conditions sufficient for infestation eradication and discuss the significance of the conditions particularly in terms of the proportion of infested trees that need to be detected and removed. If the infestation is successfully eradicated there are always a number of trees that completely escape infestation and we compute lower bounds and an approximation for this number. Finally, we present the results of some numerical simulations.

This is a joint work with Stephen Gourley.