Main determinants of human influenza phylodynamics

Sébastien Ballesteros, A. Camacho, Elisabeta Vergu, Bernard Cazelles

CMPD3

Preface

The Third Conference on Computational and Mathematical Population Dynamics (CMPD3) will take place in Bordeaux, France, from May 31 to June 4, 2010. This Conference is the third joint meeting of the Conference on Mathematical Population Dynamics (MPD) and the Conference on Deterministic and Stochastic Models for Biological Interactions (DeStoBio), with a 25-year history of international meetings.

The aim of the meeting is to bring together people from different fields (applied mathematicians, computer scientists, biologists, clinicians, epidemiologists, ecologists, ...) interested in (deterministic and/or stochastic) models for population dynamics and interactions.

Population dynamics here is intended in a very wide sense, including everything from populations of animals and plants, to populations of cells or molecules. Hence, topics of the conference will include ecology (including epidemic spread), cell population dynamics (including immunology, tumor growth, neurosciences) and molecular biology (including molecular evolution and genetics, genomics). The meeting will focus on modelling of quantitative data in these fields, analysis of models, and their applications.

CMPD 3 Organizing Committee

CMPD 3

May 31 - June 4, 2010
Université de Bordeaux 2, France

Scientific Committee

- Bedr'Eddine Ainseba, Bordeaux University
- Linda Allen, Texas Tech University
- Julien Arino, Manitoba University
- Odo Diekmann, Utrecht University
- Lou Gross, Tennessee University
- J.A.P Heesterbeek, Utrecht University
- Mimmo Iannelli, Trente University
- Yoh Iwasa, Kyushu University
- Peter Jagers, Göteborg University
- Denise Kirschner, Michigan University
- Michel Langlais, Bordeaux University
- Mark Lewis, Alberta University
- Zhien Ma, Xi'An Jiaotong University
- Maia Martcheva, Florida University
- Shigui Ruan, University of Miami
- Hyun M. Yang, Campinas University
- Jianhong Wu, York University

Organizing Committee

- Bedr'Eddine Ainseba, Bordeaux University, IMB, INRIA Anubis, President
- Jean-Baptiste Burie, Bordeaux University, IMB, INRIA Anubis
- Jacques Henry, Bordeaux University, INRIA Anubis
- Pierrick Legrand, Bordeaux University, IMB, INRIA ALEA
- Pierre Magal, Bordeaux University, IMB, INRIA Anubis
- Christine Nazaret, Bordeaux University, IMB
- Antoine Perasso, Bordeaux University, IMB
- Ingrid Rochel, Bordeaux University, IMB
- Shigui Ruan, University of Miami, USA

International Program Committee

- Bedr'Eddine Ainseba, Bordeaux University, France
- Linda Allen, Texas Tech University, USA
- Carlos Brauman, EVORA University, Portugal
- Odo Diekmann, Utrecht University
- Hassan Hbid, Cadi Ayyad University, Marrakech, Maroco
- Yoh Iwasa, Kyushu University, Japan
- Tanya Kostova, National Science Fundation, USA
- Michel Langlais, Bordeaux University, France
- Zhien Ma, Xi'An Jiaotong University, China
- Fabio Milner, Arizona state university, USA
- Andrea Pugliese, Trente university, Italia
- Eva Sanchez, Polytechnical university, Madrid, Spain

NUMERICAL INTEGRATION OF A HIERARCHICALLY SIZE-STRUCTURED POPULATION MODEL

L.M. Abia[b], **O. Angulo**[a] ,**J.C. López-Marcos**[b] and **M.A. López-Marcos**[b]

[a]Departamento de Matemática Aplicada
Universidad de Valladolid
C/ Fco. Mendizabal, 1, 47014 Valladolid, SPAIN
oscar@mat.uva.es

[b]Departamento de Matemática Aplicada
Universidad de Valladolid
Paseo del Prado de la Magdalena, s/n, 47005 Valladolid, SPAIN
lopezmar@mac.uva.es

We will study the numerical integration of nonlinear models which describe the dynamics of a hierarchical size-structured population. More precisely, we consider a model with contest competition which has the form

$$u_t + (g(x, B(x,t))\, u)_x = -\mu(x, B(x,t))\, u, \quad x_m < x < x_M,\ t > 0, \tag{1}$$

$$g(x_m, B(x_m,t))\, u(x_m,t) = C(t) + \int_{x_m}^{x_M} \alpha(x, B(x,t))\, u(x,t)\, dx\ , \quad t > 0, \tag{2}$$

$$u(x,0) = \phi(x), \quad x_m \le x \le x_M, \tag{3}$$

$$B(x,t) = \int_{x}^{x_M} w(\sigma)\, u(\sigma,t)\, d\sigma, \quad x_m < x < x_M, \quad t > 0. \tag{4}$$

The independent variables x and t represent, respectively, size and time, where x_m and x_M are, respectively, the minimum and maximum value reached by a given population. The function $u(x,t)$ is the population density with size x at time t. The population dynamics is determined by the growth rate g, the mortality rate μ, the reproduction rate α and the external inflow C. The vital functions (growth, mortality and reproduction rates) depend on the structuring variable and on the functional $B(x,t)$, used to describe the competition among individuals for available resources. In this case, contest competition, no individual in a class of smaller size can affect the amount of resource available to an individual of greater size. Existence and uniqueness of solutions for this model has been studied by Kraev (1)

In the present work, we carry out the numerical integration of equations (1)-(4) by means of a method that integrates along the characteristic curves and uses a constant number of grid nodes. The integral terms are approximated by means of second order quadrature rule.

Finally, we will apply it to the solution of a model that describe the dynamics of a mono-species model of size-structured tree population that takes into account the effect of competition for light (2). In this case the size is given by the diameter at breast height (d.b.h.) and $B(x,t)$ represents the cumulative basal area of trees greater in size than x and expresses the shading effect under light competition.

References

[1] Kraev, E.A., Existence and uniqueness for height structured hierarchical population models, Natural Resources Modeling **14**, 45-70 (2001).

[2] Zavala, M.A. & Bravo de la Parra, R., A mechanistic model of tree competition and facilitation for Mediterranean forests: scaling from leaf physiology to stand dynamics, Ecol. Modelling **188**, 76-92 (2005).

SEASONAL TRANSMISSION, YEAR ROUND TRANSMISSION AND THE EVOLUTION OF INFLUENZA A

B. Adams and **A. McHardy**

The evolution of human influenza A H3N2 is epochal. A cluster of antigenically similar strains will predominate world-wide for several years. During this period antigenically neutral or nearly neutral mutations accumulate until the genetic context allows the emergence of an antigenically distant strain that founds a new cluster and excludes the existing cluster [1,2]. These founder strains originate in a reservoir of viral diversity located in East-Southeast Asia. In this region temporally overlapping localized epidemics lead to year-round influenza incidence. In temperate regions, however, the incidence of influenza H3N2 is seasonal. There are pronounced epidemics in winter but little or no detectable incidence in summer. Here we use a mathematical model to examine how the evolutionary dynamics of influenza virus are affected by year-round and seasonal transmission regimes [3,4]. The core of the model is a single wild-type viral strain. If transmission is year-round this strain causes a continuous epidemic spanning several years. If transmission is seasonal it causes a series of temporally disjoint epidemics. Under both transmission regimes, the generation of diversity is highest around the middle of wild-type epidemics but mutant strains without very large antigenic advantages are most likely to cause significant outbreaks if they emerge early in these epidemics. Furthermore, in regions with seasonal transmission, short epidemics and limited exchange with other regions make the establishment of lineages that eventually produce the founders of new antigenic clusters unlikely. In regions with year-round transmission, however, drawn out epidemics and extensive exchange with other regions are more conducive to the emergence, persistence and development of such lineages. We conclude that the epidemiology of regions with year-round transmission is likely to be a key element governing the evolution of influenza virus, including the emerge nce of antigenically novel clusters.

MODELLING THE DYNAMICS OF AN EXPLOITED ATLANTIC SALMON POPULATION FOR MANAGEMENT ADVICE: A BAYESIAN HIERARCHICAL APPROACH

G. Dauphin[a,b,c], **C. Adams**[b], **P. Boylan**[c] and **E. Prévost**[a]

Keywords: fisheries, hierarchical Bayesian model, population dynamics, uncertainty

[a] UMR ECOBIOP
INRA
Station d'hydrobiologie, Quartier Ibarron, 64310 Saint Pée sur Nivelle, France
guillaume.dauphin@gmail.com
eprevost@st-pee.inra.fr

[b] Scottish Centre for Ecology & the Natural Environment
Glasgow University, Loch Lomond
Rowardennan, Glasgow G63 0AW, Scotland
c.adams@bio.gla.ac.uk

[c] Loughs Agency
Victoria Road, Londonderry, BT47 2AB, Londonderry Northern Ireland
p.boylan@loughs-agency.org

When population dynamics modeling aims at providing management advice, explicit consideration of both available information and uncertainty is mandatory. hierarchical Bayesian modeling (HBM) provides a framework for simultaneously (i) incorporating available knowledge about populations dynamics processes, (ii) assimilate data series heterogeneous in quality and time coverage, and (iii) accounting for diverse sources of uncertainty. We illustrate the interest of HBM on a case study dealing with Atlantic salmon in the Foyle catchment (Ireland). This study was motivated by the decline of the salmon commercial fisheries during the last four decades. It aimed at estimating the evolution of actual stock size, and at assessing the relative role of exploitation vs. environment in this evolution. A stage-structured model describing the salmon life cycle was designed for representing the population dynamics. It includes density dependence regulation, some form of spatial structure and auto-correlated environmental stochasticity. Available datasets informative about the various life stages did not cover the same time periods and were more or less reliable depending on the data collection procedure. They include various fisheries catch, electronic counter data, spawner and juvenile indexes of abundance. HBM coupled with Markov chain Monte Carlo sampling techniques allowed to make direct probabilistic statements about the various type of unknowns, i.e. models parameters, missing data and unobservable latent state variables.

A SVEIR model with Imperfect Vaccine

P. Adda[a] and **L. Nkague Nkamba**[a,b] and **G. Sallet**[a,b] and **L. Castelli**[c]

[a]EPI MASAIE
INRIA Nancy Grand Est
Université de Metz, Ile du Saulcy, Metz, France

[b]UMI UMMISCO
IRD
Université Gaston Berger, Saint-Louis, Sénégal

[c]DEEI
University of Trieste
34217, Trieste, Italy
Philippe.Adda@loria.fr ; nkague1@yahoo.fr ; sallet@loria.fr ; castelli@units.it

corresponding author : Gauthier Sallet, EPI MASAIE, INRIA, bat A ISGMP, université de Metz, Ile du Saulcy, 57045, Metz-Cedex, France
sallet@loria.fr

We consider a SVEIR model with standard mass action and differential death rates for each compartment, susceptible individuals S, vaccinated V, exposed E, infectious I and permanently removed R. The susceptible population is increased by the net in-flow (recruitment) of individuals into the region, either by birth or immigration. We assume that a proportion α of newborn are vaccinated at birth, the remainder enter the S compartment. Each subpopulation is decreased by death, the death rate being specific to each compartment. Susceptible individuals (regardless of whether they have been previously vaccinated) are further vaccinated at the rate p.

$$\begin{cases} \dot{S} &= (1-\alpha)\,\Lambda - (d_S + p)\,S - \beta\,S\,I \\ \dot{V} &= \alpha\,\Lambda + p\,S - d_V\,V - \theta\,\beta\,V\,I \\ \dot{E} &= \beta\,(S + \theta\,V) - (d_E + \varepsilon)\,E \\ \dot{I} &= \varepsilon\,E - (d_I + \gamma)\,I \\ \dot{R} &= \gamma\,I - d_D\,R \end{cases}$$

We compute the basic reproduction ratio $\mathcal{R}_0$ of this model. We prove that if $\mathcal{R}_0 \leq 1$ the disease free equilibrium is globally stable on $\mathbb{R}^5_+$. If $\mathcal{R}_0 > 1$ we prove that there exists an unique endemic equilibrium in the positive orthant, which is globally asymptotically stable on the positive orthant.
This model generalizes a previous model studied in [1]. In this reference, the model is only with an adult vaccination and only a disease induced death is considered. The proof in [1] used the Li-Muldowney techniques to ensure the global asymptotic stability of the endemic equilibrium.
We give a simple proof using Lyapunov-Lasalle techniques.

References

[1] A.B. Gumel, and C C. McCluskey and J. Watmough, "An SVEIR model for assessing potential impact of an imperfect anti-SARS vaccine", *Math. Biosci. Eng.*, vol 3 (3) p. 485-512, 2006.

PERSPECTIVES ON THE CURRENT SUCCESSES AND CHALLENGES OF CANCER GROWTH AND TREATMENT

E. Afenya

Department of Mathematics
Elmhurst College
190 Prospect Avenue, Elmhurst, IL 60126
evansa@elmhurst.edu

Varying approaches have been adopted over the years in understanding cancer growth and a number of advances have been made in cancer prevention and treatment. However, the level of recorded progress in attempts being made to finding lasting cure to cancers of various forms still indicates that great challenges lie ahead on all fronts for biomedicine. Subsequently, the notable successes that have been achieved and the recent challenges are highlighted and placed in a contextual framework. As a consequence, a look is taken at the massive process of normal hematopoiesis through the introduction of candidate models that throw light on this phenomenon. The onset and development of the cancers that arise largely as a result of hematopoietic malfunction is then studied through the use of various models that describe malignant cell behavior. In the process of doing this the models are used to investigate the plausibility of the current re-emerging views on the "cancer stem cell" hypothesis. Following this, various modeling viewpoints about advantages and disadvantages of current and emerging treatment strategies are introduced and discussed and arguments are made in favor of looking at cancer treatment as a formal problem of optimization in the clinic.

PREDATOR PREY DYNAMICS IN A UNIFORM MEDIUM LEAD TO DIRECTED PERCOLATION AND WAVE TRAIN PROPAGATION

A. Agranovich and **Y. Louzoun**

We here study a spatially extended stochastic predator-prey model in the context of the immune system versus pathogen dynamics. We analyze the fluctuations near the zero pathogen unstable fixed point of a system with a constant source of immune cells. We show that when diffusion between different regions is incorporated, a directed percolation regime emerges in a limited range of diffusion rates. In other parts of the phase space the dynamics converge to the mean field oscillatory solution, or to propagating wave trains. Interestingly, for both high and low diffusion values, the system collapses to the absorbing state, and only in the intermediate diffusion rate, a long term survival is observed.

We compute the pathogen (prey) survival probability following a weak perturbation of the non-stable zero fixed point. We show that when this probability is low, the loss of synchronization leads to two possible dynamic regimes. The first regime is a directed percolation regime based on the balance between regions leaving the zero fixed point and regions absorbed into it. The second regime is wave trains representing the transition of all space from the unstable to the stable positive fixed point. These two regimes differ from the mean-field results and do not rely on a low number of either pathogens or immune cells. These regimes can explain similar dynamics observed in biological situations. This work creates a direct bridge between the predator prey dynamics, directed percolation and train waves.

EPIDEMIOLOGY OF DENGUE FEVER: A MODEL WITH TEMPORARY CROSS-IMMUNITY AND POSSIBLE SECONDARY INFECTION SHOWS BIFURCATIONS AND CHAOTIC BEHAVIOUR IN WIDE PARAMETER REGIONS

Maíra Aguiar[a], **Bob Kooi**[b] and **Nico Stollenwerk**[a]

[a]Centro de Matemática e Aplicações Fundamentais da Universidade de Lisboa,
Avenida Prof. Gama Pinto 2,
1649-003 Lisboa, Portugal,
maira@ptmat.fc.ul.pt and nico@ptmat.fc.ul.pt

[b]Faculty of Earth and Life Sciences, Department of Theoretical Biology,
Vrije Universiteit Amsterdam,
De Boelelaan 1087, NL 1081 HV Amsterdam, The Netherlands,
bob.kooi@falw.vu.nl

Basic models suitable to explain the epidemiology of dengue fever have previously shown the possibility of deterministically chaotic attractors, which might explain the observed fluctuations found in empiric outbreak data. However, the region of bifurcations and chaos require strong enhanced infectivity on secondary infection, motivated by experimental findings of antibody-dependent-enhancement. Including temporary cross-immunity in such models, which is common knowledge among field researchers in dengue, we find bifurcations up to chaotic attractors in much wider and also unexpected parameter regions of reduced infectivity on secondary infection, realistically describing more likely hospitalization on secondary infection when the viral load becomes high. The model shows Hopf bifurcations, symmetry braking bifurcations of limit cycles, coexisting isolas, and two different possible routes to chaos, via the Feigenbaum period doubling and via torus bifurcations.

References

[1] Aguiar, M., Kooi, B., & Stollenwerk, N. (2008) Epidemiology of dengue fever: A model with temporary cross-immunity and possible secondary infection shows bifurcations and chaotic behaviour in wide parameter regions, *Math. Model. Nat. Phenom.* **3**, 48–70.

[2] Aguiar, M., Stollenwerk, N., & Kooi, B. (2009) Torus bifurcations, isolas and chaotic attractors in a simple dengue fever model with ADE and temporary cross immunity, *Intern. Journal of Computer Mathematics* **86**, 1867–77.

[3] N. Stollenwerk, M. Aguiar and B. W. Kooi "Computational aspects in the investigation of chaotic multi-strain dengue models" *Proceedings of 9th Conference on Computational and Mathematical Methods in Science and Engineering, CMMSE 2009*, ISBN 978-84-612-9727-6, edited by Jesus Vigo Aguiar *et al.*, Salamanca, 2009, pp. 995–1002.

[4] Maíra Aguiar, Bob W. Kooi, Nico Stollenwerk (2009) Multi-strain deterministic chaos in dengue epidemiology, a challenge for computational mathematics, Proceedings of the International Conference on Numerical Analysis and Applied Mathematics, ICNAAM 2009, Simos, Theodore E.; Psihoyios, George; Tsitouras, Ch., (eds.) AIP Conference Proceedings 1168, 1555-1558.

THE EFFECT OF LANDSCAPE DYNAMICS ON ECOLOGICAL SPECIATION

R. Aguilée[a] and **A. Lambert**[b] and **D. Claessen**[a]

[a]Laboratoire Ecologie & Evolution
UPMC Univ Paris 06 - Ecole Normale Supérieure - CNRS
UMR 7625, Ecole Normale Supérieure, 46 rue d'Ulm, F-75230 Paris Cedex 05, France
robin.aguilee@ens.fr
david.claessen@ens.fr

[b]Laboratoire Probabilités & Modèles Aléatoires
UPMC Univ Paris 06
UMR 7599, UPMC, 4 place Jussieu, F-75252 Paris Cedex 05, France
amaury.lambert@upmc.fr

Ecological speciation is usually considered in a fixed geographical background, allopatry or sympatry. However, populations are often subject to landscape changes at different spatiotemporal scales due to geological, climatic and ecological processes, resulting in repeated divisions and reconnections of populations. We examine under which conditions such landscape dynamics allow the formation of diversity and its maintenance until successful speciation. We use a stochastic, sexual ecological model embedded into a simple landscape dynamics model (allopatry-sympatry oscillations). We show that landscape dynamics generate diversity easier than in a fixed geographic background, but that its maintenance until complete, persistent speciation is uncertain. Under stabilizing selection, speciation occurs in allopatry but coexistence of incipient species is evolutionarily unsustainable. Permanent speciation critically depends on the characteristic time scales of the landscape dynamics. Under disruptive selection, landscape dynamics facilitate the initiation of speciation in allopatry by allowing divergence in a population stuck at a fitness minimum for e.g. genetic constraints. Repeated sympatric phases then increase the likelihood of reinforcement, completing speciation. Our results stress the importance to take into account past, current and future geographical backgrounds: their sequence is likely to considerably alter the speciation process. We discuss empirical data revealing the effect of landscape dynamics.

Keywords: landscape dynamics; ecological speciation; allopatry; sympatry; secondary contact; reinforcement; dynamic metapopulation

Existence of Positive Almost Periodic or Ergodic Solutions for Some Neutral Nonlinear Integral Equations

E.Ait Dads*, P. Cieutat†and L. Lhachimi‡

March 13, 2010

Abstract

We state sufficient conditions for the existence for the positive almost periodic or ergodic solutions of the following neutral integral equation:

$$x(t) = \gamma x(t-\sigma) + (1-\gamma)\int_{t-\sigma}^{t} f(s, x(s))ds,$$

where $0 \leq \gamma < 1$ and $f : I\!R \times I\!R^+ \to \mathbb{R}^+$ is a continuous map. We also treat the asymptotically, weakly and pseudo almost periodic solutions. Our results do not need the monotonicity of $f(t, .)$.

As we all know, the existence of periodic solutions of functional differential equations (FDE) has great theoretical and practical significance and is one of the problems of great interest to scholars in the field. Since Yoshizawa [**?**] presented an excellent result for the existence of periodic solutions to FDE with bounded delay, Cooke and Huang [**?**], Burton and Hatvani [**?**] generalized Yoshizawa's result to FDE with infinite delay. We remark that, in the nature, there is no phenomenon which is purely periodic, this gives the idea to consider the almost periodic situation.

In this paper, we consider the following neutral nonlinear integral equation

$$x(t) = \gamma x(t-\sigma) + (1-\gamma)\int_{t-\sigma}^{t} f(s, x(s))\ ds, \tag{1}$$

where $0 \leq \gamma < 1$, $\sigma > 0$ and $f : \mathbb{R} \times \mathbb{R}^+ \to \mathbb{R}^+$ is a continuous map.

*Université Cadi Ayyad, Département de Mathématiques, Faculté des Sciences, B.P. 2390 Marrakech, Morocco. E-mail: aitdads@ucam.ac.ma

†Laboratoire de Mathématiques de Versailles, Université de Versailles Saint-Quentin en Yvelines, 45 Avenue des Etats-Unis, 78035 Versailles Cedex, France. E-mail: Philippe.Cieutat@math.uvsq.fr

‡Université Cadi Ayyad, Département de Mathématiques, Faculté des Sciences, B.P. 2390 Marrakech, Morocco. E-mail: lhachimi@voila.fr

ESS VERSUS COOPERATIVE BEHAVIOUR IN A CONSUMER-RESOURCE MODEL

A.R. Akhmetzhanov[a], **F. Grognard**[a], **L. Mailleret**[b] and **P. Bernhard**[a]

[a]Project COMORE
INRIA Sophia Antipolis–Méditerranée
F-06902 Sophia Antipolis, France
(Andrei.Akhmetzhanov, Frederic.Grognard, Pierre.Bernhard)@sophia.inria.fr

[b]URIH, UR880
INRA Sophia Antipolis
F-06903 Sophia Antipolis, France
Ludovic.Mailleret@sophia.inra.fr

Keywords: ESS, optimality, life-history trait, seasonality

In this work we consider a system of two interacting populations of consumers and resources. It is assumed that the two populations interact during a season of length T and die at the end of it. Reproductive processes occurring during the season determine the size of the populations at the beginning of the next season, what corresponds to a 'semi-discrete' model [1,2].
We assume that the resource population behaves passively and the consumers have some control on their reproduction: they must choose between investing their time in foraging the resources, what increases their reproductive capacities, or in reproducing. Assuming that consumers agree to cooperate, we first study the maximization of the consumers fitness (strictly speaking their number of descendants in the next generation) as a dynamic problem of energy allocation and investment [3]. We show that such an 'optimal' strategy is not evolutionarily stable (ESS) as a population using such a strategy might be invaded by some mutants that take advantage of their small density and non-cooperative behaviour.
We then look for an ESS, using the first condition for ESS (or "Nash condition" or rather "Wardrop condition" [4,5]) for the consumers population which does not allow any mutant to invade. We show how such a strategy can be obtained as the solution of a zero-sum differential game.
Moreover, we compare these two reproductions strategies in terms of their influence on the dynamics of the system through many seasons. Though not ESS, the optimal strategy leads to a stable situation in a long-term perspective. On the contrary, the prevention strategy is ESS but it is not long-term stable and leads to an instance of the so-called "Tragedy of the Commons".

References

[1] E. Pachepsky, R.M. Nisbet, and W.W. Murdoch, "Between discrete and continuous consumer-resource dynamics with synchronized reproduction", *Ecology*, 89(1), pp. 280-288, 2008.
[2] L. Mailleret and V. Lemesle, "A note on semi discrete modelling in the life sciences", *Philosophical Transactions of the Royal Society A*, 367, pp. 4779-4799, 2009.
[3] N. Perrin, and R. M. Sibly, 1993. Dynamic-models of energy allocation and investment. *Annual Review of Ecology and Systematics* 24, 379-410.
[4] J. Weibull, "Evolutionary Game Theory" Cambridge, MA: The M.I.T. Press, 1995.
[5] P. Bernhard. ESS, population games, replicator dynamics: dynamics and games if not dynamic games. *International Symposium on the Theory and Applications of Dynamic Games: Proceedings*, n. 13, 2008.

SHARP INTERFACE LIMIT OF THE FISHER-KPP EQUATION

M. Alfaro[a] and **A. Ducrot**[b]

[a]I3M, Université de Montpellier 2,
CC051, Place Eugène Bataillon,
34095 Montpellier Cedex 5, France.
malfaro@math.univ-montp2.fr

[b]UMR CNRS 5251 IMB and INRIA Sud-Ouest ANUBIS,
Université de Bordeaux,
3, Place de la Victoire,
33000 Bordeaux France.

Reaction diffusion equations with logistic nonlinearity were introduced in the pioneer works of Fisher or Kolmogorov, Petrovsky and Piskunov. These equations, which read as

$$\partial_t u(t,x) = \Delta u(t,x) + u(t,x)(1-u(t,x))\,, \quad t>0\,, \quad x\in\mathbb{R}^N\,, \tag{1}$$

are widely used in the literature to model phenomena arising in population genetics or in biological invasions. The main properties of such equations is to admit (biologically relevant) travelling wave solutions with some semi-infinite interval of admissible wave speed. The aim of this work is to focus on the ability of equation (1) to generate some interfaces and to propagate them. In order to observe such a property, a rescaling argument yields the singular limit problem

$$(P^\varepsilon) \quad \begin{cases} \partial_t u = \varepsilon\Delta u + \dfrac{1}{\varepsilon}u(1-u) & \text{in } (0,\infty)\times\mathbb{R}^N \\ u(0,x) = u_{0,\varepsilon}(x) & \text{in } \mathbb{R}^N\,, \end{cases}$$

as the parameter $\varepsilon>0$, related to the thickness of a diffuse interfacial layer, tends to zero. We consider initial data with compact support plus, possibly, perturbations very small as $\|x\|\to\infty$.

The question of the convergence of Problem (P^ε) has been addressed by Freidlin using probabilistic methods and later by Evans and Souganidis using Hamilton Jacobi technics. First, we provide a new proof of convergence by using specific reaction-diffusion tools such as the comparison principle. By proving both generation and motion of interface properties, we show that the sharp interface limit moves by a constant speed, which is the minimal speed of some related one-dimensional travelling waves. Secondly, we provide an $\mathcal{O}(\varepsilon|\ln\varepsilon|)$ estimate of the thickness of the transition layers of the solutions u^ε to (P^ε). Last, we also exhibit initial data "not so small"at infinity which do not allow the interface phenomena.

Key Words: population dynamics, Fisher equation, singular perturbation, generation of interface, motion of interface.

NUMERICAL DYNAMICS OF NONLINEAR OPEN MARINE POPULATIONS MODELS

O. Angulo[a], **J. C. López-Marcos**[b], **M. A. López-Marcos**[b] and **J. Martínez-Rodríguez**[c]

[a]Departamento de Matemática Aplicada
Escuela Universitaria Politécnica, Universidad de Valladolid
C/ Francisco Mendizábal 1, 47014 Valladolid, Spain
oscar@mat.uva.es

[b]Departamento de Matemática Aplicada
Facultad de Ciencias, Universidad de Valladolid
Paseo del Prado de la Magdalena s/n, 47005 Valladolid, Spain
lopezmar@mac.uva.es, malm@mac.uva.es

[c]Departamento de Economía Aplicada
Facultad de Ciencias Económicas y Empresariales, Universidad de Valladolid
Avenida del Valle de Esgueva 6, 47011 Valladolid, Spain
julia@eco.uva.es

We are interested in the dynamics of a population of marine invertebrates that have sessile adults and pelagic larvae contained in a local area. The population system is essentially open, and space is the obvious and principal limiting resource. The starting point for our study is the linear age-structured population model introduced in [1], and theoretically investigated in [2] and [3]. To simulate this problem, we introduced in [4] a numerical method based on the integration along the characteristic lines that provided a useful tool to investigate the asymptotic behavior of the solutions. In this work we propose different nonlinear alternatives to the model and we test them by using suitable numerical procedures as in [4]. A numerical analysis of the dynamics is carried out.

References

[1] J. Roughgarden, Y. Iwasa and C. Baxter, "Demographic Theory for an Open Marine Population with Space-Limited Recruitment", *Ecology*, v. 66, p. 54-67, 1985.
[2] S. Zhang, H.I. Freedman and X.Z. Liu, "Analysis of a Population Model with Space-dependent Recruitment in Continuous Time", *J. Math. Anal. Appl.*, v. 232, p. 99-118, 1999.
[3] H. Inaba, "Nonlinear dynamics of open marine population with space-limited recruitment: The case of mortality control", *J. Math. Anal. Appl.*, v. 275, p. 537-556, 2002.
[4] O. Angulo, J.C. López-Marcos, M.A. López-Marcos and J. Martínez-Rodríguez, "Numerical analysis of an open marine population model with spaced-limited recruitment", *Math. Comput. Model.*, DOI: 10.1016/j.mcm.2010.02.020.

EVOLUTION OF THE MOSQUITO POPULATION DURING A DENGUE EPIDEMIC IN HAVANA

H. de Arazoza[a], **R. Lounes**[b], **J. Barrios**[a], **E. Garcia**[c], **M. Atencia**[c] and **G. Joya**[c]

[a]Department of Mathematics
University of Havana
San Lazaro y L, Ciudad de La Habana, Cuba
arazoza@matcom.uh.cu ; asp@matcom.uh.cu

[b]Laboratoire MAP5, UMR-CNRS 8145
University Paris Descartes
45 rue des Saints-Pères, 75270 Paris CEDEX 06, France
lounes@math-info.univ-paris5.fr

[c]Department of Tecnología Electrónica,
University of Málaga
Campus de Teatinos, 29071 Málaga, Spain
gjoya@uma.es

Keywords:Dengue epidemic, epidemic control, deterministic model.

ABSTRACT:
Dengue fever is a disease transmitted through mosquitoes. In Cuba the transmission vector is the Aedes Aegipty mosquito. Dengue fever is endemic in many areas in Central America and the Caribbean, but in Cuba it is always an imported disease, it is not endemic. One of the problems confronted by the Health System is finding the size of the mosquito population during an outbreak of dengue fever. There are indices studied by the surveillance system, and there are indicators that point to a possible outbreak, if a case is introduced by some traveler that is unaware of its condition as a carrier of one of the dengue viruses.
Since the first papers [1] modeling dengue fever has been a priority in the countries where it is endemic.In our work using a mathematical model with ODE adapted from [2], we fit the epidemic curve given by the model to the known cases of a dengue fever outbreak in the city of Havana, using as parameters the carrying capacity for mosquito eggs, larvae and pupa. This carrying capacity depends on the actions the Health System takes to eliminate areas where the mosquito can breed.
Once we have the parameters that fit the part of the full blown outbreak, we use these values to estimate the efficiency of the system in eliminating the mosquito and its breeding grounds. We present these results using the same epidemic outbreak in the city of Havana.

References

[1] L. Esteva and C. Vargas."A model for dengue disease with variable human population". J. Math. Biol. 1999. 38: 220-240.

[2] F.A.B. Coutinho, M.N. Burattini, L.F. Lopez and E. Massad. "Threshold conditions for a non-autonomous epidemic system describing the population dynamics of dengue". Bull. Math. Biol. (2006) 68 : 2263-2282.

ON THE STABILIZATION OF REACTION-DIFFUSION SYSTEMS MODELLING A CLASS OF MAN-ENVIRONMENT EPIDEMICS: A REVIEW

S. Anita and **V. Capasso**

A two-component reaction-diffusion system modelling a class of spatially structured epidemic systems is considered. The system describes the spatial spread of infectious diseases mediated by environmental pollution. A relevant problem, related to the possible eradication of the epidemic, is the so-called zero-stabilization. In a series of papers, necessary conditions and sufficient conditions of stabilizability have been obtained. It has been proved that it is possible to diminish exponentially the epidemic process, in the whole habitat, just by reducing the concentration of the pollutant in a nonempty and sufficiently large subset of the spatial domain. In order to model the possible seasonal variability of the environmental conditions, the relevant parameters need to be assumed periodic, all with the same period. Corresponding results for the time homogeneous case are presented too.

References

[1] S. Anita, V. Capasso. "A stabilizability problem for a reaction-diffusion system modelling a class of spatially structured epidemic model", *Nonlinear Anal. Real World Appl.*, 2002.

[2] S. Anita S., V.Capasso. "A stabilization strategy for a reaction-diffusion system modelling a class of spatially structured epidemic systems (think globally, act locally)", *Nonlinear Anal. Real World Appl.*, 2009.

[3] S. Anita, V. Capasso. "Stabilization for a reaction-diffusion system modelling a class of spatially structured epidemic systems The periodic case". In Advances in Dynamics and Control: Theory, Methods, and Applications (S. Sivasundaram, et al. Eds), Cambridge Scientific Publishers Ltd.: Cambridge, UK, 2009. In press.

INFECTIOUS DISEASES AND THE GLOBAL AIR TRANSPORTATION NETWORK

J. Arino[a,b]

[a]Department of Mathematics
University of Manitoba
342 Machray Hall
Winnipeg, Manitoba
Canada R3T 2N2

[b]The Bio.Diaspora Project
CRICH, St Michael's Hospital
Toronto, Ontario
Canada M5C 1N8

Julien_Arino@umanitoba.ca

I will present work carried out in the context of the Bio.Diaspora Project, an interdisciplinary team based in Toronto (Canada), concerning the spread of infectious diseases by means of the global air transportation network. Several aspects will be addressed: data collection and warehousing, human geography, network analysis and numerical simulations.

QUANTIFYING THE EFFECTIVENESS AND IN VIVO RELEVANCE OF THE CD8+ T LYMPHOCYTE IMMUNE RESPONSE

U. Kadolsky, A. MacNamara, M. Elemans, K. Seich Al Basatena and **B. Asquith**

Department of Immunology
Imperial College London
London W2 1PG
UK
b.asquith@imperial.ac.uk

There is good evidence that the CD8+ T lymphocyte immune response plays a role in both human immunodeficiency virus (HIV) and human T cell leukemia virus (HTLV) infection.

However, the effectiveness, in vivo relevance and attributes of a protective CD8+ T lymphocyte response are unclear.

Here we present quantitative work aimed at addressing these fundamental questions.

AGGREGATION OF VARIABLES IN SPATIAL PREDATOR PREY MODELS

P. Auger[a], **J.C. Poggiale**[b] and **T. Nguyen Huu**[a]

[a]UMI 209 IRD UMMISCO
IRD & Univ. Pierre et Marie Curie
32 avenue Henri Varagnat
93143 Bondy Cedex
pierre.auger@ird.fr

[b]Université de la Méditerranée, UMR LMGEM 6117 CNRS
Campus de Luminy, Case 901
13288 Marseille Cedex 09
jean-christophe.poggiale@univmed.fr

We present prey-predator models in a patchy environment. The inclusion of two time scales in the dynamical system allows us the reduction of the system. The reduction of the proposed system is undertaken with the help of aggregation methods which aim at studying the relationships between a large class of complex systems, in which many variables are involved, and their corresponding reduced or aggregated systems, governed by a few variables, [1], [2]. The kind of aggregation methods that we consider is based on time scale separation methods. In order to illustrate the methods in population and community dynamics, applications of aggregation methods are presented for spatial models of predator prey dynamics, among them:

- A multisite fishery model with Fish Aggregating Devices (FAD), [3].
- A predator-prey model with fast prey (resp. predator) density dependent predator (resp. prey) movement [4].
- A predator-prey model with hawk and dove predators competing for preys at a fast time scale [5].

References

[1] P. Auger, R. Bravo de la Parra, J.C. Poggiale, E. Sanchez and L. Sanz, « Aggregation methods in dynamical systems variables and applications in population and community dynamics », Physics of Life Reviews, v. 5, p. 79-105, 2008.
[2] P. Auger, R. Bravo de la Parra, J.C. Poggiale, E. Sánchez and T. Nguyen Huu, « Aggregation of variables and applications to population dynamics ». In: P. Magal, S. Ruan (Eds.), Structured Population Models in Biology and Epidemiology, Lecture Notes in Mathematics, v. 1936, Mathematical Biosciences Subseries. Springer, Berlin, p. 209-263, 2008.
[3] P. Auger, C. Lett, A. Moussaoui and S. Pioch. « Optimal number of sites in artificial pelagic multi-site fisheries ». Canadian Journal of Fisheries and Aquatic Sciences. v. 67, p. 296-303, 2010.
[4] A. Elbabdlaoui, P. Auger, R. Bravo de la Parra, B. Kooi and R. Mchich. « Effects of density-dependent migrations on stability of a two-patch predator-prey model ». Mathematical Biosciences. v. 210(1), p. 335-354, 2007.
[5] P. Auger, B. Kooi, R. Bravo de la Parra and J.-C. Poggiale. « Bifurcation Analysis of a Predator-prey Model with Predators using Hawk and Dove Tactics ». Journal of Theoretical Biology. v. 238 : p. 597-607, 2006.

ENRICHMENT PARADOX INDUCED BY SPATIAL HETEROGENEITY IN A PHYTOPLANKTON - ZOOPLANKTON SYSTEM

J. -C. Poggiale[a], **M. Gauduchon**[a] and **P. Auger**[b]

[a]Centre d'Oceanologie de Marseille
Universite de la Mediterranee - UMR CNRS 6117
Case 901 - Campus de Luminy - 13288 Marseille Cedex - France
jean-christophe.poggiale@univmed.fr
mathias.gauduchon@univmed.fr

[b]UMI 209 UMMISCO
Institut de Recherche pour le Developpement
32 Avenue Henri Varagnat - 93143 Bondy Cedex - France
Pierre.Auger@bondy.ird.fr

This talk deals with the study of a predator - prey model in a patchy environment. The model represents the interactions between phytoplankton and zooplankton in the column water. Two patches are considered with respect to light availability: one patch is associated to the surface layer, the second patch describes the bottom layer. We show that this spatial heterogeneity may destabilize the predator - prey system, even in oligotrophic system where the nutrient is low enough to avoid "paradox-enrichment" phenomenon. Indeed, in this case, an heterogeneity index can be used as a bifurcation parameter, leading to a Hopf bifurcation. Moreover, we assume that individuals can be dispersed in both patches via hydrodynamism processes, like in a mixed layer. The effect of mixing intensity is analyzed as well as interactions between dispersion and enrichment. We also show that, in some cases, spatial heterogeneity has a stabilizing effect. These contradictory results are examined by considering the non linear interaction between heterogeneity, dispersal and enrichment and some mechanisms leading to stabilization/destabilization are provided.

PREVENTION DEMAND AND EPIDEMIC DYNAMICS

E. Augeraud-Véron[a] and **L. Coudeville**[b]

[a]Department of Mathematics,
University of La Rochelle,
17042 La Rochelle Cedex, France
eaugerau@univ-lr.fr

[b]Sanofi-Pasteur,
2 avenue Pont-Pasteur,
69007 Lyon, France
Laurent.Coudeville@sanofipasteur.com

The aim of this work is to model an individual vaccination behavior in response to an epidemic using expected utility criteria, and to consider how the aggregation of all these behavior modifies the dynamics of the epidemics.

Our individual behavior departs from Bauch and al. ([1]) and Poletti and al. ([2]) as we consider microeconomic foundation for the individual behavior, as in Coudeville ([3]): We will assume that the individual choice maximizes expected intertemporal utility. From this maximization, we obtain vaccination demand, depending on the future epidemic path. The impact of side effects and length of vaccine protection on vaccine demand are then analyzed.

Herd behavior will also be taking into account and we will study how the vaccination demand is modified by this externality hypothesis.

Then the dynamic of the epidemic is studied, and vaccination coverage resulting from voluntary vaccination and central planer optimal decision are compared. Agent behavior will be described under different kind of expectations (perfect foresight, myopic, bounded rationality expectations).

Aknowledgements: Research partially supported by the Hungarian National Scientific Research Fund (OTKA 62 000, 68187 and 81279) and by the Scientific and Technological Innovation Fund (of Hungary, ES-17/2008) and the Ministry of Education and Sciences (of Spain HH2008-0023).

References

[1] C.T. Bauch, and D.J.D. Earn, "Vaccination and the theory of games", *Proceedings of the National Academy of Sciences*, v. 101(36), p. 13391-13394, 2004.
[2] P. Poletti, B. Caprile, M. Ajelli, A. Pugliese, S. Merler, "Spontaneous behavioural changes in response to epidemics", *Journal of Theoretical Biology*, v. 260, p. 31-40, 2009.
[3] L. Coudeville, Comportement individuel en matière de vaccination: une appproche bayesienne, *Revue économique*, v. 55(4), p. 745-766, 2004.
[4] T. Reluga, C. Bauch, A. Galvani, "Evolving public perceptions and stability in vaccine uptake", *Mathematical Biosciences*, v. 204, p. 185-198, 2006.

MODELLING THE LETHARGIC CRAB DISEASE BY TRAVELLING WAVES

R.P. Avila[a], **C.P. Ferreira**[b] and **P.F.A. Mancera**[b]

[a]Programa de Mestrado em Biometria
Instituto de Biociências, Unesp
Cp 510, 18618970 - Botucatu – Brasil
ricpavila@ibb.unesp.br

[b]Departamento de Bioestatística
Instituto de Biociências, Unesp
Cp 510, 18618970 - Botucatu – Brasil
pio@ibb.unesp.br, pmancera@ibb.unesp.br

Keywords: Lethargic Crab Disease (LCD), Mathematical Modelling, Wave Speed, Travelling Waves.

The mangrove crab, *Ucides cordatus*, plays a crucial role in a variety of ecosystem processes in its environment, such as nutrient cycling. Moreover it is an important component in the economy of several underprivileged communities that depend on it for their subsistence. Beginning in 1997, massive mortalities of *U. cordatus* have been report by crab-collectors, such as 85% reduction in collection rates in some regions. Crabs in areas of high mortality share several common symptoms, such as lethargy, poor motor control and inability to return to the upright position when turned upside down. Hence, this pathology is called *Lethargic Crab Disease* (LCD). Finally, in 2005, there were several evidences showing that LCD is caused by a fungus of Phylum Ascomycota, *Exophiala cf psycrophila*. Basead on the epidemiology of the LCD disease, we construct a mathematical model using a system of partial differential equations, considering diffusion and advection processes, to describe the dispersion of the disease through the mangrove complexes. The non-dimensional model is given by

$$\frac{\partial S}{\partial t} = S(1-S) - S\,F - \frac{\mu_c+\mu}{\phi}\,S + I, \quad \frac{\partial I}{\partial t} = \frac{\gamma}{\phi}\,S\,F - \frac{\gamma+\alpha+\mu}{\phi}\,I, \tag{1}$$

$$\frac{\partial F}{\partial t} = \frac{\sigma\,\alpha\,\beta\,K}{\gamma\,\phi}\,S - \frac{\mu_F}{\phi}\,F - \nu_F\sqrt{\frac{1}{D_F\,\phi}}\,\frac{\partial F}{\partial x} + \frac{\partial^2 F}{\partial x^2}, \tag{2}$$

where $S(x,t), I(x,t), F(x,t)$ are, respectively, the susceptible and infected crabs populations and fungus population, ϕ is the intrinsic fecundity rate, K is the carrying capacity, μ is the natural mortality rate, μ_c is the rate of captured and commercialized crabs by pickers, β is the interaction rate between susceptible crabs population and fungus population, α is the mortality rate to the infected crabs population, γ is a rate of the crabs population which developes resistency, σ is the amount of fungus produced by infected crab and μ_F is the death rate of the fungus population, D_F is the diffusion of the fungus population and ν_F the advection. The aim of this work is to find travelling waves solutions for the developed model, that connect two equilibrium points. For this, we analyse the characteristic polynomial from the Jacobian matrix evaluated at one of the equilibrium points, the disease-free equilibrium, and then present results about the necessary conditions for the existence of travelling wave solution, numerical simulations and, finally, estimate the minimum value of the wavefront speed of the disease dispersion.

References

[1] W.A. Boeger, M.R. Pie, A. Ostrensky and L. Patella. "Lethargic crab disease: multidiciplinary evidence supports a mycotic etiology" *Mem. Inst. Oswaldo Cruz*, v. 100(2), p. 161-167, 2005.
[2] C.P. Ferreira, M.R. Pie, L. Esteva, P.F.A. Mancera, W.A. Boeger and A. Ostrensky, "Modelling the lethargic crab disease", *Journal of Biological Dynamics*, v. 3, n. 6, p. 620-634, 2009.

COMPETITION MODEL ON A FINITE DOMAIN

F. Azevedo, **R. Kraenkel** and **D. Silva**

We consider the competitive Lotka-Volterra model with diffusion on a two-dimensional finite domain. This models competition of two biological species restrained to a patch of habitat. Taking the case of a strong competitor against a weaker one, we numerically integrate the systems of PDEs with Dirichlet boundary conditions. We show that: (i) in the limit of large areas, the stronger one drastically depletes the population of the weaker one, eventually leading to its extinction; (ii) below a certain critical area, both species go extinct (ii) there is exists a region of values of the area of the habitat where both species coexist. The results imply that the principle of competitive exclusion is not valid on fragments. Our results model competitive release mediated by area effects.

Optimal Control of Chikungunya Disease: Larvae Reduction, Treatment and Prevention

D. Moulay[a] and **M.A. Aziz-Alaoui**[a]

[a]Laboratoire de Mathématique Appliquées du Havre (LMAH)
University of Le Havre
25 rue Philippe Lebon, BP540, 76058 Le Havre Cedex
djamila.moulay@univ-lehavre.fr

Since the 1980s, there has been a worldwide re-emergence of vector-borne diseases including Malaria, Dengue, Yellow fever, or more recently chikungunya. These viruses are arthropod-borne viruses (arboviruses) transmitted by arthropods like mosquitoes of Aedes genus. The nature of these arboviruses is complex since it conjugates human, environmental, biological and geographical factors. Recent research [1] has suggested, in particular during the Reunion Island epidemic in 2006, that the transmission by Aedes Albopictus has been facilitated by genetic mutations of the virus.

Models describing the Aedes Albopictus mosquito population dynamics, vector of chikungunya virus and transmission to human population are discussed. On the one hand, we propose model using stage structured model based on the biological mosquito life cycle (eggs, larvae, pupae and adults). On the other hand the transmission to human population is described with SIR type model [2]. First we establish global stability results based on the reproductive number like in [3].

Then, based on the observation during one of the last and most important chikungunya epidemic, three main efforts are considered in order to limit the virus transmission [4]. Indeed, there is not vaccine or specific treatment against chikungunya, that's why the main measures includes:

- individual protection against mosquito bites, like the use of insect repellents or mosquito nets, wearing appropriate clothing, etc;
- symptomatic treatment of infected patient;
- control of the proliferation of adult mosquito mainly by reducing the number of larvae in breeding sites which was one of the vector control tools.

Therefore, we look at time dependent breeding sites destruction, prevention and treatment efforts, where optimal control theory is applied. Using analytical and numerical techniques, it is shown that there are cost effective control efforts.

References

[1] M. Enserink, "Epidemiology: Tropical Disease Follows Mosquitoes to Europe", *Science*, v. 317, 2007.
[2] D. Moulay, M.A. Aziz-Alaoui, and M. Cadivel,"The Chikungunya Disease : Modeling, Vector and Transmission Global Dynamics", *submitted to Mathematical Biosciences (2010).*
[3] L. Esteva, and C. Vargas, "Analysis of a dengue disease transmission model", *Mathematical Biosciences*, v. 150,p. 131-151,1998.
[4] D. Moulay, H.D. Kwon M.A. Aziz-Alaoui,"Optimal Control of Chikungunya Disease", *in preparation.*

THE BASIC REPRODUCTION NUMBER IN PERIODIC POPULATION MODELS

N. Bacaër[a]

[a]Institut de Recherche pour le Développement
32 avenue Henri Varagnat
93143 Bondy, France
bacaer@bondy.ird.fr

The basic reproduction number R_0 can be defined for periodic continuous-time population models as the spectral radius of an integral operator [1]. Various methods can be used to compute R_0: discretization of the integral equation, Fourier series, perturbation theory, and Floquet theory (in the special case of ordinary differential equations) [2]. When the seasonal factor is sinusoidal, R_0 satisfies a simple characteristic equation involving a continued fraction [3]. In some cases the external periodic factor can produce some kind of resonance, thus yielding values of R_0 that are quite different from the one obtained by averaging the model's periodic factor [4]. $R_0 = 1$ is in some sense still a threshold for the final size of epidemics [5]. Finally R_0 can also be computed in periodic discrete-time population models [6].

References

[1] N. Bacaër, S. Guernaoui, "The epidemic threshold of vector-borne diseases with seasonality", *Journal of Mathematical Biology*, v. 53, p. 421-436, 2006.
[2] N. Bacaër, "Approximation of the basic reproduction number R_0 for vector-borne diseases with a periodic vector population", *Bulletin of Mathematical Biology*, v. 69, p. 1067-1091, 2007.
[3] N. Bacaër, R. Ouifki, "Growth rate and basic reproduction number for population models with a simple periodic factor", *Mathematical Biosciences*, v. 210, p. 647-658, 2007.
[4] N. Bacaër, X. Abdurahman, "Resonance of the epidemic threshold in a periodic environment", *Journal of Mathematical Biology*, v. 57, p. 649-673, 2008.
[5] N. Bacaër, M.G.M. Gomes, "On the final size of epidemics with seasonality", *Bulletin of Mathematical Biology*, v. 71, p. 1954-1966, 2009.
[6] N. Bacaër, "Periodic matrix population models: growth rate, basic reproduction number and entropy", *Bulletin of Mathematical Biology*, v. 71, p. 1781-1792, 2009.

Savanna-Fire Model: Combined effects of tree-tree establishment competition and spatially explicit fire on the spatial pattern of trees in savannas

Flora S. Bacelar[a], **Justin M. Calabrese**[b], **Volker Grimm**[b] and **Emílio Hernández-García**[a]

[a]IFISC(CSIC-UIB) Instituto de Física Interdisciplinar y Sistemas Complejos
Campus Universitat Illes Balears, E-07122 Palma de Mallorca, Spain
florabacelar@ifisc.uib-csic.es

[b]Dept. of Ecological Modelling
Helmholtz Centre for Environmental Research (UFZ)
Leipzig, Germany
justin.calabrese@ufz.de

Savannas are characterized by robust coexistence of a discontinuous tree layer superimposed on a continuous grass layer. Savannas occur across a wide range of climatic, edaphic, and ecological conditions covering approximately one fifth of the earth's land area. In some countries these grass- dominated ecosystems are a principal biotic resource playing important roles in both the configuration of natural landscapes and in local economies. Identifying the mechanisms that facilitate tree-grass coexistence in savannas has remained a persistent challenge in ecology and is known as the "savanna problem", [1].

Several studies have suggested that adult trees can protect vulnerable juveniles from fire, thus increasing their chances of survival. Exactly how such protection works has not been intensively studied. However, given the frequent occurrence of fires in many savannas, it seems likely that the protection effect may be one of the most common forms of positive facilitation among savanna trees. Alternatively, many studies have documented the importance of competition among trees in diverse savannas. In a previous paper, Calabrese et al., [3], studied the interaction between competition and fire in a highly simplified savanna model. They showed that these two forces interact non-linearly with sometimes surprising consequences for tree population density and spatial pattern. However, because Calabrese et al., [3], treated fire in a non-spatially explicit manner, they did not study the protection effect, and thus could not fully tease apart how these contrasting of local interactions function in combination.

In the present work, a model is proposed to combine the previous savanna model, [3], with the Drossel-Schwabl forest fire model [4], therefore representing fire in a spatially explicit manner. We use the model to explore how the pattern of fire spread, coupled with an explicit, fire-vulnerable tree life stage and the negative and positive effects of adult trees on juvenile trees affects tree density and spatial pattern in savannas. Tree density depended strongly on both fire frequency and fire front patterns. Furthermore, the relationship between the post-fire grass recovery rate and the tree growth rate was a key factor in mediating tree-grass coexistence and its robustness. Different spatial patterns can be found depending on the parameters of the system: regular, random, and clumped. The last one, in contrast with the previous model, appears in two different forms, namely open and closed clusters, due to contrasts between the local interactions (protection and competition). When changing recovery rate or sparkling frequency, transitions from exponential cluster-size to power-law behavior can be found in the tree canopy distributions.

References

[1] Sarmiento, G. The ecology of neotropical savannas. Harvard University Press, Cambridge, MA, 1984.

[2] Michael E. Hochberg, Jean Claude Menaut, and Jacques Gignoux. The influences of tree biology and fire in the spatial structure of the West African savannah. *Journal of Ecology*, 82:217–226, 1994.

[3] Justin M. Calabrese, Federico Vázquez, Cristóbal López, Maxi San Miguel, and Volker Grimm. The interactive effects of tree-tree establishment competition and fire on savanna structure and dynamics. *the american naturalist*, 175(3):45-65, 2010.

[4] B. Drossel and F. Schwabl. Self-organized critical forest-fire model. *Physical Review Letters*, 69(11):1629–1632, 1992.

AN AGENT-BASED MODEL ON VECTOR-BORNE DISEASES: THE RIFT VALLEY FIEVER CASE IN FERLO (SENEGAL)

A. Bah, **A. T. Gaye**, **C. M. F. Kebe**, **J. A. Ndione**, **P. I. Ndiaye** and **S. Ndiaye**

Vector-borne diseases are highly sensitive to environment and to environmental changes. Faced with their recrudescence and public concern about the possible consequence of climate and climate change, International Programs have been launched for studying climate impacts on public health and animal health in low-incoming countries. As part of these efforts, the AMMA project, in its WP 3.4 (Health impacts) with the main objectives in West Africa, is to identify the roles of meteorological and environmental variables in patterns and diffusion of Rift Valley Fever (RVF) in Senegal. Rift Valley Fever (RVF), is a mosquito-borne zoootic virus associated with severe disease in humans and important economic consequences to the livestock sector. There are a number of accounts investigating different aspects of RVF transmission - entomological, climate and farming and livestock factors. However, no previous study has taken an overall view of all these factors to understand dis ease spread. This limitation underlines the necessity to use computational model approach based on multi-agent system in the study of vector-borne diseases transmission and diffusion.

In this paper, a multi-agent system combining conceptual model expressiveness and mathematical model rigor is used to study the transmission cycle and the climate parameter impact on the Rift valley fever outbreak in Ferlo Barkedji in North Senegal. Simulation scenarios combining various parameters (rain quality, and spacing, aggressiveness, death rate, vector ecological preference, camp dispersal around ponds etc.) are unrolled. First simulations carried out from available data on RVF outbreaks on Barkedji as part of previous studies confirm those performed in previous studies, namely RVF apizooties starting is linked to long rain pauses followed by strong and intense rainfall events at the end of the rainy season. Transmission risk maps combining the rainy season quality and vector biology are suggested.

EPIDEMICS ON RANDOM NETWORKS INCORPORATING HOUSEHOLD STRUCTURE

Frank Ball[a]

[a]School of Mathematical Sciences
University of Nottingham
University Park, Nottingham NG7 2RD, UK.
frank.ball@nottingham.ac.uk

Keywords: SIR epidemics, random social network, households, local and global contacts, branching process approximations, threshold behaviour.

There has been a growing interest in models for epidemics among structured populations, which incorporate realistic departures from homogeneous mixing whilst maintaining mathematical tractability. Two classes of structured population epidemic models that have attracted considerable recent attention are network models (in which there is a random graph describing possible infectious contacts) and household models (in which the population is partitioned into households with different contact rates for within- and between-household infection). In this talk I describe and analyse a model for the spread of an SIR (susceptible $\rightarrow$ infective $\rightarrow$ removed) epidemic that includes both of these features. The analysis includes deriving a threshold parameter which determines whether or not an epidemic with few initial infectives can become established and lead to a major outbreak, and determining the probability and expected final size of a major outbreak. The model is compared and contrasted with standard household and network models and vaccination strategies are briefly considered.

The talk is based on research done jointly with David Sirl (University of Nottingham) and Pieter Trapman (Stockholm University), described in [1,2] and supported by the UK Engineering and Physical Sciences Research Council, under research grant number EP/E038670/1 and by the Netherlands Organisation for Scientific Research (NWO) through a VICI grant awarded to Ronald Meester.

References

[1] F. G. Ball, D. Sirl, and P. Trapman, "Threshold behaviour and final outcome of an epidemic on a random network with household structure", *Adv. Appl. Prob.*, v. 41, p. 765-796, 2009.
[2] F. G. Ball, D. Sirl, and P. Trapman, "Analysis of a stochastic SIR epidemic on a random network incorporating household structure", *Math. Biosci.*, v. 224, p. 53-73, 2010.

The spread of SIR epidemics in a population of overlapping groups

Frank Ball[a], **David Sirl**[a] and **Pieter Trapman**[b]

[a]School of Mathematical Sciences
University of Nottingham
University Park, Nottingham, NG7 2RD, UK
frank.ball@nottingham.ac.uk
david.sirl@nottingham.ac.uk

[b]Department of Mathematics
Stockholm University
106 91 Stockholm, Sweden
ptrapman@math.su.se

We consider the spread of an SIR epidemic with general infectious period distribution, in a fixed population of overlapping groups. The groups might represent households, schools, workplaces etc.
The population is modeled by the so-called random intersection graph [1], which enables us to construct networks with a prescribed group-size distribution and a prescribed distribution of the number of groups a uniformly chosen individual is part of. Furthermore, it is possible to control the mean and variance of the degree distribution (i.e. the distribution of the number of neighbors of a uniformly chosen individual) and the clustering (the number of triangles in the network).
Since we do not require the infectious period to be fixed, branching process approximations are not straightforward and dependencies arise between the number of infected individuals within a group and the length of the infectious period of an individual infected in that group. However, we show how theory on multitype Galton-Watson branching processes and branching random walks [2] can be used to approximate the probability of extinction and the expected final size (fraction of the population that will be infected during the epidemic). We also give an implicit definition of a reproduction number, which has critical value 1.
We show, that contrary to what is known for epidemics in usual random network models, an infinite variance of the degree distribution does not guarantee that the probability of a large outbreak is positive.

References

[1] M. Deijfen, and W. Kets, "Random intersection graphs with tunable degree distribution and clustering", *Probab. Eng. Inform. Sci.* v. 23, p. 661-674, 2009.
[2] B. Bollobás, S. Janson and O. Riordan, "The phase transition in inhomogeneous random graphs", *Random. Struct. Algor.* v. 31, p. 3-122, 2007.

MAIN DETERMINANTS OF HUMAN INFLUENZA PHYLODYNAMICS

S. Ballesteros[a], **A. Camacho**[b], **E. Vergu**[c] and **B. Cazelles**[b,d]

[a]Centro de Matematica e Aplicacoes Fundamentais (CMAF)
Universidade de Lisboa, Lisboa, Portugal
sebastien.ballesteros@gmail.com

[b]UMR 7625 (UPMC, ENS, AgroParisTech, CNRS)
Ecole Normale Superieure, Universite Pierre et Marie Curie-Paris 6, Paris, France
camacho@biologie.ens.fr, cazelles@biologie.ens.fr

[c]MIA UR341
INRA, Jouy-en-Josas, France
elisabeta.vergu@jouy.inra.fr

[d]UMMISCO UMI 209
IRD-UPMC, Bondy, France

As stated in [1], three main mechanisms have been proposed to explain the particular phylodynamic patterns observed in human influenza viruses:
• a short-lived full cross-immunity among viral strains [2],
• a punctuated evolution of the hemagglutinin (HA) gene among antigenic types that are linked by a network of neutrally evolving sites [3],
• a continuous reuse by the viruses of a limited number of antigenic combinations [4].

We present a general framework to determine which association of these hypotheses best explains influenza phylodynamics. As source-sink dynamics [5] and additional local heterogeneity [6] have been reported to be important factors for influenza evolution, we start by considering a worldwide metapopulation model where nodes are jointly considered incorporating contact networks. Using this complex framework, we derive different simple models to characterize the processes that lead to the observed phylodynamics patterns.

Our finding reveal the key role of immune boosting, a mechanism where exposed individuals that do not contract infections nevertheless gain additional cross-protection.

References

[1] E.C. Holmes and, B.T. Grenfell, "Discovering the Phylodynamics of RNA Viruses", *PLoS Comput Biol*, 5(10): e1000505, 2009.
[2] N.M. Ferguson , A.P. Galvani and R.M. Bush, "Ecological and immunological determinants of influenza evolution", *Nature* 422 p. 428-433, 2003.
[3] K. Koelle, S. Cobey, B. Grenfell and M. Pascual, "Epochal evolution shapes the phylodynamics of interpandemic influenza A (H3N2) in humans", *Science* 314 p. 1898-1903, 2006.
[4] M. Recker, OG. Pybus, S. Nee and S. Gupta, "The generation of influenza outbreaks by a network of host immune responses against a limited set of antigenic types", *Proc Natl Acad Sci USA* 104 p. 7711-7716, 2007.
[5] A. Rambaut, O.G. Pybus, M.I. Nelson, C. Viboud, J.K. Taubenberger, and E.C. Holmes, "The genomic and epidemiological dynamics of human influenza a virus", *Nature*, 453(7195) p. 615-619, 2008.
[6] F.M. Tria, L.P. Lassig and S. Franz "A minimal stochastic model for influenza evolution", *Journal of Statistical Mechanics: Theory and Experiment* 2005: P07008, 2005.

NOISE INDUCED PHENOMENA IN DELAYED RATIO-DEPENDENT MODEL - MOMENT BASED STABILITY ANALYSIS

Malay Banerjee

Department of Mathematics and Statistics
I. I. T. Kanpur
Kanpur - 208016, INDIA
malayb@iitk.ac.in

Nonlinear dynamical analysis of prey-predator systems with prey dependent functional response has received significant attention in last three decades. Beside the ordinary differential equation models, the mathematical analysis of delay differential equation and stochastic differential equation models reveled various interesting dynamic features like delay induced oscillation, persistence, noise induced coexistence and extinction depending upon the type of interaction and magnitudes of intrinsic parameters. Relatively new classes of ratio-dependent prey-predator models remain less studied in presence of environmental driving forces. It is evident that the deterministic extinction scenario changes to stochastic coexistence in presence of environmental driving forces. The article aims to study the moment stability around various equilibrium points of a delayed ratio-dependent predator-prey model in the presence of additive and multiplicative white noise terms. The moment based stability analysis is performed in terms of first and second order moments. Qualitative change in dynamical behaviors (coexistence, persistence, periodic coexistence etc.) is analyzed for the model perturbed by additive as well as multiplicative noise; analytical findings are validated through exhaustive numerical simulation. Stability criteria in terms of stability of the moments for the solutions of stochastically perturbed system are compared to the stability criteria of unperturbed system.

SPATIO-TEMPORAL PATTERN FORMATION IN HOLLING-TANNER MODEL IN PRESENCE OF MULTIPLICATIVE NOISE

Malay Banerjee

Department of Mathematics and Statistics
I. I. T. Kanpur
Kanpur - 208016, INDIA
malayb@iitk.ac.in

Turing and non-Turing patters along with spatio-temporal chaos have been studied extensively for predator-prey models within heterogeneous environment. Most of the non-Turing patterns and spatio-temporal chaos were reported for 'Gause-type' predator-prey models where the per-capita predation rate follows prey-dependent functional response. It is evident that said models having constant per-capita death rate for predators never exhibit Turing pattern. Majority of the reported works are based upon the deterministic models, although it is known that natural systems are affected by random fluctuations, arising from either environmental or demographic stochasticity. Motivated by these facts the main objective of the present paper is to study the Turing-pattern formation in a Holling-Tanner model of predator-prey interaction and how this pattern alter in presence of multiplicative noise terms. The conditions for instability and multiplicative noise-induced pattern formation are derived based on consideration of first and higher order moments. Numerical simulations are carried out to substantiate the analytical findings.

Asymptotic dynamics of a population density under selection-mutation

Guy Barles[a], **Sepideh Mirrahimi**[b] and **Benoît Perthame**[c]

[a]Laboratoire de Mathématiques et Physique Théorique, CNRS UMR 6083, Fédération Denis Poisson,
Université François Rabelais, Department of Mathematics,
Parc de Grandmont, 37200 Tours, France,
barles@lmpt.univ-tours.fr

[b]Université Pierre et Marie Curie-Paris 6, UMR 7598 LJLL,
F-75252 Paris Cedex 5, France,
mirrahimi@ann.jussieu.fr

[c]Université Pierre et Marie Curie-Paris 6, Institut Universitaire de France, UMR 7598 LJLL,
F-75252 Paris Cedex 5, France,
benoit.perthame@upmc.fr

Speaker underlined.
Key-Words: Adaptive evolution, Lotka-Volterra equation, Hamilton-Jacobi equation, viscosity solutions, Dirac concentrations.

We study the dynamics of a population density under selection and mutations between phenotypical traits. We model the dynamics by some equations of Lotka-Volterra type. Two models are studied. In the first one a Laplace term represents the mutations. In the second one we model the mutations by an integral kernel. In both cases, we use a nonlinear birth-death term that corresponds to the competition between the traits leading to selection.

By studying the asymptotic behavior of these equations in large time, while mutations are rare, we prove that the density goes to a sum of Dirac masses that are traveling in time. In biological terms, at every moment one or several dominant traits survive while other traits go extinct. The dominant traits change in time due to the presence of mutations. We describe the limit population density by a constrained Hamilton-Jacobi equation.

References

[1] G. Barles, S. Mirrahimi and B. Perthame, "Concentration in Lotka-Volterra parabolic or integral equations: a general convergence result". *Methods Appl. Anal., to appear.*
[2] G. Barles and B. Perthame, "Concentrations and constrained Hamilton-Jacobi equations arising in adaptive dynamics". *In Recent Developements in Nonlinear Partial Differential Equations, D. Danielli editor. Contemp. Math. 439*, p. 57-68, 2007.
[3] G. Barles and B. Perthame, "Dirac concentrations in Lotka-Volterra parabolic PDEs". *Indiana Univ. Math. J. 57 (7)*, p. 3275-3301, 2008.
[4] O. Diekmann, P.E. Jabin, S. Mischler and B. Perthame, "The dynamics of adaptation: an illuminating example and a Hamilton-Jacobi approach". *Th. Pop. Biol., 67 (4)*, p. 257-271, 2005.
[5] M. Gauduchon and B. Perthame, "Survival thresholds and mortality rates in adaptive dynamics: conciliating deterministic and stochastic simulations", *Math. Med. Biol., to appear.*

NONLINEAR MATRIX MODEL FOR COMPETITION BETWEEN TWO DISCRETE-STRUCTURED PLANT POPULATIONS

Iya N. Belova

Laboratory of Mathematical Ecology, Institute of Atmospheric Physics,
Russian Academy of Sciences, 3 Pyzhevsky Lane, Moscow, 119017, Russia
iyabelova@mail.ru

The species of wood reed (*Calamagrostis epigeios* (L.) Roth) and common birch (*Betula pendula* Roth) pioneer open area after a spruce forest clear-cut, the competition between them being considered as the major mechanism of secondary forest succession at the earliest stage [1]. The wood reed rapidly colonizes the area first, thereafter tending to block the birch growth. Each species possesses a complex population structure in terms of the chronological age and ontogenetic stages, the unlimited growth of a single-species population being modeled with a linear matrix model for the age-stage-structured dynamics [2, 3]. Expert knowledge of how the outcome of competition between individual plants does depend upon the age-stage status of the competitors has enabled us to construct a graph of competition influences between (and within) the following macro-groups of plants aggregated from the detailed age-stage structures: R and P, the reproductive and post-reproductive states of the wood reed, S and T, the low-shrub and young-tree forms of the birch respectively. The competition graph then yields the following system of difference equations:

$$R(t+1) = [I(t) + R(t)]\, p(1-c)\, f_1(R(t), P(t), S(t), T(t)),$$

$$P(t+1) = R(t)\, p\, c\, f_1(R(t), P(t), S(t), T(t)) + P(t)\, s\, f_2(R(t), P(t), S(t), T(t)),$$

$$S(t+1) = [J(t) + S(t)]\, d(1-b)\, f_3(R(t), P(t), S(t), T(t)),$$

$$T(t+1) = S(t)\, b\, d\, f_3(R(t), P(t), S(t), T(t)) + q\, T(t)\, f_4(T(t)),$$

where decreasing functions $f_j(\ldots)$ of the competitor population(s) designate some nonlinear modification of the age-stage-specific survival and reproduction rates of the single-species linear models. Thus, aggregation provided for "downscaling" to the 4D level, where it appears possible to accurately calibrate model parameters on the observation data, as well as to find out a proper equilibrium and to analyze its local stability.

When calibrated, the nonlinear model exhibits convergence to steady equilibrium $[R^*, P^*, 0, T^*]$ – a state of the phytocoenosis that is interpreted as young, closed-canopy, birch forest with suppressed wood reed population and the young birch growth strongly suppressed. The model illustrates the observed course of forest renewal. The appearance of young birch shoots and the growth of birch population overpass the wood reed competitive resistance and result in domination of the birch, which thereafter exerts strong competitive impact on both the wood reed growth and the own young growth. Remarked is a potential of the model as an object of more general mathematical study and a tool to predict the course of forest renewal.

References

[1] N.G. Ulanova, I.N. Belova, D.O. Logofet, "On the competition among discrete-structured population: a matrix model for population dynamics of woodreed and birch growing together", *Zhurnal Obschei Biologii* (*Journal of General Biology*), vol.69, №6: 441-457, 2008,(in Russian, with English summary).

[2]H. Caswell, "Matrix population models: construction, analysis, and interpretation". Sunderland, MA: Sinauer Associates. 328 p.

[3]D.O. Logofet, and I.N. Belova, "Nonnegative matrices as a tool to model population dynamics: classical models and contemporary expansions", *Journal of Mathematical Sciences*, vol.155, № 6, p. 894-907.

HOPF BIFURCATIONS IN THE MARINE SIZE SPECTRUM GENERATED BY FISHING

E. Benoît[a] and **M.J. Rochet**[b]

[a]Department of Mathematics
University of La Rochelle
avenue Michel Crépeau 17042 LA ROCHELLE, France
ebenoit@univ-lr.fr

[b]IFREMER
rue de l'île d'Yeu, BP 21105, 44311 Nantes cedex 3, France
mjrochet@ifremer.fr

bf Keywords : Hopf bifurcation, functional equation, Hodgkin-Huxley, allometry, selective fishing.

Size-structured models for marine food webs are well studied (see references below). The ingredients for these models (predation, growth, mortality) are generally allometric and the most popular result is the existence of an equilibrium size-spectrum, often linear. But, fishing is not allometric. We perform simulations where the equilibrium (without fishing) breaks down with a Hopf bifurcation when fishing is increased.
We study simpler models, with the same mathematical structure, to show mathematically such Hopf bifurcations.
All the considered models are of the type

$$\frac{\partial u}{\partial t} \;=\; -\frac{\partial}{\partial x}\left\{\mathcal{G}\left(x, u(.,t)\right)\; u\right\} \;-\; \mathcal{M}\left(x, u(.,t)\right)\; u$$

where $\mathcal{G}$ and $\mathcal{M}$ are integral operators.

References

[1] E. Benoît, and M.J. Rochet, "A continuous model of biomass size spectra governed by predation and the effects of fishing on them", *Journal of Theoretical Biology*, v. 226, n. 1, p. 9-21, 2004.
[2] K.L. Denman, and H.J. Freeland, and D.L. Mackas, "Comparisons of time scales for biomass transfer up the marine food web and coastal transport processes", *Can. Spec. Publ. Fish. Aquat. Sci.*, v. 108, p. 255-264, 1989.
[3] R.W. Sheldon, and A. Prakash, and W.H.J. Sutcliffe, "The size distribution of particles in the ocean", *Limnol. Oceanogr.*, v. 17, n. 3, p. 327-340, 1972.
[4] W. Silvert, and T. Platt, "Dynamic energy flow model of the particle size distribution in pelagic ecosystems", In: W. Kerfoot (Ed.), "Evolution and Ecology of Zooplancton Communities", University Press of New England, Illanover, NH, p. 754-763, 1980.
[5] M.L. Thiebaux, and L.M. Dickie, "Models of aquatic biomass size spectra and the common structure of their solutions", *J. Theor. Biol.*, v. 159, p. 147-161, 1992.

OPTIMAL CONTROL FOR THE DYNAMICS OF CHRONIC MYELOID LEUKEMIA

C. Benosman and **B. Aïnseba**

Université Bordeaux 2
INRIA, Institut de Mathématiques de Bordeaux
3 Ter Place de la Victoire, 33076 Bordeaux, France
bedreddine.ainseba@inria.fr
chahrazed.benosman@inria.fr

The mathematical modelling of hematopoesis received a significant attention in the last few years. However, the treatment of hematological diseases is less investigated by optimal control tools. In this work, we consider the dynamics of chronic myeloid leukemia, based on a two age-structured populations: normal and cancer hematopoietic stem cells (HSC). We represent the effects of therapy as an optimal control problem to minimize the cost of treatment and the number of cancer HSC. Numerical results illustrate the optimal control and the dynamics of HSC.

References

[1] V. Barbu, and M. Iannelli, "Optimal control of population dynamics", *Journal of Optimization Theory and Applications*, v. 102, p. 1-14, 1999.
[2] S.N. Catlin, P. Guttorp, and J.L. Abkowitz, "The kinetics of clonal dominance in myeloproliferative disorders", *Blood*, v. 106, p. 2688-2692, 2005.
[3] U. Ledzewicz, and H. Schättler, "Optimal controls for a model with pharmacokinetics maximizing bone marrow in cancer chemotherapy", *Mathematical Biosciences*, v. 206, p. 320-342, 2007.
[4] L. Pujo-Menjouet, and M.C. Mackey, "Contribution to the study of periodic chronic myelogenous leukemia", *Comptes Rendus Biology*, v. 327, p. 235-244, 2004.

Modélisation de la croissance métastatique sous contrôle angiogénique : analyse mathématique et numérique

S. Benzekry[a]

[a] LATP Laboratoire d'Analyse, Topologie, Probabilités - UMR 6632
Université de Provence
CMI, Technopôle Château-Gombert, 39, rue F. Joliot Curie, 13453 Marseille Cedex 13
benzekry@phare.normalesup.org

A l'heure actuelle, le cancer est devenu la première cause de mortalité en France. Les mécanismes d'action des thérapies disponibles, cytotoxiques (qui détruisent la cellule cancéreuse) ou cytostatiques (qui stabilisent la cellule cancéreuse) sont complexes et par conséquent la modélisation mathématique s'avère être un outil fondamental afin de produire des traitements efficaces. Dans ce sens, nous avons construit un modèle décrivant la dynamique tumorale modifiée par l'intervention d'un traitement médicamenteux, qui prend en compte l'angiogénèse (création d'une néo-vasculature au voisinage de la tumeur). Ainsi nous disposons d'un outil capable d'optimiser l'efficacité antitumorale des traitements.

Dans ce travail, nous proposons un modèle simple (peu de paramètres) décrivant la dynamique de la densité de métastases. Il fait suite au modèle développé dans [2] et utilise celui de [3] pour décrire l'angiogénèse. On obtient une équation de transport en dimension 2, avec une condition aux limites non-locale (équation de renouvellement), et un champ de vitesse qui dégénère :

$$\begin{cases} \partial_t \rho + \mathrm{div}(G\rho) = 0 & \text{sur }]0,\infty[\times\Omega \\ -G\cdot\overrightarrow{\nu}\rho(t,\sigma) = N(\sigma)\int_\Omega \beta(x,\theta)\rho(t,x,\theta)dxd\theta + f(t,\sigma) & \text{sur } \Gamma\subset\partial\Omega \\ \rho(0,\cdot) = \rho^0(\cdot) & \text{sur } \Omega \end{cases} . \tag{5}$$

Nous présentons l'analyse mathématique de cette équation [1], à savoir l'existence et l'unicité des solutions ainsi que leur régularité et le comportement asymptotique.

Dans un second temps, nous proposons une discrétisation du problème. En raison de la forme particulière des caractéristiques du champ G, un schéma upwind classique se révèle peu performant. Nous utilisons plutôt une méthode lagrangienne, qui consiste à discrétiser le problème en suivant les caractéristiques. La concentration de ces dernières autour d'une caractéristique principale nous conduit à étudier la limite des solutions de l'équation (5) lorsque la fonction N tend vers un dirac. Nous en déduisons une méthode numérique particulièrement efficace pour prédire l'état métastatique du patient en tenant compte des phénomènes angiogéniques. Ces résultats ainsi que le faible nombre de paramètres dans le modèle sont très encourageants pour aborder l'optimisation des protocoles temporels d'administration des traitements anti-angiogéniques.

References

1. Benzekry, S., *Mathematical analysis of a two-dimensional population model of metastatic growth including angiogenesis.*, submitted.
2. Barbolosi, D. and Benabdallah, A. and Hubert, F. and Verga, F., *Mathematical and numerical analysis for a model of growing metastatic tumors*, Math. Biosci., 218 pp.1-14, 2009.
3. Hahnfeldt, P. and Panigraphy, D. and Folkman, J. and Hlatky, L, *Tumor development under angiogenic signaling : a dynamical theory of tumor growth, treatment, response and postvascular dormancy*, Cancer Research., 59 pp.4770-4775, 1999.

STABILITY OF SCALAR DIFFERENTIAL EQUATION WITH DISTRIBUTED DELAYS

S. Bernard

Linear scalar differential equations with distributed delays appear in the study of the local stability of nonlinear differential equations with feedback, which are common in population biology. Negative feedback loops tend to promote oscillation around steady states, and their stability depends on the particular shape of the delay distribution. Since in applications the mean delay is often the only reliable information available about the distribution, it is desirable to find conditions for stability that are independent from the shape of the distribution. We show here that the linear equation with distributed delays is asymptotically stable if the associated differential equation with a discrete delay of the same mean is asymptotically stable. Therefore, distributed delays stabilize negative feedback loops.

HYBRID MODELING OF CELL POPULATING DYNAMICS

N. Bessonov[a], **P. Kurbatova**[b], **V. Volpert**[b]

[a]Department of Mathematics
Institute of Mechanical Engineering Problems
199178 Saint Petersburg, Russia
nickbessonov@yahoo.com

[b]Department of Mathematics
Institut Camille Jordan, University Lyon 1
UMR 5208 CNRS, 69622 Villeurbanne, France
kurbatova@math.univ-lyon1.fr
volpert@math.univ-lyon1.fr

In this presentation we discuss a hybrid model of cell dynamics applied to biological and medical problems. Cells can interact with each other and with the surrounding medium mechanically and biochemically, they can divide, differentiate and die due to apoptosis. Cell behavior is determined by intra-cellular regulatory networks and by extra-cellular regulation. Each cell can grow, divide, die by apoptosis and exchange biochemical signals with the surrounding medium.

We describe motion of each cell by the displacement of its center. By Newton's second law

$$m\ddot{x}_i + \mu m\dot{x} - \sum_{j\neq i} f(d_{ij}) = 0, \tag{6}$$

where m is the mass of the particle, the second term in the left-hand side describes the friction by the surrounding medium. Dissipative forces can also be written in a different form.

Intra-cellular regulatory networks for the i-th cell are described by a system of ordinary differential equations

$$\frac{du_i}{dt} = F(u_i, u), \tag{7}$$

where u_i is a vector of intra-cellular concentrations, u is a vector of extra-cellular concentrations, F is the vector of reaction rates which should be specified for each particular application. The concentrations of the species in the extra-cellular matrix are described by the diffusion equation

$$\frac{\partial u}{\partial t} = D\ \Delta u + G(u, c), \tag{8}$$

where c is the local cell density, G is the rate of consumption or production of these substances by cells. These species can be either nutrients coming from outside and consumed by cells or some other bio-chemical products consumed or produced by cells.

This approach is applied to multi-scale modelling of normal and leukaemic erythropoiesis.

Keywords: cell dynamics, hybrid models, erythropoiesis, proliferation, apoptosis.

THE EVOLUTION OF HOST RESISTANCE WITH LOCAL INTERACTIONS

A. Best[a], **S. Webb**[b], **A. White**[c] and **M. Boots**[d]

[a]Department of Animal and Plant Sciences
University of Sheffield
Sheffield, S10 2TN
a.best@shef.ac.uk

[b]Department of Mathematics, University of Strathclyde - steven.webb@strath.ac.uk

[c]Department of Mathematics, Heriot-Watt University - a.r.white@hw.ac.uk

[d]Department of Animal and Plant Sciences, University of Sheffield - m.boots@shef.ac.uk

There is a growing body of literature that recognises the importance of spatial structure in determining the evolutionary dynamics of parasites (e.g. [1]). However, less attention has been paid to how spatial structure may affect the evolution of host resistance. Here we investigate the evolution of host resistance with local interactions using a classical host-parasite model framework (SI model) where hosts exist on a regular lattice of sites. Ecological processes, such as disease transmission and host reproduction, may occur locally between neighbouring sites or globally across the lattice. Working within the framework of adaptive dynamics we use the method of pair approximation to numerically produce pairwise invasion plots (PIPs) that predict the evolutionary dynamics. We show that as transmission and reproduction become increasingly local, hosts are selected to increase resistance. High resistance is favoured in the local system due to a combination of 'self-shading', 'kin-shading' and ecological (density-dependent) benefits. Furthermore, we find that evolutionary branching is predicted in the fully local system, but that these branching points are extremely sensitive to the level of local interactions. We also present results from stochastic simulations that confirm our predictions. Our results further highlight the importance of spatial structure in determining the evolutionary behaviour in host-parasite systems.

Keywords: host resistance, spatial structure, pair approximation, adaptive dynamics.

References

[1] M. Boots, and A. Sasaki, "'Small worlds' and the evolution of virulence: infection occurs locally and at a distance", *Proc. Roy. Soc. B*, v. 266, p. 1933-1938, 1999.

A HIV/AIDS EPIDEMIC MODEL WITH COUNSELLING AND ABSTINENCE

C.P. Bhunu[a], **S. Mushayabasa**[a], **W. Garira**[b], **H. Kojouharov**[c] and **J.M. Tchuenche**[d]

[a]Department of Mathematics
National University of Science and Technology
P. O. Box 939 Ascot, Bulawayo, Zimbabwe
cpbhunu@gmail.com

[b]Department of Mathematics and Applied Mathematics
University of Venda
P. Bag X5050 Thohoyanndou 0950 South Africa
author2@institute.com

[c]Department of Mathematics
University of Texas at Arlington
Arlington, TX 76019-0408, USA
hristo@uta.edu

[d]Department of Mathematics and Statistics
University of Guelph
Guelph, ON N1G 2W1 Canada
jtchuenc@uoguelph.ca

We discuss the problem of HIV/AIDS control, more specifically how the extensive counselling and the resulting decrease in sexual activity could affect the HIV epidemic in sub-Saharan Africa via a deterministic model. The threshold quantities are determined and stabilities analyzed. Theoretical analysis and numerical simulations support the idea that increase in the number of sexually inactive HIV positive individuals who voluntarily abstain from sex has a positive impact on HIV/AIDS control. Results from this theoretical study suggest that effective counselling and testing have a great potential to partially control the epidemic (especially when HIV positive individuals either willingly withdraw from risky sexual activities or disclose their status beforehand) even in the absence of antiretroviral therapy (ART). Sensitivity analysis shows that voluntary withdrawal from (risky) sexual behaviour of HIV positive individuals has a significant impact on the disease transmission.

References

[1] C.P. Bhunu, W. arira, and G. agombedze, “Mathematical analysis of a two strain HIV/AIDS model with antiretroviral treatment", *Acta Biotheor.*, v. 57, p. 361-381, 2009.
[2] C.S. Pedamallu, L. Ozdamar, and G-W. Weber, “A system dynamics for intentional transmission of HIV/AIDS using Cross Impact Analysis", *preprint*, 2009.

FROM INDIVIDUAL TO POPULATION LEVEL EFFECTS OF TOXICANTS IN THE TUBICIFID BRANCHIURA SOWERBYI USING THRESHOLD EFFECT MODELS IN A BAYESIAN FRAMEWORK

E. Billoir[a], **S. Charles**[a] and **V. Ducrot**[b]

[a]Laboratoire de Biométrie et Biologie Evolutive, UMR CNRS 5558
Université Lyon 1
Villeurbanne, France
billoir@biomserv.univ-lyon1.fr

[b]Ecologie et Santé des Ecosystèmes, UMR 985
INRA
Rennes, France
virginie.ducrot@rennes.inra.fr

Keywords
Ecotoxicology; Threshold stress functions; Matrix population model; *Branchiura sowerbyi*; Zinc;

Effects of zinc were studied in the freshwater worm *Branchiura sowerbyi* using partial and full life-cycle tests. Only newborn and juveniles were sensitive to zinc, displaying effects on survival, growth and age at first brood at environmentally relevant concentrations.

Threshold effect models were proposed to assess toxic effects on individuals. They were fitted to life-cycle test data using Bayesian inference [1], and adequately described life-history trait values in exposed organisms [2]. The daily asymptotic growth rate of theoretical populations was then simulated from individual-level outputs with a two stage-classified matrix model [3] based on the partial life-cycle graph of the worm. Population-level outputs were in good accordance with literature and field observations. Working in a Bayesian framework allowed incorporating parameter uncertainty and correlations in the simulation of the population-level response [4], thus increasing the relevance of test results in the context of ecological risk assessment.

References

[1] W.R. Gilks, S. Richardson and D.J. Spiegelhalter, MCMC in Practice. Chapman et Hall/CRC, 1996.
[2] E. Billoir, M.L. Delignette-Muller, A. Péry and S. Charles, On the use of Bayesian inference to estimate DEBtox parameters. *Environmental Science and Technology* 42:8978-8984, 2008.
[3] H. Caswell, Matrix Population Models - Construction, analysis, and interpretation, 2nd edition, Sinauer Associates, Sunderlands, Massachussets, 2001.
[4] S. Charles, E. Billoir, C. Lopes and A. Chaumot, Matrix population models as relevant modeling tools in ecotoxicology, in Ecotoxicology modeling, Springer, 2009.

MODELLING OF DAPHNID RESPONSE TO CADMIUM IN AQUATIC MICROCOSMS

E. Billoir[a], **H. Delhaye**[b], **B. Clément**[b], **M.L. Delignette-Muller**[a] and **S. Charles**[a]

[a]Laboratoire de Biométrie et Biologie Evolutive, UMR CNRS 5558
Université lyon 1
Villeurbanne
billoir@biomserv.univ-lyon1.fr

[b]Laboratoire des Sciences de l'Environnement
ENTPE
Vaulx-en-Velin
clement_b@entpe.fr

Keywords
Ecotoxicology; *Daphnia magna*; Cadmium; Threshold stress functions; Matrix population models;

2-L indoor microcosm experiments, in which five aquatic species were introduced (algae *P. subcapitata*, duckweeds *Lemna minor*, diptera lavae *Chironomus riparius*, amphipods *Hyallela azteca* and cladocerans *Daphnia magna*), were carried out at the ENTPE (Vaulx-en-Velin, France). Cadmium effect was studied at four treatment levels with initial concentrations of 10, 20, 40 and 80 μg.L^{-1} plus control. Experimental data were collected over 21 days in static conditions, *i.e.* the microcosms evolved without water renewal. Because of speciation, Cd concentrations in water decreased with time. Here we present a focus on *D. magna* response through three life history traits which were followed up during all the experiment: number of survivors, body length and number of offsprings. In addition, water samples were regularly collected from all microcosms to follow Cd concentration in water that daphnids were exposed to.

To model the impact of Cd on *D. magna* life history traits, we first described the exposure dynamics. Then, we adopted a strategy based on dynamics modelling in discrete time with the day as time step. Hence, we assumed that Cd effects between day $D-1$ and day D basically depended on the internal concentration (in the daphnids) at day $D-1$, by using a recurrent approach. For the three life history traits considered in this study (survival, growth and reproduction), effect models were built, including threshold stress functions. For each endpoint the toxic effect was supposed to appear when the Cd concentration exceeded a No Effect Concentration (NEC). Within a Bayesian framework, four kinds of data were fitted simultaneously (exposure, survival, growth and reproduction), with apropriate error models. Thanks to Bayesian inference, we obtained point estimates of parameters as well as associated probability distributions reflecting uncertainty.

Then, by combining above effect models and matrix population models [1], every toxic effect was extrapolated from the individual level (reduced fecundity, growth and survival) to the population one by taking into account age dependence of the response. The asymptotic population growth rate was obtained continuously vs. Cd concentration and accompanied by a confidence band echoing the uncertainty previously highlighted. Pertubation analyses were also performed to highlight critical demographic parameters in the relationship between the asymptotic population growth rate and the Cd concentration [2].

References

[1] H. Caswell, Matrix Population Models - Construction, analysis, and interpretation, 2nd edition, Sinauer Associates, Sunderlands, Massachussets, 2001.
[2] S. Charles, E. Billoir, C. Lopes and A. Chaumot, Matrix population models as relevant modeling tools in ecotoxicology, in Ecotoxicology modeling, Springer, 2009.

ESTIMATION OF THE EMERGENCE TIME OF A MULTI-VIRULENT MUTANT USING BIRTH AND DEATH PROCESS

R. Bourget[a,b], **N. Sapoukhina**[b] and **L. Chaumont**[a]

[a]LAREMA, Département de Mathématiques
Université d'Angers, UFR Sciences
2 Boulevard Lavoisier - 49045, Angers, France
bourget@math.univ-angers.fr
loic.chaumont@univ-angers.fr

[b]UMR077 Plant Pathology-PaVé
INRA Angers-Nantes
BP 60057, F-49071 Beaucouzé, France
Natalia.Sapoukhina@angers.inra.fr

In many biological contexts, humans try to control harmful populations by introducing resistance into their habitat. However, this control is rarely durable because of rapid population adaptation to this new and hostile environment. Multi-resistance is thought to be a way to delay the emergence of multi-virulent mutants. To understand the population adaptation to multi-resistance, we were interested in the impact of the resistant environment structure on the waiting time before emergence of a multi-virulent individual on a multi-resistant environment.

We formulated and analyzed a stochastic population model based on a birth and death process with mutation, migration and recombination. We included a fitness cost for individuals overcoming resistance. To study the model dynamics we used concrete biological examples. The built model is parameterized for fungi and virus responsible for plant diseases: *Venturia inaequalis*, *Uromyces viciae-fabae*, pepper-*Potato virus Y* and *Tomato mosaic virus*.

The numerical simulation showed the dependence of the emergence time on the ratio between resistant and susceptible areas in the environment. In the case of diversify environment including mono-resistant and susceptible areas, emergence time is a parabolic function of the fraction of resistant area. The emergence time was longer for low and high resistant area fractions, because of a low migration probability and a low mutation probability, respectively. For multi-resistant environment, the presence of mono-resistant areas decreased the emergence time of multi-virulent mutants on the multi-resistant areas.

The results of the model provide insight into our understanding the interplay between used resistance strategy and the emergence time of mutants overcoming the resistance. The model can be used for designing environments unfavourable for the rapid evolution of harmful populations.

Keywords: multi-resistance, stochastic model, fitness cost

EVOLUTIONARY ECOLOGY, TRADE-OFFS AND CYCLIC AND CHAOTIC POPULATION DYNAMICS

Roger G. Bowers[a], **Andy Hoyle**[b] and **Andy White**[c]

[a]Department of Mathematical Sciences
The University of Liverpool
Mathematical Sciences Building, Liverpool, L69 7ZL, U.K.
sx04@liverpool.ac.uk

[b]Department of Computing Science and Mathematics
University of Stirling
Stirling, FK9 4LA, U.K
ash@maths.stir.ac.uk

[c]Department of Mathematics and the Maxwell Institute for Mathematical Sciences
Heriot Watt University
Edinburgh, EH14 4AS, U.K
A.R.White@hw.ac.uk

Commonly, studies of the evolution of life-history traits suppose that the underlying population dynamical attractor is stable point equilibrium. However, in different circumstances, evolutionary outcomes can change significantly. Here, we use adaptive dynamics to analyze a discrete-time demographic model involving a trade-off whose shape is also an important determinant of evolutionary behaviour. We derive an explicit expression for the fitness in the region of 2-cycles and consequently present an adaptive dynamic analysis which is fully algebraic. Furthermore, (using a symbolic package) we essentially repeat this for 4-cycles. We illustrate and verify our results using simulations. We find that with equilibrium population dynamics, trade-offs with accelerating costs produce a continuously stable strategy (CSS) whereas trade-offs with decelerating costs produce a non-ES repellor. The transition to 2-cycles produces a discontinuous change: the emergence of an intermediate region in which branching points occur. The size of this region decreases as we move through the region of 2-cycles. There is a further discontinuous fall in the size of the branching region during the transition to 4-cycles. We extend our results numerically and with simulations to higher-period cycles and chaos. Simulations show that chaotic population dynamics can evolve from equilibrium and vice-versa.

OPTIMIZATION ISSUES IN STOCHASTIC ANIMAL GROWTH MODELS

C. A. Braumann[a], **P. A. Filipe**[a], **C. Carlos**[b] and **C. J. Roquete**[c]

[a]Centro de Investigação em Matemática e Aplicações
Universidade de Évora
Rua Romão Ramalho 59, 7000-671 Évora, Portugal
braumann@uevora.pt
pasf@uevora.pt

[b]Escola Superior de Tecnologia do Barreiro
Instituto Politécnico de Setúbal
Rua Américo da Silva Marinho, 2839-001 Lavradio
Clara.Carlos@estbarreiro.ips.pt

[c]Instituto de Ciências Agrárias e Ambientais Mediterrânicas
Universidade de Évora
Núcleo da Mitra, Apartado 94, 7002-774 Évora, Portugal
croquete@uevora.pt

Many deterministic models for the growth of an individual animal from birth to maturity size S can be written as $dY(t) = \beta(A-Y(t))dt$, where $Y(t) = h(X(t))$, with h a strictly increasing C^1 function, $A = h(S)$ and $X(t)$ the size of the individual at age t (e.g., Gompertz model $h(x) = \ln x$, Bertalanffy-Richards model $h(x) = x^c$). In a randomly fluctuating environment, we proposed (see [1]) the stochastic differential equation models $dY(t) = \beta(A-Y(t))dt+\sigma dW(t)$, where σ measures environmental noise intensity and $W(t)$ is a standard Wiener process.

We study properties of the transient and transition probability distributions, as well as the distribution of the time required for an animal to reach a prescribed size. In livestock production, one may want to know what is the optimal age or the optimal size to sell the animal in order to maximize profit. We study those issues assuming the income from selling the animal is proportional to animal size and some alternative assumptions on the costs to maintain the animal until selling time. Results are illustrated using cattle weight data, to which we have applied the Bertalanffy-Richards and the Gompertz stochastic models.

Keywords: animal growth, random environments, stochastic differential equations, optimization, livestock production

References

[1] P. A. Filipe and C. A. Braumann, "Modelling individual animal growth in random environments", *Proceedings of the 23rd International Workshop on Statistical Modelling*, P. H. C. Eilers, Editor, p. 232–237, 2008.

MODELING AND SIMULATING A FUNGAL DISEASE EPIDEMIC OVER A HETEROGENEOUS PLOT

J.-B. Burie[a], **A. Calonnec**[b], **M. Charron**[c] and **P. Legrand**[d]

[a]Université Victor Segalen, Bordeaux 2, UMR CNRS 5251, IMB & INRIA Bordeaux Sud-Ouest, France
3ter Place de la Victoire 33076 Bordeaux Cedex, France
jean-baptiste.burie@u-bordeaux2.fr

[b]INRA-Bordeaux
UMR1065 INRA-ENITA Santé Végétale
71 Avenue Edouard Bourleaux 33883 Villenave d'Ornon Cedex, France
calonnec@bordeaux.inra.fr

[c]INRA, Oniris, UMR1300 BioEpAR, BP 40706, F-44307 Nantes, France
Université Victor Segalen, Bordeaux 2, UMR CNRS 5251, IMB & INRIA Bordeaux Sud-Ouest, France
maud.charron@oniris-nantes.fr

[a]Université Victor Segalen, Bordeaux 2, UMR CNRS 5251, IMB & INRIA ALEA, Bordeaux Sud-Ouest, France
3ter Place de la Victoire 33076 Bordeaux Cedex, France
pierrick.legrand@u-bordeaux2.fr

This model is applied to the case of a powdery mildew epidemic over a vineyard. It is based on a variant of a SEIR model at the plant scale coupled with two PDEs that describe the short (intra-plant) and long (above the canopy) range dispersal of the spores. It also takes into account the host growth that cannot be neglected during the time span of the simulation and the progressive leaves resistance to spore contamination. The vineyard is made of rows regularly spaced. A simpler version of this model was given in [1].

The model equations are discretized in space with a spectral method (truncated Fourier series) and in time with a simple implicit/explicit Euler scheme. We prove the convergence of this scheme. For the simulations we used Matlab© software. We have built an ad hoc interface that allows the user to easily set different parameters for growth, plant susceptibility, disease transmission... for each plant or patch of plants in the plot.

The parameters of the model are given by the literature, or at the vine scale by the calibration of local model [2] using the output of a complex mechanistic model [3].

References

[1] J.-B. Burie, A. Calonnec and M. Langlais, "Modeling of the Invasion of a Fungal Disease over a Vineyard", *Mathematical Modeling of Biological Systems: Epidemiology, evolution and ecology, immunology, neural systems and the brain, and innovative mathematical methods*, Volume II. A. Deutsch et al. eds. Boston: Springer Berlin Heidelberg New York vol. II, p. 11-21, 2007.
[2] J.-B. Burie, A. Calonnec and M. Langlais, "Effect of crop growth and susceptibility on the dynamics of a plant disease epidemic: powdery mildew of grapevine", *Proceedings of PMA09*, IEEE CPS, to appear.
[3] A. Calonnec, P. Cartolaro, J.-M. Naulin, D. Bailey, "A host-pathogen simulation model: powdery mildew of grapevine", *Plant Pathology* 57, p. 493-508, 2008.

LINKING PLANKTON DYNAMICS AND FISH RECRUITMENT IN A STOCHASTIC ENVIRONMENT

J. F. Burrow[a] and **J. W. Pitchford**[b]

[a]Department of Mathematics & York Centre for Complex Systems Analysis
University of York
York, UK
jb561@york.ac.uk

[b]Department of Biology, Department of Mathematics,
& York Centre for Complex Systems Analysis
University of York
York, UK
jwp5@york.ac.uk

The number of fish larvae recruited into the adult population each year is fundamental to the long-term stability of an exploited fish stock and much emphasis is placed on understanding the link between stock size and recruitment. Here "stock" is defined as the biomass of spawning adults in a population, and "recruitment" the number of larvae and juveniles that survive to join the adult population the following year.

High variability in recruitment is well recognised [1]. Fish larvae are born into an extremely variable environment, characterised by high mortality rates, a heterogeneous prey field, and turbulence at several spatial scales, and so it is not surprising that the number of larvae surviving long enough to be recruited into the adult population is also stochastic. The available stock-recruit data are limited, both in quantity and viability for model fitting; therefore more emphasis should be placed on understanding and studying the biological and dynamical processes underlying the stock-recruitment relationship.

We argue that deterministic models are inappropriate for quantifying recruitment, since the key underlying processes are inherently stochastic. Initial work has extended simple diffusion-based models of larval growth to Lévy jump-diffusion models (representing a patchy prey field), and has examined the consequences for recruitment probabilities [2]. Here we develop a strategic coupled stochastic model of larval-zooplankton (predator-prey) interactions, with the aim of exploring the roles of temporal matching of spawning periods and peak prey abundance [3] on the relationship between stock and recruitment, and recruitment variability in particular.

References

[1] C. Minto, R. A. Myers and W. Blanchard, "Survival variability and population density in fish populations", *Nature*, v. 452, p. 344-348, 2008.

[2] J. F. Burrow, P. D. Baxter and J. W. Pitchford, "Lévy processes, saltatory foraging, and superdiffusion", *MMNP*, v. 3(3), p. 115-130, 2008.

[3] D. H. Cushing, *Marine Ecology and Fisheries*, Cambridge University Press, Cambridge, 1975.

ANALYSIS OF THE FITNESS FOR AN AGGREGATION-FRAGMENTATION EQUATION AND CONSEQUENCES IN THE MODELING OF PRION PROLIFERATION

V. Calvez[a], **M. Doumic Jauffret**[b] and **P. Gabriel**[c]

[a]Unité de Mathématiques Pures et Appliquées, Ecole Normale Supérieure de Lyon
46, allée d'Italie, 69364 Lyon, France
vincent.calvez@umpa.ens-lyon.fr

[b]BANG project-team, INRIA Rocquencourt
Domaine de Voluceau, 78153 Rocquencourt, France
marie.doumic-jauffret@inria.fr

[c]Laboratoire Jacques-Louis Lions, Université Pierre & Marie Curie
175 rue du Chevaleret, 75013 Paris, France
gabriel@ann.jussieu.fr

Polymerization of prion proteins is a central event in the mechanism of prion diseases. The cells produce naturally the normal form of this protein (PrPc) but there exists a pathogenic form (PrPsc) present in the infected cells as polymers. These polymers can grow by attaching the normal proteins, called monomers, and can also split into two smaller polymers. This polymerization process can be modelled by a size-structured PDE containing a growth and a fragmentation term. The growth term depends on the quantity of monomers and the evolution of this quantity is governed by an ODE. The eigenelements of the PDE are very useful for the analysis of the system. In this talk, we investigate the dependency of the first eigenvalue on parameters by using self-similarity arguments (see [4] for instance). Then we give biological interpretations of the results.

A first consequence of the mathematical study is that, for some parameters of the PDE, there may be numerous disease steady states for the system. In this case, we observe that an increase of the production rate of PrPc can stabilize the disease-free steady state.
Another consequence is that, in some cases, there exists an optimal fragmentation intensity which maximizes the fitness of the PDE. This phenomenon has implications in the optimization of a biological protocole called PMCA. This protocole is used to detect the presence of the pathogenic protein PrPsc and could be used to diagnose the prion diseases.
Our analysis can also be seen in a very general way as a study of the qualitative behavior of a general fragmentation and growth process with respect to its parameters.

Aknowledgements This work is partly supported by the ANR grant project TOPPAZ, http://www-roc.inria.fr/bang/TOPPAZ/

References

[1] V. Calvez, M. Doumic Jauffret and P. Gabriel, "Analysis of the fitness for an aggregation-fragmentation equation", *in prepare*.
[2] V. Calvez, N. Lenuzza, D. Oelz, J.-P. Deslys, P. Laurent, F. Mouthon and B. Perthame, "Size distribution dependence of prion aggregates infectivity", *Math. Biosci.*, v. 1, p. 88-99, 2009.
[3] M. Doumic Jauffret and P. Gabriel, "Eigenelements for a general aggregation-fragmentation model", *in press*, june 2010.
[4] M. Escobedo, S. Mischler and M. Rodriguez Ricard, "On self-similarity and stationary problem for fragmentation and coagulation models", *Ann. Inst. H. Poincaré Anal. Non Linéaire*, v. 22, p. 99-125, 2005.

GRADUAL VS PUNCTUATED ANTIGENIC DRIFT FOR INFLUENZA EVOLUTION: A QUANTITATIVE APPROACH BASED ON TIME-SERIES ANALYSIS

A. Camacho[a], **S. Ballesteros**[b], **K. Koelle**[c] and **B. Cazelles**[a,d]

[a]Department of Biology
UMR 7625, UPMC-CNRS-ENS
46 rue d'Ulm 75230 Paris cedex 05, France
camacho@biologie.ens.fr

[c]Department of Biology
Duke University
Box 90338, Durham, NC 27708, USA
katia.koelle@duke.edu

[b]Centro de Matematica e Aplicacoes Fundamentais
Universidade de Lisboa
Lisboa, Portugal
sebastien.ballesteros@gmail.com

[d]UMMISCO
UMI 209 IRD-UPMC
93142 Bondy, France
cazelles@biologie.ens.fr

Influenza is a rapidly evolving virus causing annual winter epidemics and leading to substantial morbidity and mortality in humans. The capacity of influenza to evade immune recognition is mainly attributable to the continual antigenic drift of its surface glycoproteins: hemagglutinin (HA) and neuraminidase. In this work we focus on influenza A/H3N2 which is the dominant circulating type/subtype since 1968 and whose epidemiological dynamics has been widely linked to the evolution of its HA.

While the genetic evolution of A/H3N2's HA is gradual, its antigenic evolution is characterized by discrete clusters that emerge and replace one another within 2 to 8 years [1]. This mechanism, called epochal evolution, is able to qualitatively reproduce times series of influenza incidence as well as the limited genetic diversity of A/H3N2, emphasizing the central role of large and punctuated antigenic evolution [2]. However a recent study argues that both gradual and punctuated antigenic evolution are necessary to effectively reproduce influenza A/H3N2's disease dynamics [3].

We present a new mechanistic model that takes into account both gradual and punctuated antigenic drift. We then address the question in a more quantitative manner by fitting our model to an A/H3N2 incidence time-series over 30 years. Using a rigorous statistical framework [4] to perform maximum likelihood inference and model selection, we propose to:
- Analyse the parameter identifiability of our model regarding its structure and the quality of the data.
- Detect the signal of antigenic cluster replacements in A/H3N2 time-series.
- Quantify the relative importance of gradual versus punctuated antigenic evolution.
- Compare the complete model to the purely gradual or punctuated reduced models.

The question of antigenic drift in influenza A has obvious important applications, particularly for vaccine strain selection. We will therefore discuss our results in a public health policy perspective.

References

[1] DJ. Smith, AS. Lapedes, JC. de Jong, et al. "Mapping the antigenic and genetic evolution of influenza virus", *Science*, v.305 p. 371-6, 2004.
[2] K. Koelle, S. Cobey, B. Grenfell and M. Pascual, "Epochal evolution shapes the phylodynamics of interpandemic influenza A (H3N2) in humans", *Science* v. 314 p. 1898-1903, 2006.
[3] S. Ballesteros, E. Vergu and B. Cazelles, "Influenza A Gradual and Epochal Evolution: Insights from Simple Models", *PLoS ONE* 4(10):e7426. doi:10.1371/journal.pone.0007426, 2009.
[4] EL. Ionides, C. Bretó and AA. King, "Inference for nonlinear dynamical systems", *Proc Natl Acad Sci USA* v. 103 p. 18438-43, 2006.

Epidemic spread in heterogeneous and competitive environment: a spatio-temporal model

B.I. Camara[a] , **V. Caffier**[b] , **F. Didelot**[c] , **B. Le Cam**[d] and **N. Sapoukhina**[e]

Plant Pathology (PaVé) Research unit
French National Institute for Agricultural Research (INRA)
42 rue Georges Morel BP 60057 49071 Beaucouzé
[a]bicamara@angers.inra.fr
[b]Valerie.Caffier@angers.inra.fr
[c]Frederique.Didelot@angers.inra.fr
[d]Bruno.Lecam@angers.inra.fr
[e]natalia.sapoukhina@angers.inra.fr

Abstract
Theoretical and empirical studies show that homogeneous host populations can lead to a severe epidemic while heterogeneity can potentially slow down or prevent epidemic development. Our goal is to study how the intensity of the competition with resident population on the susceptible hosts determines the control conditions of the epidemic spread in the heterogeneous environment.
Thus, we built a reaction-diffusion model describing the epidemic spread. To illustrate our approach, the model was parametrized for the spread of apple scab caused by Venturia inaequalis fungus in two-dimensional apple orchard. To estimate model parameters we used Bayesian method. Numerical simulations showed that weak competition on the susceptible hosts can greatly moderate the restrictive effect of the heterogeneity on the epidemic spread. If the competition is weak, the heterogeneity should be quite pronounced to control the epidemic dynamics. There is a certain threshold for the degree of the heterogeneity above which epidemic goes out of the control.

The results of the model provide insight into our understanding the role of competition in the epidemic control in the heterogeneous environment. The model can be used for designing diversification strategies to control epidemic spread in human populations, agro-ecosystems or forestry.

Key words : reaction-diffusion model, parameter estimation, Bayesian method.

THE ROLE OF STOCHASTICITY IN MODELLING RETINAL ANGIOGENESIS

V. Capasso[a], **G. Facchetti**[b] and **D. Morale**[a],

[a]Department of Mathematics
University of Milano
Via. C. Saldini, 50, 20133 Milano, Italy
daniela.morale,vincenzo.capasso@unimi.it

[b]SISSA
via Beirut, 2-4, 34151 Trieste, Italy
giuseppe.facchetti@sissa.it

Vascularization is an example of complex system: the endothelial cells are the building blocks for the vessels and they interact by regulation signals, forming a network of capillaries in order to reach every part of the body. Here we investigate the case of retinal vascularization, by starting from the experimental evidences mainly presented in the recent review by M. Fruttiger [1]. In a such complex system, an analytical description seems quite impossible and stochastic models seems to be reasonable. Here we discuss the modelling of the dynamics of endothelial cells via a system of N stochastic differential equations

$$dX_N^k(t) = F^k[X_N^1(t), \dots, X_N^N(t)(t), C(X_N^k(t), t)]\, dt + \sigma dW^k(t), \quad k = 1, \dots, N(t)$$

where $\{W_k\}_k$ is a family of independent Wiener processes, $C(x,t)$ is a possible underlying field, e.g. the concentration of a chemical substance, which drives the dynamics of individuals, and $N(t)$ is a counting process, which rate depends on $C(x,t)$. Usually the time scale of the field is much faster than the one of particles, so that a PDE is coupled

$$\frac{\partial}{\partial t} C(x,t) = \triangle C(x,t) + H[X_N^1(t), \dots, X_N^N(t)(t), C(xt)].$$

The high number of individuals makes these totally stochastic models not fully satisfying: computational limitations may heavily affect their efficiency. A possible solution consists in the identification of two different scales in the system, a microscale and a mesoscale: the agents which act in the mesoscale undergo the effects of an average behavior of the microscale agents, deterministically modeled via a Law of Large Numbers [2,3]. So a hybrid model is obtained and the computational cost decreases without a significant lost of the randomness of the whole system.

Here we present a possible model for retinal angiogenesis and by means of numerical simulations, we discuss the role of stochasticity [4].

References

[1] M Fruttiger. Development of retinal vasculature. Angiogenesis, 10, 177–88, 2007.
[2] V. Capasso, D. Morale, Asymptotic Behavior of a System of Stochastic Particles subject To Nonlocal Interactions, Stochastic Analysis and Applications, 27, 574 - 603, 2009.
[3] V. Capasso, D. Morale, Stochastic Modelling of Tumour-induced Angiogenesis, J. Mathematical Biology, 58, 219-33, 2009.
[4] G. Facchetti, Agent Based Methods for Retinal Vascularization: A Stochastic Multiscale Model, (Capasso, Morale Adv.s), Graduate thesis, Milano, 2009.

A SCALE-INVARIANT MODEL OF MARINE POPULATION DYNAMICS

José A. Capitán[b] and **Gustav W. Delius**[a]

[b]Grupo Inderdisciplinar de Sistemas Complejos (GISC)
Department of Mathematics, Universidad Carlos III de Madrid, Spain
jcapitan@math.uc3m.es

[a]Department of Mathematics, University of York, UK
gwd2@york.ac.uk

A striking feature of the marine ecosystem is the regularity in its size spectrum: the abundance of organisms as a function of their weight approximately follows a power law over many orders of magnitude. We interpret this as evidence that the population dynamics in the ocean is approximately scale-invariant. We use this invariance in the construction and solution of a size-structured dynamical population model [1].

Starting from a Markov chain stochastic model encoding the basic processes of predation, reproduction, maintenance respiration and background mortality, we derive a partial integro-differential equation describing the dependence of abundance on weight and time. Our model represents an extension of jump-growth model [3] and hence also of earlier models based on the McKendrick–von Foerster equation. The model is scale-invariant provided the rate functions of the stochastic processes have certain scaling properties.

We study the scale-invariant solutions, in particular the steady-state power law. The exponent of the power law is determined by the relative scaling between the rates of the density-dependent processes (predation) and the rates of the density-independent processes (reproduction, maintenance, mortality). There is a limited region in parameter space that leads to a positive population density.

We perform a linear stability analysis [2] of the steady-state against small perturbations. We find that the inclusion of maintenance respiration in the model has a strong stabilising effect. Furthermore the steady state is unstable against a change in the overall population density unless the reproduction rate exceeds a certain threshold.

We end with a discussion of possible evolutionary mechanisms that could be responsible for the approximate scale-invariance of the population dynamics.

References

[1] José A. Capitán and Gustav W. Delius, “A scale-invariant model of marine population dynamics", arXiv:1003.4674v1 [q-bio.PE]
[2] Samik Datta, Gustav W. Delius, Richard Law and Michael J. Plank, “A stability analysis of the power-law steady state of marine size spectra, arXiv:1001.3826v1 [q-bio.PE]
[3] Samik Datta, Gustav W. Delius and Richard Law, “A jump-growth model for predator-prey dynamics: derivation and application to marine ecosystems”, Bull. Math. Bio. (2010) DOI 10.1007/s11538-009-9496-5

CENTRAL MANIFOLD IN THE UNSTIRRED CHEMOSTAT: HOW LARGE DIFFUSION LEADS TO EXCLUSION.

F.Castella[a],**S.Madec**[b] and **C.Wolf**[c]

[a]Department of Mathematics
IRMAR and IRISA
Campus de Beaulieu - 35042 Rennes Cedex, France
francois.castella@univ-rennes1.fr

[b]Department of Mathematics
IRMAR
Campus de Beaulieu - 35042 Rennes Cedex, France
sten.madec@univ-rennes1.fr

[c]Department of Biology
CAREN, ECOBIO
Campus de Beaulieu 35042 RENNES Cedex, France
cedric.wolf@univ-rennes1.fr

The homogeneous chemostat leads to the well known exclusion principle : only one species can survive (see [1]). We consider an unstirred chemostat for N species in the spatial domain Ω. It reads $\partial_t \mathbf{U} = F(\mathbf{U}, x) + K(\mathbf{U})$, where for each $x \in \Omega$ the system $\partial_t \mathbf{U}(t, x) = F(\mathbf{U}, x)$ is a simple chemostat, and K is a diffusion process. In this case the exclusion principle fails and coexistence occurs. This phenomenon is not well understood.

For large diffusion, the system can be rewritten as $(S_\varepsilon) : \partial_t \mathbf{U} = F(\mathbf{U}, X) + \frac{1}{\varepsilon} K(\mathbf{U})$. The analysis of this system is still complex, but if ε is small, the Central Manifold theorem allows us to write an aggregated system approaching (S_ε) (see [2] and reference therein), of the form $\frac{d}{dt}\widetilde{U} = F_0(\widetilde{U})$. This system is in many cases a homogeneous chemostat, hence it satisfies the exclusion principle.

Our results are as follows.
Firstly, we compute the aggregated system and find the associated best competitor. Moreover, using the error estimate, we show that the problem (S_ε) satisfies the exclusion principle for *any* small enough ε.
Secondly, using bifurcation techniques, we explain how to construct a solution of the stationary N-species system $F(\mathbf{U}, x) + K(\mathbf{U}) = 0$. Extending the result of [3], we focus on the coexistence domain in the space of the consumption parameters (which are our bifurcation parameters). This new coexistence characterisation leads to a better understanding of the general principle : "Large diffusion induces exclusion" .
Finally we show, on a particular case, the importance to consider heterogeneity in the model, even when we are interested in large diffusion only (and consequently close to a homogeneous chemostat). It turns out indeed that a species which is a poor competitor everywhere (*i.e.* for any value of $x \in \Omega$) can be the best competitor for a large diffusion, provided it is the best competitor in the appropriate average meaning.

References

[1] H.L.Smith and P.Waltman, "The theory of the Chemostat", *Cambridge University Press*, (1995)
[2] F.Castella, J.P. Hoffbeck, Y. Lagadeuc, "A reduced model for spatially structured predator-prey systems with fast spatial migrations and slow demographic evolutions", *Asymptot. Anal.* , **61**, (2009), 125-175
[3] J.V.Baxley, and S.B.Robinson, "Coexistence in the unstirred chemosat", *Applied Math. And Comput.*, **39**, (1998), 41-65

DETERMINISTIC ECOLOGICAL MODEL FOR SIMULATING POPULATION DYNAMICS IN MESOCOSMS

C. Catalina[a] and **S. Charles**[b] and **Ph. Ciffroy**[c]

[a]Département LNHE
EDF R&D
6, quai Watier, 78401 Chatou, France
catalina.ciric@edf.fr

[b]CNRS UMR 5558 Laboratoire de Biométrie et Biologie Evolutive "Modélisation et Risques Biologiques"
Université Lyon 1
43 boulevard du 11 novembre 1918, Bât. G. Mendel, Villeurbanne, France
scharles@biomserv.univ-lyon1.fr

[c]Département LNHE
EDF R&D
6, quai Watier, 78401 Chatou, France
philippe.ciffroy@edf.fr

In order to assess potential effects of a chemical substance on ecosystems, extrapolations from single-species effect data are usually used. But this method fails to account for interactions that inevitably exist among the component species. Alternately, modelling at the whole ecosystem level reveals to be a powerful tool both by considering these interactions and by predicting contaminant effects on populations of nontarget species (indirect effects) [1]. In this framework, based on an extensive literature review ([2],[3]), we propose a new compartment ecological model for an aquatic ecosystem. Our compartments include primary producers (macrophytes and algae from phytoplankton and periphyton), primary consumers (juvenile fish and invertebrate grazers, shredders and collectors) and secondary consumers (invertebrate predators and fish). All compartments are related within a food web. Our model also includes the influence of abiotic factors (light, temperature and nutriments) on the different compartments.

We first calibrated and then validated the model on both literature and three experimental data sets coming from INERIS (Verneuil-en-Halatte, France). These data were collected in mesocosms (outdoor artificial streams intended to reproduce a real ecosystem) under control and contaminated conditions. We will present the first results obtained for a non-contaminated ecosystem and its ability to describe and reproduce the dynamics of the different species, as observed in data. We will also show the benefit of a sensitivity analysis to identify key parameters, with the prospect view of adding in the model perturbation functions related to a contaminant.

Keywords : ecosystem modeling, food web, simulations, mesocosms.

References

[1] F. De Laender; K.A.C. De Schamphelaere, P.A. Vanrolleghem, and C.R. Janssen, "Validation of an ecosystem modelling approach as a tool for ecological effect assessments", *Chemosphere*, v. 71, p. 529-545, 2008.
[2] R.A. Park, J.S. Clough, and M. Coombs Wellman, "AQUATOX: Modeling environmental fate and ecological effects in aquatic ecosystems", *Ecological modelling*, v. 213, p. 1-15, 2008.
[3] S.M. Bartell, G. Lefebvre, G. Kaminski, M. Carreau, and K. Rouse Campbell, "An ecosystem model for assessing ecological risks in Québec rivers, lakes, and reservoirs", *Ecological Modelling*, v. 124, p. 43-67, 1999.

A COMPETITIVE MODEL BETWEEN TWO FISHING FLEETS FOR THE SARDINE FISHERY ON THE MOROCCAN ATLANTIC COAST

N. Charouki, **N. Raïssi**, **P. Auger**, **R. Mchich** and **H. Atmani**

Keywords: Fishery model, Sardina pilchardus, Moroccan Atlantic coast, aggregation of variables, equilibrium points, stability, parameters estimation.

This work presents a model describing the dynamics of the sardine (Sardina pilchardus) fishery along the Atlantic Moroccan coast. We assume that the resource is distributed on two isolated zones and targeted by two fleets: The Moroccan coastal purse seiners are fishing along the southern and central coast and can move between these two zones while the deep sea trawlers only operate along the southern zone. The model is a set of ODEs with two components: A resource component describing the stock evolution on the fishing areas and a fleet component governing the dynamics of the fishing effort. Assuming two time scales, we use aggregation techniques to obtain a reduced model. The parameters of the model are then estimated using fitting techniques on the basis of available data time series. We particularly look for conditions leading to a sustainable fishery.

THE ARCHITECTURE OF PREDATOR-PREY FOOD WEBS INCREASES COMPLEXITY AND STABILITY

I. C. Charret[a,c], K. Luz-Burgoa[a,c], Jose A. C. Nogales[a,c], L. D. B. Faria[b,c] e C. A. Paixão[a,c]

[a]Department of Exact Science
Federal University of Lavras
Campus Universitário, P.O.Box 3037, Zip Code 37200-000 Lavras-MG, Brazil
iraziet@dex.ufla.br

[b]Department of Biology
Federal University of Lavras
Campus Universitário, p.o.box 3037, zip code 37200-000 Lavras, Brazil
lucasdbf@gmail.com

[c]Complex System Group of Lavras University

Theoretical studies predict that the stability of an ecosystem is negatively correlated with its complexity, measured by the number of interacting species. On the other hand, empirical evidence indicates that actual food webs are highly interconnected. In this manuscript we present results of our investigation on the stability two-level predator-prey food webs. We analysed exhaustively all possible topologies of connections among species compatible with the predator-prey constraint. Our findings show that those food webs fall into two classes with clearly distinct topologies and stability properties. In one of them stability is negatively correlated with complexity, according to earlier theoretical predicitons. The other group, characterized by a nested topological structure, is composed of highly interconnected, stable food webs. The stability within this group is independent of the number of species, number of connections and strengths of interactions. We review empirical evidence that corroborates our results.

Bit-String Model of Dengue Fever Including Dynamic of Virus, Vector and Human Population

I. C. Charret[a,b] and C. A. Paixão[a,b]

[a]Department of Exact Science
Federal University of Lavras
Campus Universitário, P.O.Box 3037, Zip Code 37200-000 Lavras-MG, Brazil
iraziet@dex.ufla.br
crysttianarantes@posgrad.ufla.br

[b]Complex System Group of Lavras University

Dengue fever is a disease transmitted by mosquitoes in tropical and subtropical areas around the world. In terms of the number of human infections occurring globally, dengue fever is considered to be the most important arthropod-borne viral disease in humans; currently, more than 2.5 billion people live in high risk areas of dengue fever[1]. Monitoring and predicting dengue incidence facilitates early public health responses to minimize morbidity and mortality of human population.

In this work we proposed a computational model to simulate the dengue fever spreading, including the life cycle of the vector, *Aedes aegypti* mosquitoes, the human population and the four types of virus serotypes (DEN-1, DEN-2, DEN-3, and DEN-4) of the genus Flavivirus[2], using one modified model based in bit-string thecnics.[3] Our model try to capture the essential features of the epidemiological cycle of the dengue fever and the main characteristics of the transmission of disease. It is done as follows.

The mosquitoes population are subdivided in four fases, the stage eggs, the larval, the pupation and the adult stage. The simulation begin with a certain initial quantity of eggs located in some spatial site. After a certain period of time, the larvae are born. Each site space possess a certain capacity to sustain the pupae. This introduces a competition at this stage, as expected experimentally. Finally born mosquitoes in their adulthood, not yet infected, and divided into males and females. Each individual receives a bit-string that contains all information relevant to the simulation. At this stage, mosquitoes can fly within a certain region, mating and reach new sites. And the cycle repeats. As adults, mosquitoes can be infected by one of 4 types of dengue virus if they find along their way, a human infected. Thus, the disease is introduced into the mosquito population and can spread through the environment. The viral population will also receive a bit-string with their characteristics and may suffer, throughout their life cycle, mutations, represented by changes in this bit-string. This model is being implemented and the first results are being obtained.

1 D. J. Gubler, Arch. Med. Res., 33, 2002.
2 H. M. Yang and C. P. Ferrreira, Applied Math. and Computation 198, 2008.
3 T. J. P. Penna, J. Statistical Physics, 78 (5), 1995.

BLUETONGUE SPREAD AND VACCINATION STRATEGIES IN CATTLE HERDS

M. Charron[a], **M. Langlais**[b], **P. Ezanno**[c] and **H. Seegers**[d]

[a]INRA, Oniris, UMR1300 BioEpAR, BP 40706, F-44307 Nantes, France
Université Victor Segalen, Bordeaux 2, UMR CNRS 5251, IMB & INRIA Bordeaux Sud-Ouest, France
maud.charron@oniris-nantes.fr

[b]Université Victor Segalen, Bordeaux 2, UMR CNRS 5251, IMB & INRIA Bordeaux Sud-Ouest, France
3ter Place de la Victoire 33 076 Bordeaux Cedex, France
michel.langlais@u-bordeaux2.fr

[c]INRA, Oniris, UMR1300 BioEpAR, BP 40706, F-44307 Nantes, France
henri.seegers@oniris-nantes.fr

[d]INRA, Oniris, UMR1300 BioEpAR, BP 40706, F-44307 Nantes, France
pauline.ezanno@oniris-nantes.frpauline.ezanno@oniris-nantes.fr

Since the emergence in 2006 of serotype 8 of Bluetongue virus (BTV8) in Northern Europe, infected cattle may develop important clinical signs. Therefore, the current outbreak has a large socioeconomic impact on the international trade of animals and their productivity. Mass vaccination of cattle was thus imposed in Europe. However, alternatives to this mass vaccination could limit generated costs for states and farmers. Our objective is to estimate the spread of this vector-borne disease in a homogeneous population of cattle according to the vaccination strategy used.

The virus is transmitted by biting midges named *Culicoides*. We develop an epidemiological model taking into account both hosts (cattle) and vectors (midges). Hosts can be of seven health states: susceptible, protected ($S0$) or not (S) by maternal antibodies, infectious (I), recovered (R), carrying an infected foetus (infectious (IB) and recovered (RB)) and vaccinated (V). Vertical (in utero) and pseudo-vertical (at birth) transmission can thus be accounted for. Here, only female cattle are considered, breeding occurring by artificial insemination. Vectors can be of three health states: susceptible (MS), latent (ME) and infectious (MI). The vaccination strategies are defined by two model parameters: the vaccine efficacy and coverage.

To ensure the model relevance, we quantify the stability of the disease-free equilibrium. We formulate analytically the basic reproduction number ($R0$) of the model. If $R0$ is higher than 1, the virus can spread in the population. If it is lower than 1, the epidemic fades out. By simulation, we determine coverage and efficacy values for which $R0$ decreases under the critical threshold of 1. We found that if the vaccine efficacy is 95%, to get $R0 < 1$, at least 51% of the cattle population needs to be vaccinated. For a lower efficacy, it is hardly possible to reduce $R0$ below 1 whereas for a better efficacy, an even lower coverage is sufficient to control the epidemics. A partial vaccination is therefore adequate to control BTV8 spread in a cattle population as long as vaccine efficacy is large enough. Our conclusion should be verified in a heterogeneous population in relation with spatial cattle population structure, presence of reservoir or other host species, and seasonal vector population.

SENSIVITY OF PREDATOR-PREY DYNAMICS TO FUNCTIONAL RESPONSES IN A CHEMOSTAT

F. Cordoleani[a], **D. Nerini**[a], **M. Gauduchon**[a] and **J.-C. Poggiale**[a]

[a]Centre d'Oceanologie de Marseille
Université de la Méditerranée, UMR LMGEM 6117 CNRS
Campus de Luminy, Case 901
13288 Marseille Cedex 09
flora.cordoleani@univmed.fr
david.nerini@univmed.fr
mathias.gauduchon@univmed.fr
jean-christophe.poggiale@univmed.fr

Keywords: Predator-prey model in chemostat, functional response formulation, Hausdorff distance.

The dynamics of heterotrophic bacteria within the microbial loop is very complex and depends particularly on the predation by protozoan species. We investigate the importance of the formulation of this trophic interaction on the microbial dynamics through the analysis of a bi-trophic microbial model in chemostat. Recently, many papers have reported the importance of the functional response choice on the dynamics of predator-prey systems. These results are obtained on the Rosenzweig-McArthur model, and we extended them in a chemostat model.

We first provide the analysis of the model with a general formulation of the functional response. This allows us to generalize common results about predator-prey dynamics to models in chemostat. In a second part, the chemostat model is simulated by using different functional responses. We show that even for a class of functions that have very similar shape, different quantitative and qualitative population dynamics can be observed. For a more quantitative purpose, we developed a method of sensitivity analyses by using the Hausdorff distance on the omega limits in the phase space. We compared the sensitivity to the functional response parameters and the sensitivity to the functional response mathematical expression.

It appears that the choice of the functional response formulation is of great importance and may have a stronger impact on the model dynamics than variations of parameter values. Hence, it is recommended to improve the translation of our knowledge on microbial predation into mathematical terms, in order to provide correct trophic interactions models within biological systems.

MULTISCALE MODELLING OF RED BLOOD CELL PRODUCTION: INTRACELLULAR REGULATION PATHWAYS AND STRUCTURED POPULATION DYNAMICS

F. Crauste[a]

[a]Institut Camille Jordan UMR 5208
Université Claude Bernard Lyon 1
43 boulevard du 11 novembre 1918
69622 Villeurbanne cedex, France
crauste@math.univ-lyon1.fr

This presentation will be devoted to multiscale mathematical modelling of erythropoiesis, the process of production and regulation of red blood cells. It lies upon works published recently [1, 2, 3], in collaboration with Ivan Demin, Olivier Gandrillon, Stephane Genieys, Clement Molina, Laurent Pujo-Menjouet and Vitaly Volpert (Université Lyon 1, France).

Erythropoiesis is a complex process, involving cells with different maturities, from very immature stem cells to circulating mature red blood cells. It is regulated both at the intracellular level and at the cell population scale. We propose a new multiscale model of erythropoiesis [1, 3], in which we describe together erythroid progenitor (immature red cells) dynamics and intracellular regulatory network that determines erythroid cell fate. The intracellular regulation model is based on several proteins inhibiting and activating one an other, under external actions of growth factors that influence their production. The levels of these proteins will decide of cell self-renewal, differentiation or death by apoptosis. Erythroid progenitors dynamics are described with structured models, either compartmental models (systems of ordinary differential equations) or partial differential equations [2]. In both cases, nonlinearities are considered in the models to account for cell fate regulation.

Analysis of the models is performed and computer simulations are carried out to confront the models to stress situations of anemia (blood loss). The results are compared to experimental data on induced anaemia in mice. This allows concluding on the roles of the different feedback controls incorporated in the model, acting both at the intracellular and at the cell population levels, in order to provide more insights into the regulation of erythropoiesis.

Keywords: Multiscale model, erythropoiesis, regulation pathways, maturity structured models.

References

[1] F. Crauste, I. Demin, O. Gandrillon, and V. Volpert, "Mathematical study of feedback control roles and relevance in stress erythropoiesis", *Journal of Theoretical Biology*, v. 263 (3), p. 303-316, 2010.
[2] F. Crauste, L. Pujo-Menjouet, S. Génieys, C. Molina, and O. Gandrillon, "Adding Self-Renewal in Committed Erythroid Progenitors Improves the Biological Relevance of a Mathematical Model of Erythropoiesis", *Journal of Theoretical Biology*, v. 250, p. 322-338, 2008.
[3] I. Demin, F. Crauste, O. Gandrillon, and V. Volpert, "A multi-scale model of erythropoiesis", *Journal of Biological Dynamics*, v. 4 (1), p. 59-70, 2010.

EFFECT OF VECTOR DISPERSAL AND MIGRATION ON SYLVATIC TRANSMISSION OF *TRYPANOSOMA CRUZI*

A. Britnee Crawford[a] and **B. Christopher Kribs Zaleta**[b]

[a]Department of Mathematics
A-University of Texas at Arlington
A-Arlington, TX 76019-0408 USA
britnee18@msn.com

[b]Department of Mathematics
B-University of Texas at Arlington
B-Arlington, TX 76019-0408 USA
kribs@uta.edu

T. cruzi is a parasite commonly found in many mammalian species in North and South America, transmitted by triatomine insect vectors. Although there have been several mathematical studies regarding the spread of *T. cruzi* to humans, few studies have considered the sylvatic transmission cycles in which the parasite is maintained in North America. Understanding the ecology of these overlapping cycles is crucial to assessing the potential spread of the parasite, as vector dispersal may bring *T. cruzi* to regions in which it is not currently endemic.

The metapopulation model presented is a system of ordinary differential equations representing *T. cruzi* transmission between several hosts and vectors in 3 different regions (or patches). The primary focus is on vector migration as a connection between transmission cycles. Vector migration and dispersal rates across patch boundaries depend on the size of the patch, boundary length, and vector dispersal distance. We investigate the effects of migration rates on the spread of infection among vectors and hosts in 3 patches.

MULTISPECIES INTERACTIONS IN WEST NILE VIRUS

G. Cruz-Pacheco, **L. Esteva** and **C. Vargas**

We analyze the interaction of different species of birds and mosquitoes on the dynamics of West Nile Infection. We found that the basic reproductive number is the weighted mean of the of the basic reproductive number of each species weighted by the relative abundance of its population in the location.

Modeling cell-cell adhesion with a cadherin based model

J. C. Dallon[a],**Elijah Newren**[b], and **Marc D. H. Hansen**[c]

[a]Department of Mathematics
Brigham Young University
Provo, UT 84602-6539
dallon@math.byu.edu

[b]Sandia National Laboratories
Department 1541
Albuquerque, NM

[c]Physiology and Developmental Biology
Brigham Young University
Provo, UT 84602-6539

This talk will describe a model of cell-cell adhesion based on cadherin force interactions. Each cell has a population of cadherins which can move in the cell membrane and interact with cadherins from another cell. Using the immersed boundary method, the cell membrane and cortex is modeled. The model is used to help determine what role the actin cytoskeleton plays in cell-cell adhesion.

EVOLUTION OF THE MOSQUITO POPULATION DURING A DENGUE EPIDEMIC IN HAVANA

H. de Arazoza[a], **R. Lounes**[b], **J. Barrios**[a], **E. Garcia**[c], **M. Atencia**[c] and **G. Joya**[c]

[a]Department of Mathematics
University of Havana
San Lazaro y L, Ciudad de La Habana, Cuba
arazoza@matcom.uh.cu ; asp@matcom.uh.cu

[b]Laboratoire MAP5, UMR-CNRS 8145
University Paris Descartes
45 rue des Saints-Pères, 75270 Paris CEDEX 06, France
lounes@math-info.univ-paris5.fr

[c]Department of Tecnología Electrónica,
University of Málaga
Campus de Teatinos, 29071 Málaga, Spain
gjoya@uma.es

Keywords: Dengue epidemic, epidemic control, deterministic model.

ABSTRACT:
Dengue fever is a disease transmitted through mosquitoes. In Cuba the transmission vector is the Aedes Aegipty mosquito. Dengue fever is endemic in many areas in Central America and the Caribbean, but in Cuba it is always an imported disease, it is not endemic. One of the problems confronted by the Health System is finding the size of the mosquito population during an outbreak of dengue fever. There are indices studied by the surveillance system, and there are indicators that point to a possible outbreak, if a case is introduced by some traveler that is unaware of its condition as a carrier of one of the dengue viruses.
Since the first papers [1] modeling dengue fever has been a priority in the countries where it is endemic.In our work using a mathematical model with ODE adapted from [2], we fit the epidemic curve given by the model to the known cases of a dengue fever outbreak in the city of Havana, using as parameters the carrying capacity for mosquito eggs, larvae and pupa. This carrying capacity depends on the actions the Health System takes to eliminate areas where the mosquito can breed.
Once we have the parameters that fit the part of the full blown outbreak, we use these values to estimate the efficiency of the system in eliminating the mosquito and its breeding grounds. We present these results using the same epidemic outbreak in the city of Havana.

References
[1] L. Esteva and C. Vargas, "A model for dengue disease with variable human population", *J. Math. Biol.*, v. 38, p. 220-240, 1999.
[2] F.A.B. Coutinho and M.N. Burattini and L.F. Lopez and E. Massad, "Threshold conditions for a non-autonomous epidemic system describing the population dynamics of dengue", *Bull. Math. Biol*, v. 68, p. 2263-2282, 2006.

ANALYSING IMMUNE CELL MIGRATION

Rob J. De Boer[a], **Joost B. Beltman**[a] and **Stan F. Marée**[b]

[a]Theoretical Biology
Utrecht University
Padualaan 8, 3584 CH Utrecht, The Netherlands
r.j.deboer@uu.nl, j.b.beltman@uu.nl

[b]Computational and Systems Biology
John Innes Centre
Norwich Research Park, Colney, Norwich NR4 7UH, UK
stan.maree@bbsrc.ac.uk

The visualization of the dynamic behaviour of and interactions between immune cells using time-lapse video microscopy has an important role in modern immunology. To draw robust conclusions, quantification of such cell migration is required. This is far from trivial because imaging experiments are associated with various artefacts that can affect the estimated positions of the immune cells under analysis, which form the basis of any subsequent analysis. We construct spatially explicit models of T cell and DC migration in LNs and show that several dynamical properties of T cells are a consequence of the densely packed LN environment. Our three-dimensional simulations suggest that the initial decrease in T-cell motility after antigen appearance is due to "stop signals" transmitted by activated DCs to T cells. Because imaging is typically restricted to experiments lasting 1 h, and because T cell-DC conjugates frequently move into and out of the imaged volume, it is difficult to estimate the true duration of interactions from contact data. We propose a method to properly make such an estimate of the average of the contact durations. The method is validated by testing it to our spatially explicit computer simulations.

References

[1] Beltman J.B., Marée A.F. and De Boer R.J. "Analysing immune cell migration" *Nat. Rev. Immunol.*, 9: 789-798, 2009.

[2] Beltman J.B., Henrickson S.E., Von Andrian U.H., De Boer R.J. and Marée A.F. "Towards estimating the true duration of dendritic cell interactions with T cells" *J. Immunol. Methods*, 54: 54-69, 2009.

[3] Beltman J.B., Marée A.F. and De Boer R.J. "Spatial modelling of brief and long interactions between T cells and dendritic cells" *Immunol. Cell. Biol.*, 85: 306-314, 2007.

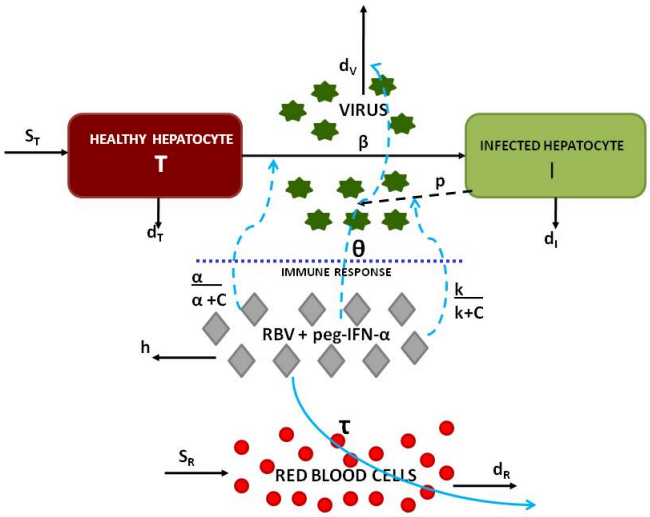
[4] Beltman J.B., Marée A.F., Lynch J.N., Miller M.J. and De Boer R.J. "Lymph node topology dictates T cell migration behavior" *J. Exp. Med.*, 204: 771-780, 2007

Evaluating Treatment of Hepatitis C for Hemolytic Anemia Management

Swati DebRoy

Department of Mathematics
University of Florida
358 Little Hall PO Box 118105 Gainesville, Fl-32611
swati@ufl.edu

The combination therapy of antiviral peg-interferon and ribavirin has evolved as one of the better treatments for Hepatitis-C. In spite of its success in controlling Hepatitis-C infection, it has also been associated with treatment-related adverse side effects. The most common and life threatening among them is hemolytic anemia, necessitating dose reduction or therapy cessation. The presence of this side effect leads to a trade-off between continuing the treatment and exacerbating the side-effects versus decreasing dosage to relieve severe side-effects while allowing the disease to progress. The drug epoietin (EPO) is often administered to stimulate the production of red blood cells (RBC) in the bone marrow, in order to allow treatment without anemia. This paper uses mathematical models to study the effect of combination therapy in light of anemia. In order to achieve



this we introduce RBC concentration and amount of drug in the body as state variables in the usual immunological virus infection model. The compartmental model can be seen in the adjoining figure. Analysis of this model provides a quantification of the amount of drug a body can tolerate without succumbing to hemolytic anemia. Indirect estimation of parameters allow us to calculate the necessary increment in RBC production to be ≥ 2.3 times the patient's original RBC production rate to sustain the entire course of treatment without encountering anemia in a sensitive patient.

Keywords: Hepatitis-C, hemolytic anemia, mathematical modeling.

References

[1] Swati DebRoy, Christopher Kribs-Zaleta, Anuj Mubayi, Gloriell M.Cardona-Melendez, Liana Medina-Rios, MinJun Kang and Edgar Diaz. Evaluating Treatment of Hepatitis C for Hemolytic Anemia Management. Accepted in *Mathematical Biosciences.*

A MATHEMATICAL MODEL FOR BACTERIOCIN PRODUCTION REGULATED BY QUORUM SENSING

Roberta Regina Delboni[a] and **Hyun Mo Yang**[b]

[a,b]Department of Applied Mathematics
Institute of Mathematics, Statistics and Scientific Computation - UNICAMP
Rua Sérgio Buarque de Holanda, 651; CEP 13083-859, Campinas, SP. Brasil.
[a]rodelboni@ime.unicamp.br
[b]hyunyang@ime.unicamp.br

Bacteriocins produced by lactic acid bacteria are defined as extracellularly primary or modified products of bacterial ribosomal synthesis, which can inhibit or kill pathogenic bacteria such as *Listeria monocytogenes*, *Staphylococcus aureus* and *Clostridium botulinum*. For instance, nisin is a bacteriocin produced by *Lactococcus lactis* and widely used in the food industry as a safe and natural preservative.

In this work, the cell-density-dependent regulation of bacteriocin production is studied, focusing on the bacteriocin nisin. This phenomenon, called quorum sensing, involves specific molecules that are directly sensed by membrane-located histidine kinases, after which the signal is transmitted to an intracellular response regulator that activates transcription of target genes.

These molecules that act as signals, accumulate in the environment as the cell density increases and activate signal transduction cascades that result in production of the autoinducer by the stimulated bacteria cell. Besides it function as antimicrobial peptide, the nisin exhibit a peptide pheromone function, that plays an essential role in quorum sensing control of it biosynthesis.

Through the knowledge of the bacteriocin biosynthesis biochemical regulation, we developed a mathematical model focusing on the production of bacteriocin nisin. This model include synthesis of nisin in order to study the dynamic of the regulatory system in a growing bacterial population. This model demonstrate a typical behavior of hysteresis. Using this model, we have shown that quorum sensing works because of a switch between two stable steady solutions, one with low levels of autoinducer and one with high levels of autoinducer.

Keywords: mathematical model, dynamical system, quorum sensing, hysteresis.

References

[1] J. D. Dockery, J. P. Keener. "A mathematical model for quorum sensing in *Pseudomonas aeruginosa*", *Bulletin of Mathematical Biology* **63**, 95-116 (2001).

[2] D. Karlsson, *et al.*. "Modeling the regulation of the competence-evoking quorum sensing network in *Streptococcus pneumoniae*", *BioSystems* **90**, 211-223 (2007).

[3] M. Kleerebezem, "Quorum sensing control of lantibiotic production: nisin and subtilin autoregulate their own biosynthesis", *Peptides* **25**, 1405-1414 (2004).

[4] O. P. Kuipers, P. G. G. A de Ruyster, M. Kleerebezem, W. M. de Vos, "Quorum sensing controlled gene expression in lactic acid bacteria", *Journal of Biotechnology* **64**, 15-21 (1998).

[5] L. E. N. Quadri, "Regulation of antimicrobial peptide production by autoinducer-mediated quorum sensing in lactic acid bacteria", *Antonie van Leeuwenhoek* **82**, 133-145 (2002).

Competitive exclusion principle for SIS and SIR models with n strains

B. Derdei[a] and **A. Iggidr**[a] and **G. Sallet**[a,b]

[a]INRIA Project MASAIE
INRIA-Nancy Grand Est & Université Paul Verlaine, Metz
I.S.G.M.P. Bât A, Ile du Saulcy, 57045 Metz Cedex 01, France.
bicharad@loria.fr; iggidr@loria.fr; sallet@loria.fr

[b]UMI 209, UMMISCO
IRD
LANI, université Gaston Berger, Saint-Louis, Sénégal

Key words: Nonlinear dynamical systems, global stability, Lyapunov methods, disease free equilibrium, boundaries equilibrium.

We consider SIS and SIR models with standard mass action and varying population, with n different strains of an infectious disease. We also consider the same models with vertical transmission. We prove that under generic conditions a competitive exclusion principle holds. For each strain a basic reproduction ratio can be defined, it corresponds to the case where only this strain exists. The basic reproduction ratio of the complete system is the maximum of each individual basic reproduction ratio.

Actually we also define an equivalent threshold for each strain. The winner of the competition is the strain with the maximum threshold. It turns out that this strain is the most virulent, i.e., the strain for which the endemic equilibrium gives the maximum population for the strain. This can be interpreted like a pessimization principle (O. Diekmann). This kind of results has already been obtained for SIR models [1]. But in this reference the winner strain maximizes its basic reproduction ratio. This is not our case. The same kind of remark applies to [2].

References

[1] H.J. Bremermann, and H. Thieme, “A competitive exclusion principle for pathogen virulence", *J. Math. Biol.*, v. 27, p. 179-190, 1989.

[2] C. Castillo-Chavez, and W. Huang, and J. Li, “Competitive exclusion and coexistence of multiple strains virulence and SIS STD model", *SIAM J. Math.*, v. 59 (5), p. 1790-1811, 1999.

On a mathematical model of Typha proliferation : equilibria and stability

Mamadou Lamine DIAGNE[ac] - **Papa Ibrahima NDIAYE**[b] and **Tefwik SARI**[c]

[a] UFR SAT, UMMISCO-Saint-Louis, LANI-UGB
BP 234, Saint-Louis, Sénégal
diagne12@yahoo.fr

[b] UFR SATIC , Université de Bambey, UMMISCO-Saint-Louis,
BP 1179, Thiés, Senegal
papaibra@yahoo.com

[c] Université de Haute Alsace, 4 rue des frère Lumière, 68093 Mulhouse, France
UMR MISTEA, SupAgro Bât 21, 2, place Pierre Viala, 34060 Montpellier, France
Tewfik.Sari@uha.fr

keywords : Growth dynamics, stability, hybrid system, Typha

The Typha is freshwater plant that grows under modest ecohydrological conditions. Its proliferation is possible by two modes of reproduction which can take place simultaneously: sexual reproduction from seed and vegetative multiplication assured by rhizomes (roots). The presence of Typha in the delta of the Senegal River contributes the economy of local people [1]. It is heavily marketed for use in the manufacture of mats, strand matches, houses, fences, etc..
But, strong proliferation of Typha in PNOD has had adverse consequences on the biodiversity, economy and health of local populations by the obstruction access to water. For example, it interferes with fishing activities, reduces production agricultural and promotes the persistence of some waterborne diseases such as bilharzia and malaria [2].
The control methods used against the cattail in the park including burning, the release of chemicals, changing the level of immersion and faucardage had variable effectiveness, often limited in time and sometimes very expensive. Thus, UNESCO has proposed a multidisciplinary study begun in 2007 which aim is to control typha proliferation with an eco-hydrological approach.
Ecohydrology is a new concept in the field of environmental sciences whose purpose is to promote the integration of hydrology and ecology at service of sustainable management of water resources. It is based on the assumption that sustainable development of water resources is dependent on the ability to maintain evolutionary processes existing flow of water, nutrients and energy flow in the basin scale, through an integrated regulatory processes biological, hydrological, ... processes as management tools [2].
In this work, we study properties of the non autonomous typha proliferation mathematical model developed in [1] in order to contribute to the Ecohydrology control approach. In fact, we analyze the existence of equilibria and the stability of mathematical models obtained by considering the four combinations corresponding to those mechanisms of reproduction. We also scrutinize the behaviour of the hybrid or cross-bred system corresponding to the alternation of periods with a sexed reproduction or without that form of reproduction.

References

[1] M.L. DIAGNE, P. I. N'DIAYE, P.D. HANNE, K. NOBA, M. T. NIANE, Modeling the spread of Typha in the vicinity of a hydraulic structure, *En preparation*.
[2] N.L. SARR, Aspects socio-économiques de la prolifération de Typha domingensis dans le delta du fleuve Sénégal. *Mémoire de DEA., ISE, UCAD Dakar, 49 p, 2003*.

INFLUENCE ON SOME CONTINUOUS STRUCTURING VARIABLES ON SPATIAL INVASION

A. Ducrot

IMB & INRIA Bordeaux Sud-Ouset ANUBIS
Université Victor Segalen Bordeaux 2
Bordeaux, France
arnaud.ducrot@u-bordeaux2.fr

Keywords: Travelling waves, age and size structured models, pattern propagation, epidemiology, ecology.

The ability of reaction-diffusion systems to generate travelling wave solutions is known since the pioneer works of Fisher and of Kolmogorov, Petrovski and Piskunov in the 30's. On the other hand, in population dynamics modelling (epidemiology or ecology for instance), continuous structuring variables such as the age of individuals, the age since infection or the size of individuals have been extensively used in order to have a better description of the interactions between individuals and populations.

In this talk, we will discuss how such continuous structuring variables may influence the spatial spread of some populations. Here we will focus on two examples, the age since infection for the spatial spread of some infectious diseases and on the influence of the size or age of individuals in the context of species invasions.

A Numerical Solver for the Population Density Function of Neural Networks.

G. Dumont[a] and **J. Henry**[b]

[a]IMB, INRIA Bordeaux sud-ouest
UFR Sciences et Modélisation, Université Victor Segalen Bordeaux 2
3 ter place de la Victoire
33076 Bordeaux Cedex
gregory.dumont@inria.fr

[b]INRIA Bordeaux sud-ouest, IMB
UFR Sciences et Modélisation, Université Victor Segalen Bordeaux 2
3 ter place de la Victoire
33076 Bordeaux Cedex
jacques.henry@inria.fr

To describe the behavior of a large population of neurons interacting in a neural network, a population density model was developed in [6] based on conservation laws. Where the particularities of the all population are directly derived from the particularies of a single neuron,Each neuron of the population is assumed to be described by the same model, in this case the Izhikevich model introduced in [1]. The evolution in time of the self coupled population is determined by a non-linear partial differential equation (PDE).

In this work, we present a finite volume method to approximate the population density equation. We compare the numerical results with a Monte Carlo simulation. Such Monte Carlo methods are easier to implement but suffer from convergence difficulties.

References

1. Eugene M. Izhikevich, Dynamical Systems in Neuroscience, The MIT Press, 2007
2. Randall J. LeVeque, Numerical Methods for Conservation Laws, Birkhauser Verlag, 1992
3. Bopeng Rao, Théorie et Approximation des systèmes de Loi de Conservation, UFR de Mathématique et d'Informatique de l'Université de Strasbourg
4. Randall J. LeVeque, Finite Volume Methods for Hyperbolic Problems, Cambridge university press, 2002
5. Fabien Marpeau, Aditya Barua, Kresimir Josic, A finite volume method for stochastic integrate and fire models, ournal of Inegrative Neuroscience, 2008
6. A. Omurtag, B.W. Knight, L. Sirovich, On the simulation of large populations of neurons : J Computational Neuroscience, 2000
7. Chi-Wang Shu Essentially non-oscillatory and weighted essentially non-oscillatory schemes for hyperbolic conservation laws : NASA, 1997

INTRAGUILD PREDATION AND CONSERVATION OF ENDANGERED SEABIRDS. MODELLING, THEORY AND NONSTANDARD APPROXIMATIONS.

Y. Dumont[a] and **J.C. Russell**[b]

[a]CIRAD
Umr AMAP
Montpellier, France
yves.dumont@cirad.fr

[b]Dpt of Environmental Science, Policy and Management
University of California
Berkeley, USA
jrussell@berkeley.edu

Keywords: population dynamics, modeling, intraguild predation, prey, mesopredator, superpredator, equilibrium, stability/unstability, nonstandard finite difference method, dynamic consistency, numerical simulation

Seabirds breeding on islands are vulnerable to introduced predators, such as rats and cats, and the removal of such predators is generally viewed as a priority for seabird conservation and restoration. However, multiple invasive mammal species interacting may generate unexpected outcomes following the removal (eradication) of one species. Generally these indirect interactions are not well understood or demonstrated.

We propose and study a prey (seabird)-mesopredator (rat)-superpredator (cat) model, taking into account the juvenile stages in the prey population, in order to direct conservation management for seabird conservation [4,5]. We give a more biologically realistic differential system than those studied before [2,3], in particular for long-lived seabird species.

We present a theoretical study and show existence and uniqueness of a positive solution as well as a qualitative study of the equilibria that may appear [5]. Because standard numerical methods, usually implemented in scientific softwares, like Scilab or Matlab, can fail to give the right biological approximations [1,5], we propose a reliable algorithm that preserves most of the qualitative properties of the continuous system, using the theory of nonstandard finite difference methods. We illustrate our approach with Barau's Petrel, an endemic seabird from Réunion Island.

References

[1] R. Anguelov, Y. Dumont, J.M-S. Lubuma and M. Shillor," Dynamically consistent nonstandard finite difference schemes for epidemiological models", submitted.
[2] F. Courchamp, M. Langlais and G. Sugihara, "Cats protecting birds: modelling the mesopredator release effect." Journal of Animal Ecology v. 68, p. 282-292, 1999.
[3] M. Fan, Y. Kuang and Z. Feng, "Cats protecting birds revisited", Bulletin of Mathematical Biology v. 67, p. 1081-1106, 2005.
[4] J.C. Russel, V. Lecomte, Y. Dumont, and M. Le Corre, "Intraguild predation and mesopredator release effect on long-lived prey", Ecological modelling v. 220, p. 1098-1104, 2009.
[5] Y. Dumont, J.C. Russel, V. Lecomte, and M. Le Corre, "Conservation of endangered endemic seabirds within a multi-predator context: the Barau's Petrel in Réunion Island", to appear in Natural Resource Modelling.

BIOLOGICAL VECTOR CONTROL WITH THE STERILE INSECT TECHNIQUE FOR THE CHIKUNGUNYA DISEASE

Y. Dumont[a] and **J.M. Tchuenche**[b]

[a]CIRAD
Umr AMAP
Montpellier, France
yves.dumontcirad.fr

[b]Department of Mathematics and Statistics
University of Guelph
ON N1G 2W1 Canada
jtchuenc@uoguelph.ca

Keywords: Vector-control, pulse control, Sterile Insect Technique, Mathematical Epidemiology, Analysis, nonstandard finite difference scheme, numerical simulations.

Chikungunya is a vector-borne Disease, usually localized in Asia and East-Africa, with *Aedes albopictus* mosquito as the principal vector for the Chikungunya virus. In 2005 and 2006, Réunion Island faced two epidemics of Chikungunya: the 2006's epidemic was particularly dramatic. This was the first time that a developed country, like Réunion Island, was affected by this virus. In July 2007, a small outbreak occurred in Italy, indicating that the South of Europe is potentialy threatened.
In recent works [1,2], we proposed and studied a mathematical model to explain the outbreak of 2005 and possible links with the explosive epidemic of 2006. These studies specifically focus on the comparison of different mosquito control tools (adulticide, larvicide, and mechanical control) in order to know if it would have been possible to contain or to completely avert the 2006 epidemic. We showed that the combination of the three control tools (with a suitable period of release and a sufficient duration of the treatment) would have been useful to control the explosive epidemic of 2006 [2].
As far as we know, *Aedes albopictus* in Réunion Island is yet sensitive to Deltamethrin, the only authorized adulticide, but can become resistant, like in Martinique, a West Indies French Island. Moreover, Réunion Island is a hot spot of endemicity and, thus, the use of chemical control tools can be limited. It is also necessary to study and to check the feasibility of other vector control tools such as the Sterile Insect Technique (SIT). To this effect, a project called TIS (Technique d'Insecte Stérile), funded by the French Ministry of Health, the European Union and the Regional Council is ongoing in Réunion Island.

The aim of this talk is to give a short introduction to the TIS project and to present some recent mathematical results related to the SIT-LSIR model considered for the Chikungunya disease. Moreover, because mechanical control (destruction of breeding sites) is a very cheap and sustainable alternative, we combine mechanical control and SIT control. We present several numerical simulations to assess the efficacy of the SIT vector-control in comparison with the Chemical vector control, studied in [2]. We show that SIT (impulse) control could be useful to control the wild mosquito population and thus lower the risk of an epidemic.

References

[1] Y. Dumont, F. Chiroleu and C. Domerg, "On a temporal model for the Chikungunya disease: modelling, theory and numerics", *Math. Biosc.* , v. 213, p. 80-91, 2008.
[2] Y. Dumont, and F. Chiroleu, "Vector-control for the Chikungunya disease", *Math. Biosc. Eng.* , v. 7 (2), p. 315-348, 2010.

A NON-LOCAL CELL ADHESION MODEL IN N-DIMENSIONAL SPACE WITH CELL AGE

J. Dyson[a], **S. A. Gourley**[b], **R. Villella-Bressan**[c] and **G. F. Webb**[d]

[a]Mansfield College,
University of Oxford,
Oxford, OX1 3TF, UK
janet.dyson@mansfield.ox.ac.uk

[b]Department of Mathematics,
University of Surrey,
Guildford, Surrey, GU2 7XH, UK
s.gourley@surrey.ac.uk

[c]Dipartimento di Matematica Pura e Applicata,
Università di Padova,
Padova, Italy
rosannav@math.unipd.it

[d]Department of Mathematics,
Vanderbilt University,
Nashville, Tennessee, USA
glenn.f.webb@vanderbilt.edu

Keywords: non-local, adhesion, reaction-diffusion, analytic semigroup, fractional power, existence, regu- larity, positivity.

A model for cell-cell adhesion, based on a model originally proposed by N. J. Armstrong et al., is studied. The model consists of a non-linear partial differential equation for the cell density in an N-dimensional infinite domain, with cell age used to track progression of cells through the cell cycle. It has a nonlocal flux term which models the component of cell motion attributable to cells having formed bonds with other cells within its sensing radius. Using the theory of fractional powers of analytic semigroup generators and working in spaces with bounded uniformly continuous derivatives, the local existence of classical solutions is proved. The method of characteristics is used to prove positivity and bounds are then obtained, leading to the existence of global solutions.

References

[1] N. J. Armstrong, K.J. Painter, J.A. Sherratt, "A continuum approach to modelling cell-cell adhesion", *J. Theor. Biol.*, v. 243 p. 98-113, 2006.
[2] J.A. Sherratt, S.A. Gourley, N.J. Armstrong, and K.J. Painter, "Boundedness of solutions of a nonlocal reaction-diffusion model for adhesion in cell aggregation and cancer invasion", *Euro. J. Appl. Maths.*, v. 20, p. 123-144, 2009.
[3] G. F. Webb, "Population models structured by age, size and spatial position", *Structured Population Models in Biology and Epidemiology, Lecture Notes in Mathematics*, No. 1936, Mathematical Biosciences Series , P. Magal and S. Ruan, Eds., p. 1-49, 2008.
[4] J. Dyson, R. Villella-Bressan, G.F. Webb, "A spatially structured model of tumor growth with cell age, cell size and mutation of cell phenotypes", *Math. Model. Nat. Phenom.*, v. 2, p. 69-100, 2007.

MATHEMATICAL MODELING OF CANCER IMMUNOTHERAPY USING A VESICULAR STOMATITIS VIRUS AND AN ADENOVIRUS

R. Eftimie[a] and **J.L. Bramson**[b,c] and **D.J.D. Earn**[a,c]

[a]Department of Mathematics and Statistics
McMaster University
Hamilton, ON, Canada L8S 4K1
reftimie@math.mcmaster.ca

[b]Centre for Gene Therapeutics, Department of Pathology and Molecular Medicine
McMaster University
Hamilton, ON, Canada, L8N 3Z5
bramsonj@mcmaster.ca

[c]Michael G. DeGroote Institute for Infectious Disease Research
McMaster University
Hamilton, ON, Canada, L8N 4L8
earn@math.mcmaster.ca

With the new insights into the molecular mechanisms of viral cytotoxicity, many viruses have been genetically engineered these past years to be used as oncolytic viruses (i.e., viruses that selectively infect and replicate in cancer cells, but not in normal cells). Unfortunately, many of these viruses are highly immunogenic, being rapidly eliminated by the immune response of the host (tumor-bearing hosts may have partially intact immune antiviral mechanisms). This diminishes the anti-tumor effect of the viruses. However, recent experimental results have shown that the treatment of a particular type of murine melanoma with two viruses which express the same tumor associated antigen, namely a replication-incompetent adenovirus (which boosts the immune response) and an oncolytic vesicular stomatitis virus, extends the survival rate of mice (even if it does not cure them) [1]. This is the result of the secondary immune response against the tumor antigen, which dominates the primary response against the virus.

Here we derive a mathematical model to investigate the observed interactions among immune cells, cancer cells, and two types of viruses: one which boosts the immune response (the adenovirus), and an oncolytic virus (the vesicular stomatitis virus). We use experimental data from our lab to validate the model and estimate parameter values. This allows us to discuss conditions that lead to tumor growth, and to propose hypotheses for tumor elimination which can be tested experimentally. In particular, we suggest that suppression of the immune response might improve viral oncolysis but cannot lead to a cure (i.e., no stable tumor-free steady states). However, complete elimination of cancer cells is possible in the presence of an immune response, and depends on the magnitude of this response as well as the parameters that control virus proliferation and tumor cell lysis. We also show that the temporary reduction in tumor size observed in the absence of adenovirus boost is solely the result of the oncolytic virus, since the immune response is too weak to account for any tumor reduction.

Keywords: mathematical modeling, cancer immunotherapy, adenovirus, vesicular stomatitis virus

References

[1] B.W. Bridle, J.E. Boudreau, B.D. Lichty, J. Brunelliere, K. Stephenson, S. Koshy, J.L. Bramson and Y. Wan, “Vector to prime antitumor immunity amenable to rapid boosting with adenovirus", *Molecular Therapy*, v. 17, p. 1814-1821, 2009.

A Mathematical Model for the Effects of HER2 Over-expression on Cell Cycle Progression in Breast Cancer

Amina Eladdadi, David Isaacson
Department of Mathematical Sciences
Rensselaer Polytechnic Institute, Troy, NY 12180, USA

March 4, 2010

Abstract

One of the major cellular processes affected by HER2 signaling is the cell cycle. Recent experimental studies show that the increased HER2-receptor signaling resulting from the receptor over-expression contributes to the deregulation of the G1/S transition. The phenotypic consequence of HER2 over-expression is a shortening of the G1-phase of the cell cycle and early S-phase entry, which results in uncontrolled cell proliferation. In this study, we formulate and validate a mathematical model to study the effects of the HER2-over-expression on the cell cycle progression in breast cancer. The model consists of a system of ordinary differential equations that relates the cell cycle transition rates to the signaling properties of the EGFR-HER2 s through their binding kinetics, and to the population dynamics of cells in the corresponding cell-cycle phase. The model is analyzed using numerical simulations. In agreement with experimental observations, the simulation results show that with key parameter values: (1) HER2 over-expression results in a shorter G1-phase and early S-phase entry; (2) with a 1-to-1 ratio between EGFR and HER2 , the growth advantage in HER2 over-expressing cells is indeed associated with the increase in the HER2 expression level; (3) EGFR plays a key role in regulating cell proliferation rate in HER2-over-expressing cells. This mathematical model also elucidates the interaction and roles of other model parameters in determining cell-cycle transition rates of HER2-over-expressing cells.

Keywords:

HER2. EGFR. Cell Cycle. Cell Proliferation. Receptor Modeling. Mathematical Modeling. Breast Cancer. HER2 over-expression.

References

[1] Eladdadi,A., Isaacson, D., A Mathematical Model for the Effects of HER2 Over-expression on the Cell Proliferation in Breast Cancer. The Bulletin of Mathematical Biology 2008;70(6):1707-29
[2] Eladdadi,A., Isaacson, D., Modeling the Effects of HER2 Over-expression on the Cell Proliferation in Breast Cancer. The Bulletin of Mathematical Biology (in press 2009)

SIZE-STRUCTURED POPULATION MODEL WITH CONSTANT SIZE AND CONSTANT INFLOW OF NEWBORNS

M. El-Doma[a]

[a]Department of Applied Mathematics
University of Khartoum
Faculty of Mathematical Sciences, P. O. Box: 321, Khartoum-Sudan
biomath2004@yahoo.com

We study a size-structured population dynamics model when there is a constant inflow of newborns from an external source and determine necessary and sufficient conditions for the population to be of constant size. We also give conditions for the steady state to be globally stable. Moreover, we illustrate our results by several examples. The results in this paper generalize previous results given in Kostova, et al. (1991).

References

[1] T. Kostova and F. Milner, "Nonlinear age-dependent population dynamics with constant size", *SIAM J. MATH. ANAl.*, Vol. 22, No. 1, p. 129-137, 1991.
[2] M. El-Doma, "Remarks on the stability of some size-structured population models III: The case of constant inflow of Newborns", *AAM: Intern. J.*, Vol. 3, No. 2, p. 200-217, 2008.

ANALYSIS OF A TWO-STRAIN TRANSMISSION MODEL USING COMPUTER ALGEBRA

M. EL Kahoui[a] and **A. Otto**[b] and **M.-F. Roy**[c] and **T. Van Effelterre**[d,]

[a]Department of Mathematics, Faculty of Sciences Semlalia, P.O. Box 2390, Marrakech, Morocco
elkahoui@ucam.ac.ma

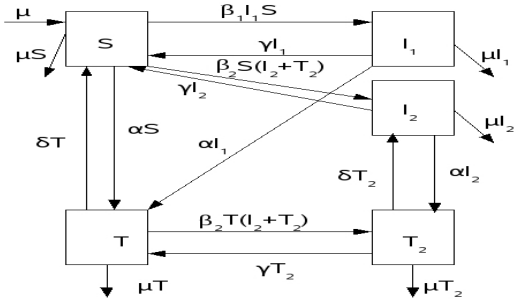
[b]IREM de l'Université Abdou Moumouni, BP 10896 Niamey, Niger
otto_adamou@yahoo.com

[c]IRMAR URA CNRS 305, Université de Rennes 1, Campus de Beaulieu 35042, Rennes, France
marie-francoise.roy@univ-rennes1.fr

[d]GlaxoSmithKline Biologicals, 20 rue Fleming, B-1300 Wavre, Belgium
Thierry.Van-Effelterre@gskbio.com

We present a typical example of a compartmental transmission model that can be dealt with algebraically. We use exact methods from real algebraic geometry and computer algebra to find all the equilibria of the ODE system describing the model and to study their stability as well as their bifurcations.

The model concerns a host population, a part of its individuals are under antibiotic (Ab) treatment against a two-strain bacterial pathogen. Individuals who are not under Ab treatment can be colonized by an antibiotic-susceptible (Ab-S) strain or by an antibiotic-resistant (Ab-R) strain of a bacterial pathogen, but not by both at the same time (i.e., maximal competition), while those under antibiotic treatment can only be colonized by the Ab-R strain. We assume there is a fitness cost for resistance such that the Ab-R strain is somewhat less transmissible than the Ab-S strain. The host population is sub-divided into 5 compartments representing the fractions of the population in each state, 3 states for individuals not under Ab treatment: susceptible (S), colonized by the Ab-S strain (I_1) and colonized by the Ab-R strain (I_2), and 2 states for individuals under Ab treatment: susceptible (T), and colonized by the Ab-R strain (T_2). We give below the transfer diagram of the model



The model has a unique colonization free equilibrium (CFE). The basic reproduction number of the Ab-S (resp. Ab-R) strain in the absence of the Ab-R (resp. Ab-S) strain is $R_{0S} = \frac{(\delta+\mu)\beta 1}{(\alpha+\gamma+\mu)(\alpha+\delta+\mu)}$ (resp. $R_{0R} = \frac{\beta 2}{\mu+\gamma}$). The CFE is hyperbolic and locally asymptotically stable if and only if $R_{0S} < 1$ and $R_{0R} < 1$. When $R_{0S} > 1$ or $R_{0R} > 1$ three kinds of equilibria, depending on the values of R_{0S} and R_{0R}, enter the scene. We give a complete characterization of their existence and stability and we show that all codimension-one bifurcations are transcritical. This model demonstrates that computer algebra gives the means to characterize exactly the equilibria and loci of bifurcations of simple transmission models.

Hopf bifurcation in predator-prey models

M. Falconi[a] and **Jaume Llibre**[b]

[a]Departmento de Matemáticas
Facultad de Ciencias, UNAM
C. Universitaria, México, D.F. 04510
falconi@servidor.unam.mx

[b]Department de Matemátiques
Universitat Autónoma de Barcelona
08193 Bellaterra, Barcelona, Catalonia, Spain
jllibre@mat.uab.cat

We are concerned with the role of the age structure of the prey in the dynamic of a predator-prey model. In particular, we analyze the existence of periodic orbits when the predator feeds on each one of the two age classes of prey. Three families of models are considered in correspondence with different prey and predator behaviors. Specifically, a constant predation rate on the non reproductive class of the prey is considered in the first family; a Holling predation of type two on the non reproductive class is incorporated in the second and in the third family; a defense group mechanism of the reproductive class is introduced in the third family. We prove that these three families of models exhibit Hopf bifurcations and that the Hopf periodic orbit is a local attractor. The stage-structured models have received much attention in recent years, see for instance [1] and [2], and references therein.

References

[1] M. Falconi, "The effect of the prey age structure on a predator-prey system", *Sc. Math. Jap.*, **64**(2), p. 267-275, 2006.

[2] M. Wang, "Stability and Hopf bifurcation for a prey-predator model with prey-stage structure and diffusion", *Math. Biosc.*, 212, p.149-160, 2008.

A Compartmental Model of Hepatitis B with vertical transmission

A. A. Fall[a], **A. Iggidr**[b] and **G. Sallet**[a,b]

[a]MASAIE team, INRIA-Nancy Grand Est.
UMI IRD 209, UMMISCO
LANI, Université de Saint-Louis, Sénégal.

[b]MASAIE team, INRIA-Nancy Grand Est.
LMAM(UMR CNRS 7122), Université Paul Verlaine, Metz.
Ile du Saulcy, 57045 Metz Cedex 01, France.

Abdoulaziz.Fall@loria.fr, iggidr@loria.fr, sallet@loria.fr

A new model of the transmission dynamics of Hepatitis B Virus (HBV) in countries with high transmission rates is presented. Details of hepatitis B natural history, such as the existence of infectious and non-infectious carriers, the influence of age on the development of the hepatitisB carrier state. are taken into account in this model.The model is age stratified, and includes asymptomatic carriers and acute infection as well.

Our model aims to answer to the controversy, with regard to the incidence of the vertical and perinatal transmission of the virus in some parts of Africa. Two concurrent hypothesis are assumed between the World Health Organization (WHO) on one hand and Hepatitis B's specialists in Senegal on the other hand.

We compute the basic reproduction ratio $\mathcal{R}_0$ and present an analysis of the stability of the models using Lyapunov techniques.

We prove that the disease free equilibrium (DFE) is globally asymptotically stable if $\mathcal{R}_0 \leq 1$ and prove the existence and uniqueness of an endemic equilibrium (EE) when $\mathcal{R}_0 > 1$.

Numerical simulations are done to illustrate the behaviour of the model, using data collected during the campaign against epidemic hepatitis B in Senegal and from published literature. These models enable to estimate the influence of the vertical and perinatal transmission of the Hepatitis B virus.

Keywords: *Modelling, basic reproduction ratio $\mathcal{R}_0$, epidemiological models, Infectious diseases, Hepatitis B Virus (HBV), vertical transmission, numerical simulation, public health.*

Global Existence For an Age-Structured Model With Vertical Transmission

S. Fekih-Rahou ∗, S.M. Bouguima †, and B. E. Ainseba‡

and

∗ *Faculté des Sciences, Département de Mathématiques, Université de Tlemcen-Algérie*
si_fekih@yahoo.fr
† *Faculté des Sciences, Département de Mathématiques, Université de Tlemcen-Algérie*
bouguima@yahoo.fr
‡ *UFR Sciences Modélisation (SCIMS) 33076 Bordeaux cedex*
bedreddine.ainseba@u-bordeaux2.fr

Version:

Key Words: Age structure, Reaction diffusion, Epidemics, Vertical transmission, Global existence in L^1, Steady states.-

1. ABSTRACT

We perform a mathematical model that describes the evolution of population in presence of a disease which transmitted vertically i.e there are probabilities that the newborns are infected.

The model is an SI age structured system which contains two non local reaction-diffusion equations. The force of infection is depending on the total population. More precisely we are concerned with following model:

$$\begin{cases} \partial_t u + \partial_a u + \mu_u(a)\, u - d_1 \Delta_x u = \gamma(a, P_1, P_2)\, v - \delta(a, P_1, P_2)\, u & in\ (0, a^+) \times (0, T) \times \Omega \\ \partial_t v + \partial_a v + \mu_v(a)\, v - d_2 \Delta_x v = -\gamma(a, P_1, P_2)\, v + \delta(a, P_1, P_2)\, u & in\ (0, a^+) \times (0, T) \times \Omega \\ u(0, t, x) = \lambda \int_0^{a^+} \beta_u(a)\, u(a, t, x)\, da & in\ (0, T) \times \Omega \\ v(0, t, x) = \int_0^{a^+} \beta_v(a)\, v(a, t, x) + (1 - \lambda)\, \beta_u(a)\, u(a, t, x)\, da & in\ (0, T) \times \Omega \\ u(a, 0, x) = u_0(a, x) & in\ (0, a^+) \times \Omega \\ v(a, 0, x) = v_0(a, x) & in\ (0, a^+) \times \Omega \\ \partial_\nu u = 0 & in\ (0, a^+) \times (0, T) \times \partial\Omega \\ \partial_\nu v = 0 & in\ (0, a^+) \times (0, T) \times \partial\Omega \end{cases}$$

Existence of global solutions and steady states are obtained, using semi group theory and monotone operators in the Banach space $L^1((0, a^+) \times \Omega; \mathbb{R}^n)$; the space of equivalence classes of Lebergue integrable functions from $(0, a^+) \times \Omega$ in $\mathbb{R}^n$

OPTIMAL STRATEGY OF FISHING PROBLEM ON HERMAPHRODITE POPULATION

Adel Ferchichi

In this work, we introduce some economic and biologic aspects of renewable resource exploitation, applied to hermaphrodite fishes. We also developed optimization models to predict the population dynamics and to identify the features of optimal resource exploitation strategies [1]. Our goal is to find an optimal strategy of fishing problem for hermaphrodite population, taking into account fishing at different stages. For that, we modify the structured model of [2] by introducing the maximization of a total discounted net revenues derived from exploitation of the resource. More precisely, the grouper is a species, which is able to produce successively, during his life, female and then male gametes. Thus, it is suitable to subdivide the population into n classes according to the length of the individual: Young grouper (Eggs + Larva), Immature adult, Female and Male. These classes are characterized by a difference in the demo-ecological parameters. The complete model is as following:

$$\begin{cases} n_1(t+1) = k_1 s_1 n_1(t) + k_1 f g(n_4(t)) n_3(t) \\ n_2(t+1) = K_2(n_2, n_3, n_4) t_{12} n_1(t) + K_2(n_2, n_3, n_4) s_2 n_2(t) \\ n_3(t+1) = k_3 t_{23} n_2(t) + k_3 s_3 n_3(t) - q_3 E_3(t) n_3(t) \\ n_4(t+1) = k_4 t_{34} n_3(t) + k_4 s_4 n_4(t) - q_4 E_4(t) n_4(t) \end{cases} \tag{9}$$

Where n_i is the individual number of the class i, k_i is the rate of the remaining individuals in the class i after dispersion, f is the fertility, t_{ij} and s_i are survivals rates, K_2 the function giving the rate of remaining immatures after dispersion, E_i the fishing effort and q_i the catch-ability. Then, we define the optimization principle and try to find an optimal policy of the fishing activities. We prove the existence of an optimal strategy for the fishing activities: maintaining the system state fisheries around an equilibrium point by ensuring a sustainable development of the resource and ensuring at the same time a maximization of fishing revenues by using an optimal control.

REAL-TIME GROWTH RATE FOR SIR EPIDEMICS IN SOCIALLY STRUCTURED POPULATIONS

L. Pellis, **N. M. Ferguson** and **C. Fraser**

Department of Infectious Disease Epidemiology
Imperial College London
St. Mary's Hospital, Norfolk Place, London, W2 1PG
l.pellis05@imperial.ac.uk

Keywords: real-time growth rate, Malthusian parameter, SIR model, stochastic epidemic, epidemic dynamics, households, workplaces, social structure, household reproduction number

SIR epidemic models for socially structured human populations (e.g. including households, workplaces, schools...) naturally suggest a stochastic modelling approach to deal satisfactorily with frequent mixing in small groups. In particular, such models have found a florid ground in the so-called standard stochastic SIR model.

Unfortunately, such a model is somewhat not designed to focus on the dynamics of infection spread. Therefore, despite being of great practical interest, results about the epidemic dynamics in socially structured populations are very limited (usually restricted only to the unrealistic Markovian case of constant recovery rate).

Here I present a refinement of the standard stochastic model that is more suitable to realistically characterise the dynamics of the infection spread and suggest a novel and coherent framework for the numerical computation of the real-time growth rate (i.e. the simplest piece of information concerning the epidemic dynamics in a large and fully susceptible population) in a model with households and (time permitting) with households and workplaces.

Because the real-time growth rate is often one of the first pieces of information readily available during an emerging outbreak, a practically useful application consists in reversing the methodology to allow the estimation of the household reproduction number from the real-time growth rate.

References

[1] L. Pellis, "Mathematical models for emerging infections in socially structured populations: the presence of households and workplaces, *PhD Thesis*, 2008, http://hdl.handle.net/10044/1/4696
[2] C. Fraser, "Estimating individual and household reproduction numbers in an emerging epidemic", *PLoS ONE*, 2(8), e758, 2007, doi:10.1371/journal.pone.0000758

CELLULAR AUTOMATA FOR ECOEPIDEMIC MODELS

L. Ferreri[a] and **E. Venturino**[a]

[a]Università di Torino
Dipartmento di Matematica "Giuseppe Peano"
via Carlo Alberto 10, 10123 Torino
lucaferreri@yahoo.it
ezio.venturino@unito.it

The goal of this work is to present a new Cellular Automaton (CA) model to simulate an epidemics spreading in predator-prey models. Cellular automata are a tool for modelling pattern formations, [1-5,8]. Systems in which diseases affect interacting populations are nowadays known as ecoepidemic models, [7]. While such systems have been considered in the recent literature and have been modeled in space via reaction-diffusion equations, [6], the approach by cellular automata does not appear to have been yet exploited in this context. To our knowledge, this study represents in fact the first attempt of this kind, in the literature of ecoepidemic models, [7].

In the first part of the investigation the case of the disease affecting the prey only is studied. The main focus here lies on the possible different predation dynamics, such as: predators eating only infect prey, predators feeding only on sound prey, and finally predators hunting both sound and infected prey with no preference. These dynamics are considered with particular attention to their effects on the spread of the epidemics.

In the second part the case of diseases among the predators is presented. In this situation the simulations highlight the epidemics' consequences on the prey growth.

The CA used in this context is structured on a double layer grid respectively representing the different states of families. The possible states for the prey are: sound, infected and empty. For the predator they are: hungry, fertile and empty. The evolution law has a local and deterministic form. A specific feature of the CA is represented by the fact that movement of the species is not considered directly. It does indeed appear in the simulations, but it is rather incorporated in the CA as an effect of the reproduction dynamics. Simulations with different neighborhood types are carried out and reported.

References

[1] M. Gerhardt, and H. Schuster, "Cellular automaton describing the formation of spatially ordered structures in chemical systems", *Physica D*, no. 36, p. 209-221, 1989.
[2] J.M. Greenberg, C. Greene, and S.P. Hastings, "A computational problem arising in the study of reaction diffusion equations", *SIAM JOURNAL Algebraic Discrete Methods*, v. 1, no. 1, p. 34-42, 1980.
[3] J.M. Greenberg and S.P. Hastings, "Spatial patterns for discrete models of diffusion in excitable media", *SIAM journal on applied mathematics*, v. 34, no. 3, p. 515, 1978.
[4] J.M. Greenberg, S.P. Hastings, and B.D. Hassard, "Pattern formation and periodic structures in systems modeled by reaction-diffusion equations", *Bulletion of the AMS*, v. 84, no. 6, p. 1296, 1978.
[5] A. Ilachinski, "Cellular automata, a discrete universe", *World Scientific*, 2001.
[6] H. Malchow, S. Petrovskii, E. Venturino, "Spatiotemporal patterns in Ecology and Epidemiology", *CRC*, 2008.
[7] E. Venturino, "Ecoepidemiology 15 years later: a review", Numerical Analysis and Applied Mathematics, T. Simos (Editor), *Proceedings of ICNAAM 2007*, AIP #936, p. 31-34, 2007.
[8] S. Wolfram, "A new kind of science", *Wolfram Media*, 2002.

A MODEL OF FREQUENCY-DEPENDENT BIAS FOR THE SPREAD OF OPINIONS IN A LATTICE

J. F. Fontanari[a]

[a]Instituto de Física de São Carlos
Universidade de São Paulo
Caixa Postal 369, 13560-970 São Carlos, SP, Brazil
fontanari@ifsc.usp.br

The pertinence of culture for understanding the hominid evolutionary process has been persuasively advocated by Laland & Brown [1] using the biological concept of niche construction [2]. Hence understanding how opinions and, more generally, cultural traits disseminate through a population is crucial to fully comprehend human evolution. Of particular interest here is the understanding of the mechanisms that lead to the appearance of stable domains characterized by distinct cultural traits, given that people's beliefs have a tendency to become more similar to each other's as people interact repeatedly [3]. Here we study an extreme version of the frequency-dependent bias model in which an individual adopts the trait/opinion shared by the majority of its neighbors - the majority-vote rule model [4], [5]. We assume that the individuals are fixed in the sites of a square lattice of linear size L and that they can interact with their four nearest neighbors only. Within a mean-field framework, we derive the equations of motion for the density of individuals adopting a particular opinion in the site and pair approximations. However, this framework predicts a single cultural domain that takes over the entire lattice whereas extensive Monte Carlo simulations indicate the existence of a rich distribution of cultural domains or clusters, the number of which scales as $a_F L^2$ and the size of the largest cluster scales as $b_F \ln L$. Here the integer label F stands for the number of binary cultural traits that characterize the culture of the individuals and the coefficients of the scaling laws are given by $1 - a_F \approx \exp(-F/5)$ and $b_F \approx F^{-1.5}$. In addition, the analysis of the sizes of the cultural domains shows that they are exponentially distributed for $F > 1$, but they follow a power-law distribution for $F = 1$. Finally, we compare these results with those obtained through the random assignment of cultural characteristics to each individual in the lattice.

References

[1] K. N. Laland, and G. R. Brown, "Niche Construction, Human Behavior, and the Adaptive Lag Hypothesis", *Evolutionary Anthropology*, v. 15, p. 95-104, 2006.
[2] K. N. Laland, J. Odling-Smee, and M. W. Feldman, "Evolutionary consequences of niche construction and their implications for ecology", *Proceedings of the National Academy of Sciences USA*, v. 96, p. 10242-10247, 1999.
[3] R. B. Boyd, and P. J. Richerdson, *Culture and the Evolutionary Process*. Chicago: The University of Chicago Press, 1988.
[4] T. M. Liggett, *Interacting Particle Systems*. New York: Springer Verlag, 1985.
[5] D. Parisi, F. Cecconi, and F. Natale, "Cultural Change in Spatial Environments", *Journal of Conflict Resolution*, v. 47, p. 163-179, 2003.

On a Model for the Initiation of Cell Movement

J. Fuhrmann and **Angela Stevens**

In [1] we proposed a one dimensional model for the polarization of the actin cytoskeleton in initially symmetric cells upon some external stimulus. We showed by simulations how a very concise model for the cytoskeleton is sufficient to account for the initiation of an asymmetric distribution of actin filaments which may drive the cell into directed motion. Now we describe the further polarization and the actual initiation of movement by a free boundary problem for the cell body spanned by the cytoskeleton. Here we assume that at an initial stage of motion the cell membrane exerts only negligible forces on the growing filaments which allows for characteristic boundary conditions. We will comment on the well posedness of the resulting model for small time and show first numerical results. Furthermore there will be some remarks made on possible gradient blow up within finite time. Moreover we have investigated a reduced system to understand the hyperbolic dynamics of the proposed model. We will demonstrate some interesting stability properties of the resulting system of no more than 2 equations. In particular a genuine difference is observed between linear and nonlinear stability indicating sensitivity to the shape and size of perturbations of the trivial steady state. Furthermore various types of long time behavior will be shown by numerical simulations.

Gene Expression Time Delays and Turing Pattern Formation

S. Seirin Lee[a] and **E.A. Gaffney**[b]

[a]Graduate School of Mathematical Sciences
The University of Tokyo, JAPAN
seirin.lee@gmail.com

[b]Centre for Mathematical Biology
Mathematical Institute, University of Oxford, UK
gaffney@maths.ox.ac.uk

There are numerous examples of morphogen gradients controlling long range signalling in developmental and cellular systems. The prospect of two such interacting morphogens instigating long range self-organisation in biological systems via a Turing bifurcation has been explored, postulated or implicated in the context of numerous developmental processes. However, modelling investigations of cellular systems typically neglect the influence of gene expression on such dynamics, even though transcription and translation are observed to be important in morphogenetic systems. Critically, the previous study [1] observed that even these relatively small gene expression delays substantially amplify patterning lags, thus demonstrating the potential for gene expression dynamics to exert enormous temporal influence on pattern formation.

The investigations of our study demonstrate that the behaviour of Turing models profoundly changes on the inclusion of gene expression dynamics and is sensitive to the sub-cellular details of gene expression [2]. Furthermore, they also highlight that domain growth can no longer ameliorate the excessive sensitivity of Turing's mechanism in the presence of gene expression time delays [3, 4]. These results also indicate that the behaviour of Turing pattern formation systems on the inclusion of gene expression time delays may provide a means of distinguishing between possible forms of interaction kinetics, and also emphasises that sub-cellular and gene expression dynamics should not be simply neglected in models of long range biological pattern formation via morphogens. Finally, our study calls into question the plausibility of the Turing mechanism for pattern formation in biology where robustness is a key requirement. Turing's mechanism would generally require a novel and extensive secondary mechanism to control reaction diffusion patterning.

Keywords Pattern Formation, Cellular Dynamics, Time Delays, Reaction-Diffusion System

References

[1] E.A. Gaffney, N.A.M. Monk, *Gene expression time delays and Turing pattern formation systems*, Bull.Math.Bio. 68 (2006) 99–130.

[2] S. Seirin Lee, E.A. Gaffney, N.A.M. Monk, *The Influence of Gene Expression Time Delays on Gierer-Meinhardt Pattern Formation Systems*, to appear in Bull.Math.Bio. (2010).

[3] S. Seirin Lee, E.A. Gaffney, *Aberrant Behaviours of Reaction Diffusion Self-organisation Models on Growing Domains in The Presence of Gene Expression Time Delays*, to appear in Bull.Math.Bio. (2010).

[4] S. Seirin Lee, E.A. Gaffney, R.E. Baker, *An Investigation of Pattern Formation on Morphogen-controlled Growing Domains with Gene Expression Time Delays and Domain Response Delays*, preprint.

OPPORTUNISM AS OPTIMAL FORAGING STRATEGY

J. Garay[a], **F. Szigetib**[b], **T. Cabelloc**[c] and **R. Carreño**[d]

[a]Department of Plant Taxonomy and Ecology,
Eötvös LoràND University,
Budapest, Pázmány sétány 1/c, 1117 Hungary
garayj@ludens.elte.hu

[b]Department of Control Systems,
University of the Andes,
Merida 5101, Venezuela
szigeti.ferenc@gmail.com

[c]Department of Applied Biology,
University of Almería,
La Cañada de San Urbano, s/n, 04120 Almería, Spain
tcabello@ual.es

[d]Department of Applied Biology,
University of Almería,
La Cañada de San Urbano, s/n, 04120 Almería, Spain
ramon@ual.es

A model for cell-cell adhesion, based on a model originally proposed by N. J. Armstrong et al., is studied. The model consists of a non-linear partial differential equation for the cell density in an N-dimensional infinite domain, with cell age used to track progression of cells through the cell cycle. It has a nonlocal flux term which models the component of cell motion attributable to cells having formed bonds with other cells within its sensing radius. Using the theory of fractional powers of analytic semigroup generators and working in spaces with bounded uniformly continuous derivatives, the local existence of classical solutions is proved. The method of characteristics is used to prove positivity and bounds are then obtained, leading to the existence of global solutions.

Aknowledgements: Research partially supported by the Hungarian National Scientific Research Fund (OTKA 62 000, 68187 and 81279) and by the Scientific and Technological Innovation Fund (of Hungary, ES-17/2008) and the Ministry of Education and Sciences (of Spain HH2008-0023).

References

[1] Cressman, R. Garay, J., "The effect of opportunistic and intentional predators on herding behaviour of prey" (mimeo)
[2] J. Garay, F. T. Móri, "When is the predator's opportunism remunerative ?" (mimeo)
[3] L. Berec, V. Krivan. "A mechanistic model for partial preference", *Theor. Pop. Biol.*, v. 58, p. 279-289, 2000.

Spatial patterns and extinction threshold in forest dynamics with height structure

J.L. Garcia-Domingo[a] and **J. Saldaña**[b]

[a]Dept. d'Economia i Empresa
Universitat de Vic
08500 Vic, Spain
jlgarcia@uvic.cat

[b]Dept. Informàtica i Matemàtica Aplicada
Universitat de Girona
17071-Girona, Spain
jsaldana@ima.udg.edu

In this communication we present and analyze a pair approximation (PA) model for spatial forest dynamics defined on a regular lattice. The model assumes a simple vertical layering of the forest in terms of the canopy (the tallest layer which is composed of mature trees), the understory (the layer composed of saplings of canopy trees), and the shrub layer (the lowest layer of woody vegetation). This vertical forest stratification, although simple, allows for the inclusion of a new type of local interaction among individuals, namely, the competition for sunlight, leading to more elaborated lattice models and more complex spatial patterns. In particular, this vertical forest structure generalizes the one in the first lattice models which were focussed on the forest gap dynamics and, hence, sites in the lattice could be only in one of two possible states: empty (gap) and occupied [1]. A richer vertical layering has been considered in previous papers dealing with cellular automata models of forest dynamics (see [2,3]). The nonlinearities in the model are associated to the processes of light interference, gap expansion, and recruitment.

We obtain an expression of the basic reproduction number R_0 for the PA model which generalizes the one that follows from the mean-field approximation since it includes information about the spatial arrangement of individuals close to extinction. In particular, the expression contains the conditional probabilities that an immature tree has a mature tree as a neighbor, and that a mature tree has a neighboring gap site, at the extinction equilibrium. These probabilities describe the environmental conditions experienced by immature and mature trees. The strict positiveness of such probabilities at the extinction equilibrium is proved by rescaling the model by means of a change of variable similar to the one introduced in [4]. Moreover, we analyze the corresponding survival-extinction transition of the forest in a similar manner as in [5], and the spatial correlations among gaps, immature and mature trees close to this critical. Finally, predictions of the pair approximation model are compared with those of a cellular automaton.

References

[1] T. Kubo, Y Iwasa, and N. Furumoto, "Forest spatial dynamics with gap expansion: total gap area and gap size distribution", *Journal of Theoretical Biology*, v. 180, p. 229-246, 1996.
[2] S.C. Manrubia and R.V. Solé, "On forest spatial dynamics with gap formation", *Journal of Theoretical Biology*, v. 187, p. 159-164, 1997.
[3] C. Pagnutti, M. Anand, and M. Azzouz, "Lattice geometry, gap formation and scale invariance in forests", *Journal of Theoretical Biology* v. 236, p. 79-87, 2005.
[4] I. Siekmann, H. Malchow, and E. Venturino, "An extension of the Beretta-Kuang model of viral diseases", *Mathematical Biosciences and Engineering*, v. 5, p. 549-565, 2008.
[5]N. Stollenwerk, J. Martins, and A. Pinto, "The phase transition lines in pair approximation for the basic reinfection model SIRI", *Physics Letters A*, v. 371, p. 379-388, 2007.

ANALYSIS OF SYNCHRONIZATION IN A NEURAL POPULATION BY A POPULATION DENSITY APPROACH

A. Garenne[a], **J. Henry**[b] and **O. Tarniceriu**[c]

[a]Basal Gang, Laboratoire Mouvement, Adaptation, Cognition,
CNRS-UMR
5227, Bordeaux, France
agaren100p@u-bordeaux2.fr

[b]INRIA Bordeaux Sud Ouest IMB
351, Cours de la Libération, 33405 Talence cedex, France
Jacques.Henry@inria.fr

[c]Department of Sciences
Al. I. Cuza University
Iaşi, Romania
tarniceriuoana@yahoo.co.uk

In this presentation we deal with a model describing the evolution in time of the density of a neural population in a state space, where the state is given by Izhikevich's two - dimensional single neuron model. The main goal is to mathematically describe the occurrence of a significant phenomenon observed in neurons populations, the *synchronization*. To this end, we are making the transition to phase density population, and use Malkin theorem to calculate the phase deviations of a weakly coupled population model. Special conditions under which the solution of the phase density population system tends to a solution expressing the synchronized state of the system are derived.

References

[1] F. C. Hoppensteadt and E. Izhikevich, "Weakly connected neural networks", Springer-Verlag, New York, 1997.
[2] E. M. Izhikevich, "Dynamical Systems in Neuroscience: The geometry of excitability and bursting", The MIT Press, 2007.
[3] J. Modolo, A. Garenne, J. Henry, A. Beuter, "Development and validation of a neural population model based on the dynamics of discontinuous membrane potential neuron model", *J. Integr. Neurosci.*, v. 6, No. 4, p. 625-656, 2007.
[4] J. Modolo, J. Henry, A. Beuter, "Dynamics of the subthalamo-pallidal complex in Parkinson's Disease during deep brain stimulation", *J. Biol. Phys.*, v. 34 , p. 351-366, 2008.

DENSITY-DEPENDENT DISPERSAL IN A STRUCTURED METAPOPULATION MODEL

M. Gyllenberg[a], **S.A.H. Geritz**[b] and **P. Ondracek**[c]

[a]Department of Mathematics and Statistics
University of Helsinki
Gustaf Hällströmin katu 2b, 00014, Finland
mats.gyllenberg@helsinki.fi

[b]Department of Mathematics and Statistics
University of Helsinki
stefan.geritz@helsinki.fi

[c]Department of Mathematics and Statistics
University of Helsinki
petr.ondracek@helsinki.fi

We study the evolution of density-dependent dispersal in a structured metapopulation subject to local catastrophes that eradicate local populations. To this end we use the theory of structured metapopulation dynamics and the theory of adaptive dynamics.
The set of evolutionarily possible dispersal functions (i.e., emigration rates as a function of the local population density) is derived mechanistically from an underlying resource-consumer model. The local resource dynamics is of a flow-culture type and consumers leave a local population with a constant probability per unit of time κ when searching for resources but not when handling resources (i.e., eating and digesting). The time an individual spends searching (as opposed to handling) depends on the local resource density, which in turn depends on the local consumer density, and so the average *per capita* emigration rate depends on the local consumer density as well.
The derived emigration rates are sigmoid functions of local consumer population density. The parameters of the local resource-consumer dynamics are subject to evolution. In particular, we find that there exists a unique evolutionarily stable and attracting dispersal rate κ^* for searching consumers. The κ^* increases with local resource productivity and decreases with resource decay rate. The κ^* also increases with the survival probability during dispersal, but as a function of the catastrophe rate it reaches a maximum before dropping off to zero again.

References

[1] M. Gyllenberg, S.A.H. Geritz and P. Ondracek, "Evolution of density-dependent dispersal in a Structured Metapopulation", Mathematical Biosciences, 219, p. 142-148, 2009

ANTIGENIC VARIATION OF TRYPANOSOMES: FROM PARASITE GENETICS TO WITHIN-HOST DYNAMICS AND EVOLUTION

E. Gjini, **C. A. Cobbold**, **D. T. Haydon** and **J.D. Barry**

The antigenic archive of pathogens plays an important role in within-host infections. Antigenically varying pathogens escape host immunity by switching expression of different variants. The timing of this process and the number of antigenic variants expressed over the course of an infection are often stochastic and independent of host immune pressure. They result mainly from genetic properties of the antigenic archive of the pathogen. However, their subsequent interaction with dynamic host immune mechanisms determines the outcome of an infection: its peak, duration, and nature of chronicity.

African Trypanosomes are a prominent example of such pathogens exploiting antigenic variation to establish chronic infection in their hosts. With increasing availability of parasite genomic data, more realistic within- host models are now possible, through the integration of features of the antigenic archive. This integration has the potential to significantly improve our understanding of the fundamental mechanisms of parasite immune evasion.

Here we study the within-host dynamics of trypanosomes. We use a mathematical model to investigate how antigenic archive properties modulate the balance between specific and general control of the pathogen. This balance depends crucially also on the time delay between pathogen challenge and immune initiation, and the presence of immune memory. We show how the genetics of the parasite dictates critical thresholds at the within-host level, which besides influencing infection progression in a single host, have further implications for parasite fitness.

A Stage Structured Fishery Model with Reserve Area and Pulsed Harvesting

Mini Ghosh[a]

[a]School of Mathematics and Computer Applications
Thapar University
Patiala-147004, India
minighosh@thapar.edu

In this paper, a stage structured fishery model with reserve area is formulated and analyzed. It is assumed that fishing resource system consists of two zones, a free fishing zone and a reserve zone where fishing is prohibited. Only mature fish are migrating and are being harvested while they are in fishing zone and the immature fish are diffusing between reserved and unreserved zone. The equilibria of the proposed model and their stability are investigated. The model is simulated for varied set of parameters and sensitivity of the parameters and effect of diffusion are also studied. Here comparison of pulsed and continuous harvesting is also performed using numerical simulation.

Keywords: Fishery model, reserve area, pulsed harvesting, diffusion.

TIME TO EXTINCTION OF INFECTIOUS DISEASES THROUGH BELLMAN-HARRIS BRANCHING PROCESSES

M. González[a] and **R. Martínez**[b]

Department of Mathematics
University of Extremadura
[a] mvelasco@unex.es
[b] rmartinez@unex.es

When an infectious disease is strongly detrimental to the population in which it is spreading, control measures are needed to protect susceptible individuals. Vaccination programs represent one of the most effective forms of control (see [3]). However, immunizing the whole population is not an option in most cases (either because it is logistically impossible or because it is too costly), so that only a proportion of susceptible individuals can be vaccinated. How to determine the necessary proportion is an important public health problem in its own right, and depends on multiple factors. One of these factors that is particularly significant for public health authorities to assess vaccination efficiency is the time that elapses for the infectious disease to become extinct, known as the disease's time to extinction.

The aim of the present work is to provide an approach to this problem by modeling epidemic spread using stochastic models, namely branching processes. Specifically, we use the Bellman-Harris branching process (see [2]) to describe the outbreaks of an infectious disease following an SIR scheme where the contagious time is negligible with respect to the incubation period. First we study the properties of the disease's time to extinction depending on the proportion of the immunized individuals into the population. We consider that this proportion may be variable over time. Thus, for example, before vaccination it may be null, may increase during the vaccination process and may be constant after vaccination. In this way, the obtained results generalize those given in [1], where the proportion of the immunized individuals is considered stable over time. Then, from these results, we suggest an optimal vaccination policy based on the quantiles of the disease's time to extinction. Finally, we provide a simulation-based method to determine numerically the optimal vaccination level and show the accuracy of the method by way of an example.

Acknowledgements Research supported by the Ministerio de Ciencia e Innovación and the FEDER through the Plan Nacional de Investigación Científica, Desarrollo e Innovación Tecnológica, grant MTM2009-13248.

References

[1] M. González, R. Martínez, and M. Slavtchova-Bojkova, "Stochastic monotonicity and continuity properties of the extinction time of Bellman–Harris branching processes: an application to epidemic modelling", *J. Appl. Probab.*, v. 47(1), in press, 2010.
[2] P. Haccou, P. Jagers, and V. Vatutin, "Branching processes: variation, growth and extinction of populations", *Cambridge University Press*, 2005.
[3] M.E.. Halloran, J.R. Longini, M. Ira, and C.J. Struchiner "Design and analysis of vaccine studies", *Springer-Verlag*, 2010.

SPATIAL DISPERSAL AND PROLONGED DIAPAUSE IN A PATCHY STOCHASTIC ENVIRONMENT

S. Gourbière, **T. Mahdjoub** and **F. Menu**

Spatial dispersal and prolonged diapause are two major traits in evolution and populations dynamics. Often considered as being two alternative strategies, the simultaneous impacts of these two traits on the population growth rate are little studied. The growth rate of a metapopulation, made up of two populations connected by a certain dispersal rate, living on two different qualities patches, is determined by application of the "vec- permutation" method [1]. The growth rate variations are studied in two stages populations, under a constant environment then a stochastic one. Results show that, in the case of a constant environment, the growth rate is optimal when the metapopulation is with simple diapause and the dispersal rate is null. However, under a stochastic environment, the mean stochastic growth rate is optimal for mixed prolonged diapause strategies (bet-hedging) and a non null dispersal rate.

EXCLUSION FROM FORCED ECO-EPIDEMIOLOGICAL SYSTEMS.

[a]J. V. Greenman and [b]A. S. Hoyle

[a]Department of Computing Science and Mathematics,
University of Stirling, Stirling, FK9 4LA, Scotland, UK.
j.v.greenman@stir.ac.uk

[b]Department of Computing Science and Mathematics,
University of Stirling, Stirling, FK9 4LA, Scotland, UK.
ash@maths.stir.ac.uk

The urgency in understanding the dynamics of complex eco-epidemiological systems is due in part to the increasing occurrence of virulent pathogens crossing species boundaries, often a concomitant of local or global environmental change [1]. The complexity is compounded when there is vertical as well as horizontal interaction between species and when the system is periodically or stochastically forced by environmental fluctuations. We focus on the problem of how best to exclude a pathogen or species from the system. This means exploring the properties of the network defined by the system's exclusion thresholds. This can be done geometrically (and often algebraically) at low dimensions or by nonlinear constrained optimisation theory in general. The impact of external forcing complicates the problem by distorting the exclusion thresholds [2]. This distortion is highly sensitive to the lags between forcing components, This sensitivity can be exploited by management using correctly lagged cyclically varying controls to reduce the effort involved in pathogen or species exclusion under periodic forcing. Applications to a range of ecological systems are provided to illustrate the underlying theory and show how exclusion can be achieved and also what can happen if control is mismanaged [3].

References

[1] A. Dobson, "Population dynamics of pathogens with multiple host species", American Naturalist v. 164, p. S64-S78, 2008.

[2] J. V. Greenman and R. A. Norman, " Environmental forcing, invasion and control of ecological and epidemiological systems", Journal of Theoretical Biology v. 247, p. 492-506, 2007.

[3] M. Choisy and P. Rohani, "Harvesting can increase severity of wildlife disease epidemics", Proceedings of the Royal Society London B v. 273, p. 2025-2034, 2006.

Keywords: exclusion threshold, ecological control, nonlinear constrained optimisation, environmental forcing, resonance, coexistence.

MATHEMATICAL MODEL: STEM CELLS AND TUMOR DEVELOPMENT

N.M. Gualberto-Leite[a], **M.E. Gouvêa**[b], and **C.H.C. Moreira**[c]

[a]Instituto Federal Norte de Minas Gerais
Campus Januária
Fazenda São Geraldo, Km 6, s/n, Januária, 39480-000, MG, Brazil
neilaleite@yahoo.com.br

[b]Instituto Federal de Minas Gerais
Campus Formiga
Rua Padre Alberico, s/n, São Luiz, Formiga, 35570-000, MG, Brazil
meg@dppg.cefetmg.br

[c]Departamento de Matemática
Universidade Federal de Minas Gerais
Av. Antônio Carlos 6627, Pampulha, Belo Horizonte, 31270-901 MG, Brazil

Keywords: Cancer; Hallmarks of cancer; Genetic instability; Hayflick limit

We present a mathematical model based on a hierarchical architecture composed of three compartments: the stem cells, the early progenitors and the specialized cells. The model assumes that tumorigenesis depends on a sequential accumulation of mutations that give the cell some selective advantages. The parameters of the model, that is, self-renewal, mitosis, differentiation and mortality rates assume constant values that account for physiological and environmental effects. The results obtained show that mutations affecting the earlier lineage, the stem cell compartment, are more deleterious and lead to a faster development of a large population of abnormal cells. The model treats four of the six hallmark capabilities that a normal cell must acquire [1] to become a cancer cell: self-sufficiency in growth signals, insensitivity to anti-growth signals, evasion of apoptosis and limitless replicative potential. Our results show that cancer develops faster through a particular ordering starting with the mutation that disrupts genetic stability. This conclusion is consistent with the results obtained by Michor et al [2], with a linear model for colon cancer initiation, and by Ashkenazi et al [3], through a mathematical model considering a hierarchical structured tissue. Ashkenazi et al also conclude that mutations that change the stem cell division pattern (symmetrical or anti-symmetrical) or lead to the acquisition of some degree of immortality in progenitors are alternatives for the early onset of cancer. A novel result of our model is the behavior of the mutated population of specialized cells when, due to the accumulation of mutations, they become able to avoid senescence and can undergo an unlimited number of mitosis. In this case, the combination of mitosis and mortality rates can lead to an explosion of the tumor. Therefore, our model suggests that mature mutated cells that acquire the capability of evading telomerase can give the most important contribution to a devastating tumor evolution.

Acknowledments The authors are grateful to FAPEMIG - (*Fundação de Amparo à Pesquisa do Estado de Minas Gerais - Brasil*) for financial support.

References

[1] D. Hanahan and R. A. Weinberg, “The Hallmarks of cancer" - *Cell*, v. 100, p. 57-70, (2000).
[2] F. Michor, Y. Iwasa, H. Rajagopalan, C. Lengauer and M.A. Nowak, "Linear model of colon cancer initiation", Cell Cycle, v. 3, p. 358-362, 2004.
[3] R. Ashkenazi, S.N. Gentry and T.L. Jackson, " Pathways to tumorigenesis - Modeling mutation acquisition in stem cells and their progeny" -*Neoplasia*, v. 10, p. 1170-1182, (2008).

Equations with infinite delay as models for physiologically structured populations

M. Gyllenberg

A large class of structured population models can be formulated as delay equations (Volterra equations) coupled with delay-differential equations. In many models the delay is infinite and this leads to mathematical complications due to non-compactness. In this talk I present recent results on (in)stability of steady-states of such models.

The talk is based on joint work with Odo Diekmann.

An Ecoepidemiological Model In Two Competing Species

Mats. Gyllenberg, **Xiaoli. Liu**, and **Ping. Yan**

Department of Mathematics and Statistics
University of Helsinki
P.O. Box 68 FI-00014 University of Helsinki
firstname.lastname@helsinki.fi

Abstract. The population size of species is affected not only by ecology interaction, but also by the infectious disease. In this place, we propose a simple model combing competition and disease, and try to understand how the disease affect the competing species. We assume that only one species suffers the disease, and the infected individuals do not give reproduction. Additionally, we assume that the infected individuals have the same competition strength as the susceptibles but suffer disease induced death rate. It is proved that if in the absence of the disease, there is competitive exclusion between two species, then the introduction of the disease can lead to stable or oscillatory coexistence of both species, which are partially determined by the basic reproduction number $\hat{R}_0$. If in the absence of the disease, the two species can coexist, then the introduction of the disease are determined by another basic reproduction number R_0. More If $R_0 > 1$, then the unique positive equilibrium is globally stable under certain conditions, while if $R_0 < 1$, then the disease will die out eventually.

Key words. Lotka-Volterra Competition, Hopf bifurcation, basic reproduction number, globally stable

EVOLUTIONARY INSIGHTS FROM SEMI-DISCRETE PLANT EPIDEMIC MODELS

F.M. Hamelin[a], **M. Castel**[a] and **L. Mailleret**[b]

[a]UMR 1099 BiO3P, INRA - Agrocampus - Univ. Rennes 1
65 rue de St Brieuc, CS 84215, 35042 Rennes Cedex
fhamelin@agrocampus-ouest.fr, castel@agrocampus-ouest.fr

[b]UR 880 URIH, INRA
400 route des Chappes, BP 167, 06903 Sophia Antipolis Cedex
ludovic.mailleret@sophia.inra.fr

Keywords: epidemiology, seasonality, adaptive dynamics, invasibility analysis

Many cropping systems are seasonal and involve abrupt changes in plant density due to planting and harvesting, as periodic impulses. *Semi-discrete* modelling, which is used in various fields of mathematical biology [1], is the way one usually deals with seasonal cropping in plant epidemiology [2]. We are interested in the evolutionary epidemiology of crop plant pathogens, as recently addressed by van den Berg et al [3].

We consider a classical SIR epidemic model in ODE during the n-th cropping seasons of length T:

$$\forall t \in [nT,\, (n+1)T)\,,\ \begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI\,, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \alpha I\,, \end{cases} \tag{10}$$

which is coupled to a difference equation system that depicts the inter-seasonal disease transmission:

$$\begin{cases} S((n+1)T^+) = p((n+1)T)S_0\,, \\ I((n+1)T^+) = (1-p((n+1)T))S_0\,, \end{cases} \tag{11}$$

where the $+$ superscript denotes the instant right after T and p is the probability of not being infected by the inoculum resulting from the current season epidemic at the very beginning of the next cropping season. This formulation and the explicit form of p arise from a mechanistic description of a preliminary primary infection phase (a rather generic feature in plant epidemiology), as originally suggested in [4].

Thus, the model combines the approaches of [4] and of [3] but differs from the latter in that the total host density is kept constant throughout the seasons, however the pathogen survives the intercrop season. This makes explicit the way density dependance acts at the beginning of each season, during the primary infection phase. As a result, evolution unlikely maximizes a quantity such $\mathcal{R}_0$ [5]. We will present first insights arising from evolutionary invasibility analysis, as illustrated by Pairwise Invasibility Plots (PIPs).

References

[1] L. Mailleret, and V. Lemesle, “A note on semi-discrete modelling in the life sciences", *Philosophical Transactions of the Royal Society Series A*, v. 367, p. 4779-4799, 2009.
[2] L.V. Madden, G. Hughes, and F. van den Bosch. “The Study of Plant Disease Epidemics", APS Press, St. Paul, MN, 2007.
[3] F. van den Berg, N. Bacaer, J.A.J. Metz, C. Lannou, and F. van den Bosch, “Periodic host absence can select for both higher or lower parasite transmission rates", preprint, 2009.
[4] L.V. Madden, and F. van den Bosch, “A Population-Dynamics Approach to Assess the Threat of Plant Pathogens as Biological Weapons against Annual Crops", *BioScience*, v. 52, p. 65-74, 2002.
[5] S.D. Mylius, and O. Diekmann, “On evolutionary stable life histories, optimization and the need to be specific about density dependence", *Oikos*, v. 74, p. 218-224, 1995.

MODELING ADAPTATION IN METAPOPULATIONS

I. Hanski, **T. Mononen** and **O. Ovaskainen**

Department of Biosciences
University of Helsinki
P.O. Box 65, FI-00014 University of Helsinki, Finland
tommi.mononen@helsinki.fi

We present a metapopulation model that combines demographic extinction-colonization dynamics and the dynamics of local adaptation. The model is one possible extension of [1]. Our main interest is to analyze the emergence of different patterns of adaptation in a patch network in relation to model parameters, including especially the amount of genetic variance, the strength of selection, the scale of migration, and the difference in the environmental quality of habitat patches. The demographic model is a stochastic patch occupancy model [2], and each population has a mean phenotype modeled with a single value. The two types of dynamics are coupled via the effect of maladaptation (the difference between the optimal and current mean phenotype) on the rate of population extinction, which is modeled using a discrete-time ceiling model of population growth [3]. A newly-established population gets its phenotypic value as a weighted mean of phenotypic values of the source populations.

The stochastic model is approximated by a deterministic difference equation model. The equilibria can be calculated by fast iteration. The approximation is mathematically valid and leads to qualitatively similar results as the original stochastic model.

The model shows four distinct patterns of adaptation. In *local adaptation* each population-specific phenotypic value converges close to the optimal value in the patch. On the contrary, in *network adaptation* all the mean phenotypic values converge close to each other and are intermediate between the patch-specific optimal values. In *network specialization* all mean phenotypic values converge towards the optimal phenotype in one patch type (in which case the populations in the alternative habitat type are very poorly adapted). This pattern may occur at the scale of the entire network but also at the scale of sub-networks, producing *mosaic adaptation*, in which clusters of habitat patches exhibit such specialization, but different clusters show opposite specializations. We analyze the emergence and further qualities of the mosaic adaptation.

References

[1] O. Ronce and M. Kirkpatrick, "When sources become sinks: migrational meltdown in heterogeneous habitats", *Evolution*, v. 55, p. 1520-1531, 2001.
[2] O. Ovaskainen and I. Hanski, "From individual behaviour to metapopulation dynamics: unifying the patchy population and classic metapopulation models", *American Naturalist*, v. 164, p. 364-377, 2004.
[3] R. Lande, "Risks of population extinction from demographic and environmental stochasticity and random catastrophes", *American Naturalist*, v. 142, p. 911-927, 1993.

DYNAMICS OF PRION PROLIFERATION,

FROM BIOLOGICAL EXPERIMENTS TO MATHEMATICAL MODELS

E. Hingant[a,*] and **J.-P. Liautard**[b] and **L. Pujo-Menjouet**[a]

[a]Institut Camille Jordan
CNRS UMR 5208 - Université Lyon 1
43 bd du 11 novembre 1918, 69622 Villeurbanne, France
[*]hingant@math.univ-lyon1.fr

[b]Centre de Recherche sur les Pathogènes et Biologie pour la Santé
Université Montpellier 2

Prion is a protein able to polymerize under certain conditions. Some of the consequences of this polymerization lead to neurodegenerative diseases. Produced naturally in our cells, prion is in a monomer and non pathological form. But sometimes, different protein conformations appear to be pathological and sometimes infectious. This phenomenon can start in a sporadic (spontaneous) way in a body through a stochastic process called nucleation or it can be transmitted through a deterministic process still misunderstood by the biologists.

In close collaboration with a team of experimental biologists, our goal is to understand the mechanisms of polymerization during the nucleation and infectious processes. We particularly make the following hypothesis: different strains of prion diseases, probably due to different conformations of monomers determine their properties leading to different characteristics. In other words, are there strains more infectious than others; or more unstable, more sensitive to the concentration of inoculum, etc. Our objective is to develop different models of prion proliferation to fit as close as possible the biologists' experiments going from discrete model for the "Biacore" experiment - where polymers are anchored on a plaque and put in a constant flow of proteins - to a spatial and size structured model where the dependance on fragmentation with respect to agitation in a solution is studied.

References

[1] M. Doi, and S. F. Edwards, *The theory of polymer dynamics*, Oxford Univ. Press, 1988.
[2] M. L. Greer, L. Pujo-Menjouet, and G. F. Webb, "A mathematical analysis of the dynamics of prion proliferation", *Journal of Theoretical Biology*, v. 242, p. 598-606, 2006.
[3] J. Masel, V. A.A. Jansen, and M. A. Nowak, "Quantifying the kinetic parameters of prion replication", *Biophysical Chemistry*, v. 77, p. 139-152, 1999.

ANALYSIS OF A MODEL FOR TRANSFER PHENOMENA IN BIOLOGICAL POPULATIONS

P. Hinow[a], **F. Le Foll**[b], **P. Magal**[c] and **G. Webb**[d]

[a]Department of Mathematics
University of Wisconsin Milwaukee
Milwaukee, WI USA 53201-0413
hinow@uwm.edu

[b]Laboratory of Ecotoxicology
Université de Le Havre
Le Havre, France
frank.lefoll@wanadoo.fr

[c]Institut de Mathématiques de Bordeaux
Université de Bordeaux
Bordeaux, France
pierre.magal@gmail.com

[d]Department of Mathematics
Vanderbilt University
Nashville, TN USA 37240
glenn.f.webb@vanderbilt.edu

P-glycoprotein (P-gp) is a protein overexpressed in cancer cells that causes multi-drug resistance to cancer therapy. Recent experimental evidence demonstrates that P-gp is transferred directly cell-to-cell in *in vitro* cell cultures of breast tumor cell lines. A mathematical model quantifies the transfer process of P-gp in *in vitro* cultures of MCF-7 human breast adenocarcinoma cells. The model supports the hypothesis that P-gp is transferred directly cell-to-cell and provides a framework for optimizing chemotherapy regimens.

References

[1] P. Hinow, F. Le Foll, P. Magal, and G. Webb "Analysis of a model for transfer phenomena in biological populations", *SIAM J. Appl. Math*, v. 70, p. 40-62, 2009.

OPTIMAL CONTROL OF PATHOGENS IN MULTI-HOST SYSTEMS

Andy Hoyle[a] and **Jon Greenman**[b]

[a]Department of Computing Science and Mathematics
University of Stirling
Stirling, UK
ash@maths.stir.ac.uk

[b]Department of Computing Science and Mathematics
University of Stirling
Stirling, UK
jvg@maths.stir.ac.uk

Increasing concerns about the changing environment and the emergence of pathogens that cross species boundaries (e.g. from wildlife to livestock) have added to the urgency of understanding the dynamics of ever more complex ecological systems infected by pathogens. More importantly are how to control, by which we mean eradicate, these pathogens. Particular questions that arise are which species must have controls applied to them and at what levels. Initially I will talk about a multi-host, shared pathogen system under apparent (indirect) competition and discuss the various methods of finding feasible control strategies, including introducing the concept of control maps. These control maps can be used to plot the network of exclusion (invasion) thresholds, in order to readily identify which control strategies will work and why others will not, and how optimisation techniques can be applied to these problems. I then go on to discuss models where there are direct interactions between the host species, for example predator-prey. These interactions can complicate matters with certain control methods making the problem worse, for example by excluding other species as well as the pathogen. However control maps can again be used to find a solution. These can be expanded to higher dimensions by a computational scheme that will generate the best control strategy. This method is applied to the analysis of parasite exclusion in wildlife systems. However any control method can be dramatically affected by seasonality!

References

[1] J.V. Greenman, and A. Hoyle, "Exclusion of generalist pathogens in multi-host communities", *American Naturalist*, v. 172, p. 576-584, 2008.

2009 pH1N1 Epidemic: Early Onset, Reproduction Numbers, and Turni

Y.-H. Hsieh, **D. Fisman** , **V. Lee**, **W.Y. Lim**, **S. Ma**, and **J. Wu**

The 2009 pH1N1 influenza pandemic posed a great challenge for public health policymakers and medical/scientific research, due to limited information on outbreak/pathogen, stochastic variations of disease epidemiology, and changing case definitions and surveillance practices in each affected country. A simple mathematical model, the Richards model [1-2], and its variants [3-4] are used to fit the cumulative epidemic curves (case, hospitalization, death, etc.) in order to obtain estimates for the turning points of the outbreaks, the basic reproduction number R_0, as well as the final outbreak size for a wave of infections in the absence of new artificial interventions. The procedure is utilized to analyze and compare the past epidemic from the early onset in Mexico, the spring/summer outbreak in Canada, the winter flu season in the Southern Hemisphere countries, and the fall/winter outbreak in Taiwan.

References

[1] Hsieh YH, Lee JY, Chang HL. SARS epidemiology. *Emerging Infectious Diseases* 2004; 10(6):1165-1167.
[2] Hsieh YH and Ma S. Intervention Measures, Turning Point, and Reproduction Number for Dengue, Singapore, 2005. Am J Trop Med Hyg. 2009; 80:66-71.
[3] Hsieh YH, Cheng YS. Real-time forecast of multi-wave epidemic outbreaks. *Emerging Infectious Diseases* 2006; 12(1):122-127.
[4] Hsieh YH, Chen CWS. Turning Points, reproduction number, and Impact of Climatological events on Multi-Wave Dengue Outbreaks. Trop. Med. Internat. Health. 2009; 16(4):1-11.

WITHIN-HOST POPULATION DYNAMICS OF CELL-MEDIATED RESPONSE AGAINST MYCOBACTERIUM TUBERCULOSIS

Eduardo Ibarguen-Mondragon[a] and **Lourdes Esteva**[b]

[a] Departamento de Matemáticas y Estadística, Facultad de Ciencias,
Universidad de Nariño, C. U. Torobajo, Clle 18 - Cra 50, PBX 27311449, Pasto, Colombia
Posgrado en Ciencias Matemáticas, Universidad Nacional Autónoma de México, 04510 México, DF.
edbargun@udenar.edu.co

[b] Departamento de Matemáticas, Facultad de Ciencias,
Universidad Nacional Autónoma de México
Delegación Coyoacan, 04510, México, DF. Tel-facsimile number 56224866
lesteva@matematicas.unam.mx

The world resurgence of the Tuberculosis has outlined the necessity to improve strategies for controlling the incidence. Recently, it has become clear that, in order to develop a more efficient vaccine, a better understanding of the relation between the immune response of the host and the tubercle bacillus is needed. Cell mediated response against Tuberculosis plays a fundamental role in the outcome of *Mycobacterium tuberculosis* infection. We formulate and analyze a mathematical model for cellular immunology of Tuberculosis. From the analysis of the model, equilibria and local stabilities are determined. Among interesting dynamical behaviors of the model exist forward and backward bifurcations which raises many new challenges to effective infection control.

Keywords: mathematical model, tuberculosis, cell mediated response, forward and backward bifurcations.

References

[1] JM. Davis, L. Ramakrishnan, "The role of the granuloma in expansion and dissemination of early tuberculous infection", *Cell*, v. 136(1), p. 37-49, 2009.
[2] D. Sud, C. Bigbee, JL. Flynn, and DE. Kirschner,"Contribution of CD8+T Cells to Control of Mycobacterium tuberculosis Infection", *The Journal of Immunology*, v. april 1;176(7)1, p. 4296- 4314, 2006.
[3] Palomino-Leäo-Ritacco, Tuberculosis 2007, From basic science to patient care. Tuberculosis- Textbook.com, first edition.
[4] J. Alavez, J. Avendaño, L. Esteva, J. Florez, J. Fuentes, G. García, G. Gómez, J. López, "Within-host population dynamics of antibiotic-resistant M. tuberculosis". *Mathematical Medicine and Biology*, v. 24(1), p. 35-56, 2006.
[5] JG. Egen, AG. Rothfuchs, CG. Feng, N. Winter, A. Sher, RN. Germain, "Macrophage and T cell dynamics during the development and disintegration of mycobacterial granulomas", *Immunity*, Feb;28(2):271-84, 2008.

Estimate of viral productivity and infectivity *in vitro*

S. Iwami[a1, a2, a3 *], **Y. Takeuchi**[b], **T. Igarashi**[c] and **T. Miura**[c]

[a1] PRESTO (PRESTO: Precursory Research for Embryonic Science and Technology),
Japan Science and Technology Agency (JST),
[a2] Graduate School of Mathematical Sciences,
The University of Tokyo,
[a3] Institute for Virus Research,
Kyoto University,
3-8-1 Komaba Meguro-ku Tokyo 153-8914, Japan
siwami@ms.u-tokyo.ac.jp* (Corresponding author)

[b] Graduate School of Science and Technology,
Shizuoka University
3-5-1 Johoku Naka-ku Hamamatsu Shizuoka 432-8561, Japan
takeuchi@sys.eng.shizuoka.ac.jp

[c] Institute for Virus Research,
Kyoto University,
53 Shogoinkawaramachi Sakyo-ku Kyoto 606-8507, Japan
tigarash@virus.kyoto-u.ac.jp, tmiura@virus.kyoto-u.ac.jp

Keywords: HIV/SIV/SHIV, Mathematical model, Viral experiment, Viral infectivity and productivity

The availability of potent antiviral drug and mathematical models have reveal important viral properties such as half-life of HIV virion, short and long lived productively infected cells for the last two decade. These findings really improved our understanding of HIV infection and the strategy of drug therapy. However, even now, we do not have any analytical methods to estimate viral productivity (virus burst size) and infectivity (infection rate). In this talk, I am going to show our mathematical and experimental approach for the estimation.

Our conceptual idea is that very simple viral experiment can be explained by a mathematical model. We use the following mathematical model to describe *in vitro* viral replication experiment:

$$x' = -dx - \beta xv, \quad y' = \beta xv - ay, \quad v' = ky - rv \quad (1).$$

Here x, y, v are the number of target cells, infected cells, and virions, respectively. If we can completely inhibit *de novo* replications (i.e., $\beta = 0$), then model (1) is reduced to

$$y' = -ay, \quad v' = ky - rv \quad (2).$$

Because model (2) is linear differential equations, we can obtain the explicit solution and then the viral productivity (k) is

$$k = \frac{\left(v(t_2) - v(t_1)e^{-r(t_2-t_1)}\right)(r-a)}{y(t_1)(e^{-a(t_2-t_1)} - e^{-r(t_2-t_1)})} \quad (3).$$

On the other hand, for example, if the time-scale of virion is sufficiently faster than that of the infected cells, then we can find a constant of motion for model (1) and therefore can derive the viral infectivity (β) as follows:

$$\beta = \frac{a \log x(t_2)/x(t_1) + d \log v(t_1)/v(t_2)}{k/r\left(x(t_2) - x(t_1)\right) + v(t_1) - v(t_2)} \quad (4).$$

Here t_1 and t_2 are arbitrary times during the experiment.

Based on the theory of the formulations (3) and (4), we have designed and developed *in vitro* viral replication system and are trying to estimate the viral productivity and infectivity.

THE BASIC REPRODUCTION NUMBER FOR INFECTIOUS DISEASES IN HETEROGENEOUS ENVIRONMENTS

H. Inaba

During the last two decades, the definition and computing methods for the basic reproduction number R_0 for infectious diseases have been widely accepted and developed to become a most important keystone of the mathematical epidemiology. However, R_0 is originally defined by renewal systems with time-independent parameters describing the disease invasion process, it can not be applied to formulate the threshold principle for infectious diseases in time-heterogeneous environments, which are described by non-autonomous dynamical systems.

Recently, several authors ([1], [2], [4]) proposed some ideas to extend the definition of R_0 to the case of infectious diseases in periodic environments. In a most general abstract setting, Thieme [3] formulated the next generation operator (NGO) for infinite-dimensional non-autonomous dynamical system based on the idea of evolutionary semigroups, and has successfully established the threshold relation between the extended R_0, which is given as the spectral radius of the NGO, and the spectral bound of the evolutionary system describing the linear population dynamics in heterogeneous environments.

In my talk, I would like to propose another approach to the definition of R_0 in heterogeneous environments. First we review existing ideas of the definition of R_0 in heterogeneous (in particular, periodic) environments. Next we reconstruct the definition of R_0 introduced by [4] for the ODE model and by [1] for the infection-age dependent model and show the threshold principle that the sign of $R_0 - 1$ determines the sign of the intrinsic growth rate. Moreover we formulate R_0 for the age-duration dependent SIR model with periodic coefficients. Our arguments depend on the weak ergodicity of the positive evolutionary system and the existence of exponential solution, so we can avoid heavy mathematical equipments as the evolutionary semigroup, and clear the basic idea underlying the extended R_0 and the NGO. Its advantage is to be able to determine the dominant exponential solution representing the asymptotic behavior of the basic system, the price we pay is to assume the irreducibility of the evolution process.

SEMICONSERVATIVE QUASISPECIES EQUATIONS FOR POLYSOMIC GENOMES: THE GENERAL CASE

Eran Itan[a] and **Emmanuel Tannenbaum**[b]

[a]Department of Chemistry
Ben-Gurion University of the Negev
Beer Sheva 84105 ISRAEL
itan@bgu.ac.il

[b]Department of Chemistry
Ben-Gurion University of the Negev
Beer Sheva 84105 ISRAEL
emanuelt@bgu.ac.il

This Research develops a formulation of the quasispecies equations appropriate for polysomic, semi-conservatively replicating genomes. This research is an extension of previous work on the subject, which considered the case of haploid genomes. Here, we develop a more general formulation of the quasispecies equations that is applicable to diploid and even polyploid genomes. Interestingly, with an appropriate classification of population fractions, we obtain a system of equations that is formally identical to the haploid case. As with the work for haploid genomes, we consider both random and immortal DNA strand chromosome segregation mechanisms. However, in contrast to the haploid case, we have found that an analytical solution for the mean fitness is considerably more difficult to obtain for the polyploid case. Accordingly, whereas for the haploid case we obtained expressions for the mean fitness for the case of an analogue of the single-fitness-peak landscape for arbitrary lesion repair probabilities (thereby allowing for non-complementary genomes), here we solve for the mean fitness for the restricted case of perfect lesion repair.

References

[1] J. Cairns," Mutation, selection and cancer", Nature 255, 197, 1975.
[2] C.S. Potten, G. Owen, and D. Booth, "Intestinal stem cells protect their genome by selective segregation of template DNA strands". J. Cell. Sci. 115, 2381 ,2002.
[3] J.R. Merok, J.A. Lansita, J.R. Tunstead and J.L. Sherley, " Cosegregation of Chromosomes Containing Immortal DNA Strands in Cells That Cycle with Asymmetric Stem Cell Kinetics" Cancer Research 62, 6791, 2002.
[4] E. Tannenbaum, J.L. Sherley, and E.I. Shakhnovich, "Evolutionary dynamics of adult stem cells: Comparison of random and immortal-strand segregation mechanisms". Phys.Rev. E 71, 041914, 2005.

Evolution of masting – synchronized and intermittent reproduction of trees

Y. Iwasa, **Y. Tachiki** and **A. Satake**

Trees in mature forests often show intermittent reproduction (masting), synchronized over a long distance. According to the dynamics of the resource reserve of individuals, trees can show a large between-year fluctuation in the seed crop even in a constant environment. Reproduction of different trees may be synchronized if fruit production is limited by the availability of outcross pollen. We study conditions for masting to evolve. Assumptions are: the forest consists of many sites, each occupied by a single canopy tree. After a canopy tree falls, the vacant site (gap) becomes available for recruitment. We first show that masting never evolves if all vacant sites are filled by individuals from seeds produced in the same year. Then we demonstrate that masting can evolve if some seedlings survive for several years, forming a seedling bank on the forest floor, where seeds produced in different years compete for gap acquisition. We also study the effect of specialist seed predators. In the complete absence of seeding bank, masting never evolves even in the presence of seed predators. If seedling survivorship is positive but small, then the presence of seed predators can promote the evolution of masting.

Optimal control for population dynamics of incomplete data

B. Jacob[a] and **A. Omrane**[b]

[a]Department of Mathematics
University of Wuppertal
42097 Wuppertal (Germany)
jacob@math.uni-wuppertal.de

[b]Department of Mathematics, CEREGMIA
University of Antilles-Guyane
97159 Pointe à Pitre Cedex (France)
aomrane@univ-ag.fr

We study the control of a linear model describing the dynamics of a single species population where age structure is an important factor and where initial data are missing or incomplete. Let $A > 0$ be the life expectancy of individuals (the maximum age) and $[0, T]$ be the time interval we are looking at. Denote by

$$Q :=]0, T[\times\Omega\times]0, A[, \quad \Sigma :=]0, T[\times\partial\Omega\times]0, A[, \quad \Omega_T :=]0, T[\times\Omega, \text{ and } \Omega_A := \Omega\times]0, A[.$$

Let then $p(t, x, a)$ be the distribution of the population at time t, located at x and having age a. The evolution of those individuals is here described by the partial differential equation

$$\begin{aligned} \frac{\partial p}{\partial t}(t,x,a) + \frac{\partial p}{\partial a}(t,x,a) + \mu(t,x,a)p(t,x,a) - k\Delta p(t,x,a) &= v(t,x,a), \quad (t,x,a) \in Q, \\ \frac{\partial p}{\partial \nu}(t,x,a) &= 0, \quad (t,x,a) \in \Sigma, \\ p(t,x,0) &= \int_0^A \beta(a)p(t,x,a)\,da, \quad (t,x) \in \Omega_T, \\ p(0,x,a) &= p_o(x,a), \quad (x,a) \in \Omega_A, \end{aligned} \tag{12}$$

where $p_o(x, a) \in L^2(\Omega_A)$ is unknown. Further, v is a control function, which we want to choose in such a way that the state function p approaches a given measurement for $t > 0$; i.e. Minimize

$$J(v, p_o) := \Big\|p(v, p_o) - p_g\Big\|^2_{L^2(Q)} + N\Big\|v\Big\|^2_{L^2(Q)}, \tag{13}$$

where p_g is the given observation, $N > 0$. We are concerned with optimal controls $v \in \mathcal{U} := L^2(Q)$ of the population dynamics problem (12)-(13) with incomplete initial data p_o, i.e.

$$\inf_{v\in\mathcal{U}} J(v, p_o) \qquad \forall\, p_o \in L^2(\Omega_A).$$

Since the vector space $L^2(\Omega_A) \neq \{0\}$, the above minimization problem has no sense (L^2 having infinite elements). A natural idea is then to solve the *minmax* problem

$$\inf_{v\in\mathcal{U}} \left(\sup_{p_o\in L^2(\Omega_A)} J(v, p_o) \right). \tag{14}$$

But here, J is not upper bounded; indeed

$$\sup_{p_o\in L^2(\Omega_A)} J(v, p_o) = +\infty.$$

One then has to look for another idea!

References

[1] B. Jacob, and A. Omrane, "Optimal control for age-structured population dynamics of incomplete data", *J. of Math. Anal. and Appl. (to appear).*

THE OPTIMAL EXPLOITATION OF A MODEL FOR COMMERCIAL FISHING

C. Jerry[a] and **N. Raissi**[b]

[a]Department of Mathematics and Informatics
Ibn Tofail University
Equipe E.I.MA, Department of Mathematics and Informatics,
Faculty of Science, Ibn Tofail University,
B.P 133, Kenitra, Maroc
jechakib@yahoo.fr

[b]Department of Mathematics and Informatics
Ibn Tofail University
Equipe E.I.MA, Department of Mathematics and Informatics,
Faculty of Science, Ibn Tofail University,
B.P 133, Kenitra, Maroc
n.raissi@mailcity.com

A non-linear dynamic model, as in the paper of A. Leung and Ar-Y. Wang, in two state variables and one control presented for the purpose of finding the optimal combination of exploitation and capital investment in the commercial fishing industry. This optimal combination is determined in terms of management policies. Exploitation and capital are controlled through the rate of exploitation. A novel feature in this model is that the variation of the capital depends on the income.
Key Words: Rate of discount, Capital investment, Management policy.

References

[1] A. Leung, and AR-Y. Wang, "Analysis of Models for Commercial Fishing: Mathematical and Economical Aspects", *Econometrica*, v. 44, N. 2, p. 295-303, 1976. [2] G.R. Munro, "The Optimal Management of Transboundary Renewable Resources", *Canadian Journal of Economics*, v. 12, N. 3, p. 355-376, 1979. [3]C.W. Clark, "Mathematical Bioeconomics: the Optimal Management of Renewable Resources", *2nded.: A Wiley-Interscience*, 1990. [4] CW. Clark, and G. Munro, "The Economics of Fishing and Modern Capital Theory: A Simplified Approach", *J. Environmental Econom. and Management*, v. 2, p. 92-106, 1975. [5] F.H. Clarke, "The Maximum Principle under Minimal Hypotheses", *SIAM J. Control Optim.*, v. 14, p. 1078-1091, 1976. [6] C. Jerry , and N. Raïssi , "Can management measures ensure the biological and economical stabilizability of a fishing model? ", *Mathematical and Computer Modelling*, v. 51, p. 516-526, 2010.

A VIABILITY ANALYSIS FOR AN EXPLICIT INSHORE-OFFSHORE MODEL

M. Jerry[a], **N. Raissi**[b] and **A. Rapaport**[c]

[a]Department of Economy
Université Sultan Moulay Slimane
Faculté Polydisciplinaire de Béni Mellal, Maroc
jemounir@yahoo.fr

[b]Department of Mathematics and Informatics
Ibn Tofail University
Equipe E.I.MA, Department of Mathematics and Informatics,
Faculty of Science, Ibn Tofail University,
B.P 133, Kenitra, Maroc
n.raissi@mailcity.com

[c]UMR Analyse des Systèmes et Biométrie
I.N.R.A
INRA, Montpellier, France
rapaport@supagro.inra.fr

In the exploitation of renewable resources, in particular fisheries, one is often inclined towards looking for the optimization of the net income generated by the exploitation (i.e. the best steady state or optimal management strategies). Such objectives bring a base of valuable knowledge in order to determine the impact and the efficiency of control theoretic strategies and deduce from them future decisions for rational stock management.

Nevertheless, directives established by such studies are rarely directly applied in practice, especially in the fishermen community. Asking fishermen to follow a precise strategy for reaching desirable objectives without taking into account external environment (climate change, nourishment shortage ...etc) is of course unrealistic. In addition, optimal decisions are based on models with a lot of simplifications in face of real world (several assumptions are chosen for making the optimization problem solvable). In the present work, we abandon the idea of determining a precise strategy optimizing a given criterion. Instead, we are looking for satisfactory management policies, in the sense that it should be easy to be followed by fishermen and of valuable interest on both economic and ecological levels. From the mathematical point of view, we aim at determining a viability domain that ensures the stock durability to protect the environment and afford a minimum income for fishermen. Instead, we emphasize analytical determinations for this precise problem with the help of elementary geometric considerations.
Key Words: Inshore-offshore model; subpopulations; viability Kernel.

References

[1] D. Ami, P. Cartigny and A. Rapaport, "Can marine protected areas enhance both economic and biological situations? ", *Comptes Rendus Biologies*, v. 328, p. 357-366, 2005. [2] C. W. Clark, "Mathematical Bioeconomics: The optimal Management of Renewable Resources (second ed.)", *John Wiley and Sons, New York*, 1990. [3] L. Doyen and C. Bene, "Sustainability of fisheries through marine reserve: a robust modeling analysis ", *Journal of Environmental Management*, v. 69, N. 1, p. 1-13, 2003. [4] M. Jerry, A. Rapaport and P. Cartigny, "Can protected areas potentially enlarge viability domains for harvesting management?", *Nonlinear Analysis: Real World Applications*, v. 11, p. 720-734, 2010.

EPIDEMICS AND DIFFUSION ON COMPLEX NETWORKS

D. Juher[a], **J. Ripoll**[a] and **J. Saldaña**[a]

[a]Departament d'Informàtica i Matemàtica Aplicada
Universitat de Girona
Campus Montilivi, 17071 Girona, Spain
juher@ima.udg.edu, jripoll@ima.udg.edu, jsaldana@ima.udg.edu

Realistic epidemic models take into account some spatial heterogeneity. The approach of complex networks can be suitable to deal with sets of local populations connected among themselves by diffusion/migration. We have extended our previous works ([1] and [2]) to the model:

$$\frac{d}{dt}\,\rho_{S,k}(t) = \rho_{I,k}\left(\mu - \beta\, c(\rho_k)\frac{\rho_{S,k}}{\rho_k}\right) - D_S\left(\rho_{S,k} - k\sum_{k'} P(k'|k)\,\frac{1}{k'}\,\rho_{S,k'}\right), \quad (1)$$

$$\frac{d}{dt}\,\rho_{I,k}(t) = \rho_{I,k}\left(\beta\, c(\rho_k)\frac{\rho_{S,k}}{\rho_k} - \mu\right) - D_I\left(\rho_{I,k} - k\sum_{k'} P(k'|k)\,\frac{1}{k'}\,\rho_{I,k'}\right), \quad (2)$$

which combines the movement of individuals among patches (nodes) with an epidemic dynamics of SIS type within each patch. Now the system includes a density-dependent contact rate $c(\rho_k)$ defined as the number of contacts an individual makes in a local population of size $\rho_k = \rho_{S,k} + \rho_{I,k}$. The state variables refer to the average number of susceptible and infected individuals in patches of degree k. We have in mind three cases: limited or non-limited transmission, and a saturating contact rate like $c(\rho_k) = c_0\rho_k/(c_0 + \rho_k)$ which provides an interpolation between the two former cases. The spatial structure is described by both the connectivity (degree) distribution, $p(k)$, and the conditional probabilities $P(k'|k)$ that a patch of degree k has one connection (link) to a patch of degree k'. The total number of individuals is conserved at the metapopulation level (consistency condition).

The disease-free equilibrium is determined by the diffusion process and it is explicitly given by $\rho^*_{S,k} = \frac{k}{\langle k\rangle}\rho^0$, where ρ^0 is the initial average number of individuals per patch and $\langle k\rangle = \sum_k k\,p(k)$ is the average network degree. We can define the basic reproduction number for local populations of connectivity k as

$$\mathcal{R}_{0,k} = \frac{\beta\, c(\rho^*_{S,k})}{\mu + (1 - P(k|k))D_I} \quad (3)$$

whereas for the whole network the basic reproduction number $\mathcal{R}_0$ is computed as the spectral radius of the matrix

$$\frac{\beta}{\mu + D_I}\,\mathrm{diag}\left(c(\rho^*_{S,k})\right)\left(Id - \frac{D_I}{\mu + D_I}\,\mathcal{C}\right)^{-1}, \quad (4)$$

where $\mathcal{C}$ is the connectivity matrix, i.e. $\mathcal{C}_{kk'} = \frac{k}{k'}\,P(k'|k)$, and $\mathrm{diag}(a_k)$ stands for a diagonal matrix whose k-th element is a_k. The endemic equilibrium is also characterized and a linear stability analysis is performed. Finally, we have implemented several Monte Carlo simulations which show a good agreement with the analytical predictions of the model.

Keywords: Complex metapopulations, infectious diseases, reaction-diffusion processes.

References

[1] D. Juher, J. Ripoll, and J. Saldaña, Analysis and Monte Carlo simulations of a model for the spread of infectious diseases in heterogeneous metapopulations, *Physical Review E* 80, 041920 (2009).
[2] J. Saldaña, Modelling the spread of infectious diseases in complex metapopulations, *Math. Model. Nat. Phenom.* (2010).

ON MODELS OF EARLY BIOLOGICAL EVOLUTION: DOES THE ERROR THRESHOLD REALLY EXIST?

G. Karev

A modification of the Eigen model of molecular evolution with site-specific mutation rate, selective advantage and the cost of error is proposed. We reconsider and generalize a well-known model of mutation-selection balance in infinite population with additive fitness landscape; there are clear biologically plausible assumptions that lead to this model. The principal point is that a system can evolve toward increasing complexity if and only if increase of sequence length results in an increase in fitness. The model is solved in explicit form. The error threshold catastrophe occurs only if the increase in sequence length is not accompanied by a selective advantage. A population goes extinct if the "error cost" exceeds a specified threshold. At fixed sequence length the error catastrophe implies population extinction and hence is non-observable. The error catastrophe may occur when the lengths of sequences increase indefinitely but the required conditions are more restrictive than those given by Eigen. The stochastic version of the model, which describes the evolution of finite populations, is also studied.

References

[1] E. Baake, and H. Wagner, Mutation-selection models solved exactly with methods of statistical mechanics, Genet. Res., v.78, p. 93-117, 2001.

[2] M. Eigen, Self-organization of matter and the evolution of biological macromolecules, Naturwissenschaft, v.58, p.465-523, 1971.

[3] T. Wiehe, Model dependency of error thresholds: the role of fitness functions and contrasts between the finite and infinite sites models, Genet. Res. Camb. v.69, 127-36, 1997.

TRIMORPHIC SPECIALIST-GENERALIST COEXISTENCE ON TWO SPATIAL RESOURCES

I. Karonen

To investigate the effect of spatial heterogeneity on diversity, I've examined the effects of adding exogenous environmental variation to a simple lattice contact process model of a viscous spatial population.

I find that simply dividing the lattice sites into two types, on which individuals have different establishment probabilities, can permit the stable coexistence of three (or more) strains differing in their degree of specialization for one of the two site types. I further find that such trimorphic coexistence is not only evolutionarily stable, but can be reached via evolutionary branching from an initial monomorphic state.

Very rich ecological and evolutionary dynamics appear to emerge particularly when site types are strongly positively correlated, with ecological interactions between competing strains amplifying the dispersal barriers created by the exogenous environment and leading to the formation of locally isolated and adapted subpopulations stable over significant timescales.

Continuous mathematical model for hermaphrodite population dynamics

A. KEBIR[a], **S. BEN MILED**[b] and **M. L. HBID**[c]

[a]DIMACS
RUTGERS University
New jersey, USA
amira.kebir@gmail.com

[b]Institut Pasteur de Tunis & ENIT-LAMSIN
Tunis, Tunisia
slimane@pasteur.rns.tn

[c]LMDP
Faculté des sciences Semlalia, Cadi Ayyad University
Marrakesh, Morocco
hassan.hbid@gmail.com

Keywords: Population dynamics, Hermaphrodite, PDE, Solution, stability.

In this work, we introduce and analyze a model structured by size and by sexual maturity for the sequential hermaphroditism, such as the grouper. This model is in the framework of continuously structured population models with sexual reproduction. The formulated model is a system of non linear partial differential equations. For instance to analyze this model, we took an approach similar to that developed in the book of G. Webb [3]. At the moment, results of existence and uniqueness were obtained, by introducing additional hypothesis, namely, suitable Lipschitz conditions on the birth function or on the sex allocation function and the mortality rate. We show the existence and uniqueness of global solutions which are non-negative and biologically meaningful for the present model. By following we have studied the stationary problem. We have found that the existence and the stability of the positive equilibrium solutions are very linked to the form of the sex allocation function. After that, by the stability analysis of the trivial solution, we have determined that the extinction equilibrium is always locally asymptotically stable, displaying the Allee effect ([2] and [1]). On the other hand, we have determined a sufficient condition to have a global extinction this result is a classical statement in population dynamics which means that if the minimum mortality exceeds the maximum fertility then there is no possibility of non-trivial dynamics.

References

1. W.C. Allee, Animal Aggregations: A Study in General Sociology, *University of Chicago Press*, 1931.
2. W.C. Allee, Cooperation among animals, *Henry Schuman* , 1951.
3. G.F Webb, *Theory of nonlinear age-dependent population dynamics*, Pure and Applied Mathematics, Marcel Dekker Inc., New York and Basel, 1985.

SURNAMES AND SPECIES, GENERA AND GENOMES: THE ULTIMATE NEUTRAL MODEL EXPLAINS (ALMOST) EVERYTHING

D. Kessler

Deducing quality (e.g., fitness) from success (large abundance, long lifetime, high returns on investments) is a common feature of many eco, econo and bio-theories; in particular it sets the conceptual framework for the theory of evolution by means of natural selection. Huge differences in success among species, with an uneven, fat-tailed distribution of abundance, seems to support these claims. Many heretics, however, suggest neutral models in which the main factor beyond success is merely luck. The uneven statistics is attributed to the effect of multiplicative stochastic process (drift). We show that the Kummer statistic, emerges from the neutral birth-death- mutation process, fits marvelously many empirical distributions in complex systems (e.g., species within genera, species abundance ratio, surname frequencies, cluster statistics of trees in the tropical forest and much more).

While previous neutral theories have been focused on the power-law tail, Our method allows explains not only the (noisy) tail but also the typical "shoulder" at small families.

Scale-free networks are subject to slightly different underlying process but the resulting statistics is identical with that obtained for BDM. We thus suggest the BDM distribution as a standard neutral model: effect of fitness and selection are to be identified by substantial deviations from it.

NOVEL EXPONENTS CONTROL THE QUASI-DETERMINISTIC LIMIT OF THE EXTINCTION TRANSITION

D. Kessler and **N. Shnerb**

The quasi-deterministic limit of the generic extinction transition is considered within the framework of standard epidemiological models. The susceptible-infected- susceptible (SIS) model is known to exhibit a transition from extinction to spreading, as the infectivity is increased, described by the directed percolation equivalence class. We find [1] that the distance from the transition point, and the prefactor controlling the divergence of the (perpendicular) correlation length, both scale with the local population size, N, with two novel universal exponents. Different exponents characterize the large N behavior of the susceptible-infected-recovered (SIR) model, which belongs to the dynamic percolation class. Extensive numerical studies in a range of systems lead to the conjecture that these characteristics are generic and may be used in order to classify the high density limit of any stochastic process on the edge of extinction.

A temperature-dependent Leslie model to describe the dynamics of a bullhead (*Cottus gobio*) population

J. Kielbassa[a] and **A. Chaumot**[a] and **M. L. Delignette-Muller**[a,b] and **S. Charles**[a]

[a]Laboratoire de Biométrie et Biologie Evolutive
Université Lyon 1; CNRS, UMR5558
43 boulevard du 11 novembre 1918, F-69622, Villeurbanne, France
{kielbass, scharles}@biomserv.univ-lyon1.fr

[b]Ecole Nationale Vétérinaire de Lyon
1, avenue Bourgelat, F-69280 Marcy l'Etoile, France
ml.delignette@vetagro-sup.fr

The main objective of our study was to develop a temperature-dependent age-class Leslie matrix model, based on experimental field data, in order to predict short and long-term population dynamics of a bullhead population in the Bez River network (Drôme, France), for example in view of water temperature fluctuations. Bullhead, a cryophilic species rarely manipulated by humans, is known for its sensitivity to temperature [1] and is thus an appropriate model organism to study the impact of temperature.

For the purpose of our model, the Bez River network was divided into six sampling sites differing in their thermal conditions. Local temperatures were recorded at all sampling sites in the river. Census was annually performed between 2002 to 2008 providing body length data, while fecundity data were available for 2002 and 2003. The demographic model parameters (fecundity, survival rates) were then calibrated from part of this experimental data (2002 to 2004). Fecundity was directly related to body length by an allometric equation. Adult survival rates were estimated from data of successive years and were in agreement with values provided in the literature. Juvenile survival rates were modelled using a density-dependent function and larval as well as juvenile dispersion processes between sampling sites were taken into account.

We validated our matrix model by predicting the population dynamics in the Bez River network for the years 2005 to 2008 and compared the simulation results to the experimental field data of this period. In order to calculate the age-dependent fecundity, we computed the body lengths of the simulated age classes by using a temperature-dependent von Bertalanffy growth function [2], since previous studies have already shown that temperature had an impact on growth and thus also on reproduction [3]. A global uncertainty analysis was performed by taking into account the uncertainty of all model parameters simultaneously. It allowed us to quantify the uncertainty of the model outputs.

References

[1] D. Pont. Conséquences potentielles du changement climatique sur les biocénoses aquatiques et riveraines françaises. GICC-AQUABIO, 2003.

[2] J. Kielbassa, M. L. Delignette-Muller, D. Pont, and S. Charles. "A temperature-dependent von Bertalanffy growth model applied to bullhead (*Cottus gobio*)". Submitted to Ecol. Model., 2010.

[3] A. Abdoli, D. Pont, and P. Sagnes. "Infuence of female age, body size and environmental conditions on annual egg production of the bullhead". J. Fish Biol., 67 :1327 - 1341, 2005.

Keywords: population dynamics, Leslie matrix, spatial modeling, bullhead (*Cottus gobio*), temperature

SEARCHING FOR THE MOST COST-EFFECTIVE STRATEGY OF CONTROLLING EPIDEMICS

Katarzyna Oleś [a] and **Dr Adam Kleczkowski**[b]

[a]Faculty of Physics, Astronomy and Applied Computer Science
M. Smoluchowski Institute of Physics, Mark Kac Center for Complex Systems Research
ul. Reymonta 4, 30-059 Kraków, Poland

[a]Department of Computing Science and Mathematics
University of Stirling
Stirling FK9 4LA, United Kingdom
kas@cs.stir.ac.uk

[b]Department of Computing Science and Mathematics
University of Stirling
Stirling FK9 4LA, United Kingdom
ak@cs.stir.ac.uk

Models for control of spread of an infectious disease on local, small-world, and random networks are considered. We assume that only partial information is available about the status of individuals as they can be infectious before showing symptoms and thus can avoid detection. We consider various control strategies and search for the most cost-effective one. Traditionally, epidemiology concentrated purely on a reduction of number of cases without considering economic and social costs. If costs are associated with both reactive (hospitalisation) and preventive (vaccination or culling) treatment, three main strategies emerge, with treating all individuals indiscriminately (global strategy), treating only individuals in a well-defined neighbourhood of a detected case (local strategy) and allowing the disease to spread unchecked (null strategy). The choice of the optimal strategy is governed mainly by a relative cost of preventive and reactive treatments. If the disease spreads locally, the local strategy is optimal unless the cost of a single vaccine is much higher than the cost associated with hospitalisation. In the latter case, it is most cost-effective to refrain from prevention. Destruction of local correlations, either by long-range (small-world) links or by inclusion of many initial foci, expands the range of costs for which the null strategy is most cost-effective. The global strategy emerges for the case when the cost of prevention is much lower than the cost of treatment and there is a substantial non-local component in the disease spread. The details of the local control strategy, and in particular the optimal radius of control, are determined by the epidemiology of the disease. The properties of the pathogen might not be known in advance for emerging diseases, but the choice of the strategy can be made based on economic analysis only.

Keywords: epidemiological modelling, disease spread, stochastic modelling, epidemiological control, dispersal patterns.

DIPLOIDY AND THE SELECTIVE ADVANTAGE FOR SEXUAL REPRODUCTION IN UNICELLULAR ORGANISMS

M. Kleiman and **E. Tannebaum**

The existence of sex is among the most intensively studied problems in evolutionary biology. The current predominant theories for the existence suffer from a number of drawbacks, making the question of the evolution and maintenance of sex an open problem in biology. The various theories either require what may be overly restrictive assumptions to obtain a selective advantage for sex, or assumptions that are not realistic. For example, The Deterministic Mutation Hypothesis requires a relatively high mutation rate, and a fitness landscape that exhibits what is known as synergistic epistasis, whereby successive mutations are increasingly deleterious. The first assumption is overly restrictive, since sexual reproduction exists at relatively low mutation rates, and synergistic epistasis may not correspond to the fitness landscapes of actual genomes. Other theories suffer from other drawbacks: The Muller's Ratchet Hypothesis relies on the assumption of small populations, and The Red Queen Hypothesis requires co-evolutionary dynamics generated by host-parasite interactions. Here, we develop mathematical models comparing asexual and sexual reproduction strategies based on the asexual and sexual pathways in Saccharomyces cerevisiae (Baker's yeast), a diploid, unicellular organism that engages in a form of sexual reproduction when stressed. Our modeling assumes semiconservatively replicating, diploid genomes. We find that the explicit consideration of diploidy, a feature lacking in previous models comparing asexual and sexual reproduction, leads to a selective advantage for sexual reproduction under far less restrictive conditions than previous models. In particular, no assumptions need to be made regarding the mutation rate, it is not necessary to assume synergistic epistasis, and no assumptions need to be made about the size of the population or the nature of the environment (i.e. sex has an advantage in a static environment). We also find an intimate connection between diploidy, sex, and recombination. That is, diploidy only has a selective advantage over haploidy with sex, and sex only has a selective advantage under relatively relaxed conditions when the genome is diploid. Furthermore, sex in a diploid organism is only advantageous with meiotic recombination. These results suggest a plausible explanation for the ubiquity of diploidy and its association with sex in biology, as well as for the existence of meiosis in sexually reproducing organisms.

References

[1] M. Kleiman and E. Tannenbaum, "Diploidy and the Selective Advantage for Sexual Reproduction in Unicellular Organisms", *Theory in Biosciences*, 128, 249-285 (2009)

THE DERIVATION AND APPLICATION OF DIMENSIONLESS NUMBERS FOR UNDERSTANDING AND ANTICIPATING THE SHAPES OF VIRAL PHYLOGENIES

K. Koelle , **J. Mattingly**, **V. Pasour** and **O. Ratmann**

High mutation rates and large population sizes of RNA viruses enable them to rapidly evolve, frequently within the lifespan of their hosts [1]. While some of these evolutionary changes are or appear neutral, others are associated with phenotypic change. These include changes in virulence, in viral transmissibility, and in antigenic characteristics that allow for escape from herd immunity. Changes in antigenic phenotype are especially important for understanding the evolutionary dynamics of viruses whose infections are acute and only temporarily immunizing, such as influenza [2- 4], norovirus, respiratory syncytial virus (RSV), and rotavirus. Here, we focus on developing an analytical framework for synthetically understanding the evolutionary dynamics of RNA viruses such as these solely by considering their antigenic phenotypes. Towards this end, we categorize the evolutionary dynamics of viral antigenic proteins into distinct topological sets and derive two dimension less numbers that should, theoretically, be able to distinguish between these viral topologies. Through epidemiological simulations and (preliminary) applications to real host-virus systems, we show that these numbers appear effective at predicting the shapes of viral phylogenies, despite the simplifying assumptions made in their derivation.

References

[1] Grenfell, B.T., et al., Unifying the epidemiological and evolutionary dynamics of pathogens. Science, 2004. 303: p. 327-332.
[2] Ballesteros, S., E. Vergu, and B. Cazelles, Influenza A gradual and epochal evolution: insights from simple models. PLoS One, 2009. 4(10): p. e7426.
[3] Gökaydin, D., et al., The reinfection threshold regulates pathogen diversity: the case of influenza. Journal of The Royal Society Interface, 2007. 4(12): p. 137 - 142.
[4] Koelle, K., et al., Epochal evolution shapes the phylodynamics of influenza A (H3N2) in humans. Science, 2006. 314: p. 1898-1903

Bifurcation theory, adaptive dynamics and DEB-structured populations of iteroparous species

B.W. Kooi[a] and **J. van der Meer**[a,b]

[a]Department of Theoretical Biology, Faculty of Earth and Life Sciences,
Vrije Universiteit, de Boelelaan 1087, 1081 HV Amsterdam, The Netherlands
kooi@bio.vu.nl

[b]Royal Netherlands Institute for Sea Research (NIOZ)
PO Box 59, 1790 AB Den Burg, Texel, The Netherlands
Jaap.van.der.Meer@nioz.nl

A technique to evaluate the evolutionary dynamics of the timing of spawning for iteroparous species is described. The life cycle of the species consists of three life-stages, embryonic, juvenile and adult. The dynamics of the population is studied in a semi-chemostat environment where the inflowing food concentration is periodic (annual). A Dynamic Energy Budget (DEB) [1] based continuous-time model is used to describe the uptake of the food, storage in reserves and allocation of the energy to growth, maintenance, development (embryo's, juveniles) and reproduction (adults). A discrete event process is used for modelling reproduction. At a fixed spawning date of the year the reproduction buffer is emptied and a new cohort is formed by eggs with a fixed size and energy content. The population consists of cohorts: for each year one consisting of individuals with the same age which die after their last reproduction event. The resulting mathematical model is a finite dimensional set of ordinary differential equations (ODE)s with fixed one-year periodic boundary conditions yielding a stroboscopic map. We will study the evolutionary development of the population using the Adaptive Dynamics (AD) [2,3] approach. The trait is the timing of spawning. Pairwise and Mutual Invasibility Plots are calculated. To that end a bifurcation analysis of the stroboscopic map is performed. As bifurcation parameters the traits of the resident and mutant populations are used [4,5]. The Evolutionary Singular Strategy (ESS) value belonging to the evolutionary endpoint for the trait allows for an interpretation of the reproduction strategy of the population. In a case study, parameter values from the literature for the bivalve *Macoma balthica* are used.

References

[1] S.A.L.M. Kooijman. *Dynamic Energy and Mass Budgets in Biological Systems*. Cambridge University Press, Cambridge, 2000.

[2] J.A.J. Metz, S.A.H. Geritz, and R.M. Nisbet. How should we define 'fitness' for general ecological scenarios? *Trends in Ecology & Evolution*, 7:198–202, 1992.

[3] S.A.H. Geritz, É. Kisdi, G. Meszéna, and J.A.J. Metz. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evolutionary Ecology*, 12:35–57, 1998.

[4] B.W. Kooi and T.A. Troost. Advantages of storage in a fluctuating environment. *Theoretical Population Biology*, 70(4):527–541, 2006.

[5] T.A. Troost, B.W. Kooi, and S.A.L.M. Kooijman. Bifurcation analysis of ecological and evolutionary processes in ecosystems. *Ecological Modelling*, 204:253–268, 2007.

Keywords

Adaptive Dynamics, Bifurcation analysis, bivalve *Macoma balthica*, DEB structured model, Iteroparous species

Corresponding author B.W. Kooi

MATHEMATICAL MODELLING OF DIROFILRIOSIS INFECTION

N. Korepanova

Dirofilariosis is a mosquito-borne zoonotic disease caused by different species of dirofilaria (dirofilaria immitis and dirofilaria repens) which reservoirs include many different mammals, specially canids. Human dirofilariosis (HD) is uncommon.
From all existing mathematical models of infections distributed by a disease carrier, Bowman-Gumel and Wonham-de-Camino-Beck models of West Nile fever with a slight modification were chosen. And on basis of the real data of the diseased and dead dogs in our region and the approximate amount of infected and healthy mosquitoes, the dirofilariosis carrier, the different models of infection dynamic were obtained: the model of endemia, the models of the full dogs recovery and death.
The main aim of investigation in this stage is to constract a reliable mathematical model of dirofilariosis that allow to predict the disease evolution. That would give the chance to control the dirofilariosis expansion by the modification of available model parameters such as amount of mosquitos and the host recovery coefficient.

INTERPLAY BETWEEN NETWORK TOPOLOGY AND EPIDEMIC RATES IN THE DYNAMICS OF EPIDEMIC NETWORKS

T. Kostova[a]

[a]National Science Foundation, USA
tvassile@nsf.gov

We present and analyze a discrete-time SIS epidemic network model which represents each host as a separate entity and allows heterogeneous hosts and contacts. We establish a necessary and sufficient condition for global stability of the disease - free equilibrium of the system (defined as epidemic controllability) which defines the epidemic reproduction number of the network. When this condition is not fulfilled, we show that the system has a unique, locally stable equilibrium. We further derive sufficient conditions for control of the epidemic in terms of the epidemic rates and the network topology.

[1] Kostova T., Interplay of node connectivity and epidemic rates in the dynamics of epidemic networks, *Journal of Difference Equations and Applications*, v. 15, 4, 415-428, 2009

ON EIGEN'S MOLECULAR QUASISPECIES MODEL AND GENOTYPE DOMINANCE

T. Kostova[a] and **C. Zhou**[b]

[a]Division of Mathematical Sciences
National Science Foundation
tvassile@nsf.gov

[b]Lawrence Livermore National Laboratory
zhou4@lln.gov

We discuss quasispecies theory and the limitations of Eigen's model [1], [2] with regard to real viral quasispecies [3]. We perform analysis of Eigen's quasispecies model, focusing on the question: what happens with the dominant, most prevalent genotype within a quasispecies, when the mutation rate increases. Rigorous mathematical analysis is possible in the two extremes: when the mutation rate is near zero or when it is close to one. In the first extreme, as expected, the Eigen model predicts that the fittest genotype, i.e., the one which has the highest fitness. In the other extreme, we show that, depending on whether certain relations among the replication and degradation rates are fulfilled, any one of the genotypes could become the dominant type in the quasispecies. We derive formulas that enable exact prediction as to which genotype will emerge as dominant at high mutation rates and demonstrate via numerical examples the validity of the predictions. Importantly, we do not restrict the analysis to the (usually considered) single-peaked fitness landscape, nor to the case with equal degradation rates (as done in previous simulations by other authors) [4].

[1] Eigen M, Selforganization of matter and the evolution of biological macromolecules. *Naturwissenschaften* 58, 465-523, 1971

[2] Domingo, EC Biebricher, M. Eigen, and J. J. Holland, Quasispecies and RNA virus evolution: principles and consequences, Eurekah.com/Landes Bioscience, Georgetown, Texas, 2001

[3] Vignuzzi M, Stone JK, Arnold JJ, Cameron CE, Andino R. Quasispecies diversity determines pathogenesis through cooperative interactions in a viral population, Nature, 439,19, 344-348, 2006

[4] Swetina J. Schuster P, Self-replication with errors – a model for polynucleotide replication , *Biophys. Chem*. 16, 329-345, 1982

COHORTING AND WARD SIZE EFFECTS IN NOSOCOMIAL ROTAVIRUS TRANSMISSION

C. Kribs-Zaleta[a,b] and **S. Charles**[b]

[a]Department of Mathematics
University of Texas at Arlington
Arlington, Texas, USA 76019-0408
kribs@uta.edu

[b]Department of Biometry and Evolutionary Biology
University Claude Bernard Lyon 1
Villeurbanne, FRANCE
scharles@biomserv.univ-lyon1.fr

The annual cost of nosocomial gastroenteritis outbreaks remains high in both lives and healthcare resources, with rotavirus one of the chief causes. In settings such as infant pediatric wards and nursing homes where patient mobility is limited, transmission of infection via contaminated equipment, surfaces and healthcare workers' (HCWs) hands can act as a major vector for such outbreaks. This talk uses a variety of models (deterministic and stochastic dynamical systems) to investigate the effects of cohorting, in which HCWs are assigned to care for disjoint groups of patients, upon nosocomial rotavirus epidemics at levels ranging from a single ward to an entire facility. Discrete-space stochastic models suggest for small populations a reduction in mean prevalence below the level predicted by classical deterministic systems: that is, the simplifying assumption that infected populations can be treated as continuous tends to overestimate mean prevalence of infection in small populations.

Keywords: hospital-acquired infections, rotavirus, stochastic model, dynamical systems, cohorting

MODELING NOSOCOMIAL TRANSMISSION OF ROTAVIRUS IN PEDIATRIC HOSPITAL WARDS

C. Kribs-Zaleta[a,b] and **S. Charles**[b]

[a]Department of Mathematics
University of Texas at Arlington
Arlington, Texas, USA 76019-0408
kribs@uta.edu

[b]Laboratory of Biometry - Evolutionary Biology
UMR CNRS 5558
University C. Bernard Lyon 1
43 boulevard du 11 novembre 1918
69622 Villeurbanne Cedex, FRANCE
scharles@biomserv.univ-lyon1.fr

Nosocomial, or hospital-acquired, transmission of viral and bacterial infections is a major problem worldwide, affecting millions of patients (and causing hundreds of thousands of deaths) per year. These infections are often caused by breaches of infection control practices and procedures, unclean and non-sterile environmental surfaces, and/or ill employees. The causal agents of nosocomial infections include viruses, bacteria, parasites and yeast, but the most frequently encountered are rotavirus, astrovirus or calicivirus, which account for a third of the acute gastroenteritis risk. Among high-risk patients, children under five years are of particular concern to health authorities, as it is believed that most children throughout the world experience at least one episode of acute gastroenteritis due to rotavirus before reaching five years of age.

Our study considers the transmission of rotavirus between patients (young children) and health care workers (HCWs) in a single pediatric hospital ward, with a focus on some specific preventive measures and their likely effects in an environment where differences among individuals (of both types) can have an important impact, here measured through stochasticity. We thus present deterministic and stochastic models for the transmission of rotavirus in a pediatric hospital ward and draw on published data to compare the efficacy of several possible control measures in reducing the number of infections during a 90-day outbreak, including improving compliance with preventive hygiene measures and vaccination. Although recently approved vaccines have potential to curtail most nosocomial rotavirus transmission in the future, even short-term improvement in preventive hygiene compliance following contact with symptomatic patients may significantly limit transmission as well, and remains an important control measure, especially where resources are limited.

Keywords: epidemiological modeling, hospital-acquired infections, single ward model, rotavirus, preventive control measures.

TWO-STRAIN COMPETITION IN SYLVATIC TRANSMISSION OF *T. CRUZI*

C. Kribs-Zaleta[a,b], **A. Mubayi**[a] and **T. Seaquist**[a]

[a]Department of Mathematics
University of Texas at Arlington
Arlington, Texas, USA 76019-0408
kribs@uta.edu

[b]Department of Biometry and Evolutionary Biology
University Claude Bernard Lyon 1
Villeurbanne, FRANCE
scharles@biomserv.univ-lyon1.fr

Two strains of *Trypanosoma cruzi*, the parasite which causes Chagas' disease, are enzootic in the United States among such hosts as raccoons, opossums, and woodrats. One strain appears to have adapted to oral and vertical transmission at the expense of virulence in the more traditional vector-borne route. The cross-immunity conferred by infection with one strain leads to competitive exclusion in a deterministic model (a classical nonlinear dynamical system), which can be used to estimate the evolutionary gains and losses necessary for the adapted strain to win the competition. This talk also presents stochastic analyses (using SDEs and agent-based models) that estimate the probability of the losing (non-adapted) strain persisting in a sylvatic environment. Finally, incorporation of reservoir hosts for the non-adapted strain permits estimation of its residual persistence in hosts for which the strains compete.

Keywords: vector-borne infection, Trypanosoma cruzi, dynamical systems, stochastic models, competition, evolutionary adaptations

A PREDATOR-PREY MODEL WITH ALLEE EFFECT AND FAST STRATEGY EVOLUTION DYNAMICS OF PREDATORS USING HAWK AND DOVE TACTICS

Mgr. Jitka Kühnová[a] and **RNDr. Lenka Přibylová, Ph.D.**[b]

[a]Department of Mathematics and Statistics
Faculty of Science, Masaryk University
Kotlářská 2, 611 37 Brno, Czech Republic
jitka.kuhnova@mail.muni.cz

[b]Department of Mathematics and Statistics
Faculty of Science, Masaryk University
Kotlářská 2, 611 37 Brno, Czech Republic
pribylova@math.muni.cz

In this contribution we present the predator-prey model with Allee effect and hawk and dove tactics in fighting over caught prey implemented as fast strategy evolution dynamics. We extend the work of Auger, Parra, Morand and Sánchez (2002) using the prey population embodying Allee efect and analogously to this work we get two connected submodels with polymorphic and monomorphic predator population. We get much richer dynamics, in each submodel we find local bifurcations (saddle–node and Hopf caused by Allee effect) and a global bifurcation of limit cycles caused by the strategy evolution that is not possible in any of the submodels.

Keywords
Prey–predator model, Allee effect, strategy evolution, bifurcation, hawk–dove evolution.

References
[1] P. Auger, R. B. Parra, S. Morand, E. Sánchez, "A predator–prey model with predators using hawk and dove tactics", *Mathematical Biosciences*, p. 185–200, 2002.

A method for global stability analysis of multigroup epidemic models

Toshikazu Kuniya[a]

[a]Graduate School of Mathematical Sciences
JSRS Research Fellow, the University of Tokyo
3-8-1 Komaba, Meguro-ku, Tokyo 153-8914, Japan
tkuniya1041@akane.waseda.jp

There have been many researches for multigroup epidemic models, in which a heterogeneous population is divided into several homogeneous groups based on individual behaviour. In the present work, we mainly focus on the mathematical properties of several multigroup epidemic models.
For multigroup models in ordinary differential equations, van den Driessche and Watmough (2002) [1] showed that the basic reproduction number R_0, which is defined as the spectral radius of the next generation matrix (Diekmann *et al.* 1990 [2]), is the threshold for local stability of the disease-free equilibrium. In particular, for a multigroup SIR epidemic model, the global stability of each equilibrium have been shown recently by a graph-theoretic approach (Guo *et al.* 2006 [3]).

In this work, we are interested in the applicability of the graph-theoretic approach to several different multigroup epidemic models, which are seemed to have relatively complicated forms. To establish the global stability of equilibria of such multigroup models, we use not only the graph-theoretic approach but also a technique of maximum value functions we develop. One of the models we shall analyze is the following multigroup vaccination model concerned with the vaccine giving a life-long, non-waning, but from the beginning imperfect protection against the disease:

$$\begin{aligned}
\frac{\mathrm{d}S_k(t)}{\mathrm{d}t} &= b_k - \sum_{j=1}^{n} \beta_{kj} S_k(t) I_j(t) - (\mu_k + v_k)\, S_k(t), \\
\frac{\mathrm{d}V_k(t)}{\mathrm{d}t} &= v_k S_k(t) - \sum_{j=1}^{n} \varepsilon \beta_{kj} V_k(t) I_j(t) - \mu_k V_k(t), \\
\frac{\mathrm{d}I_k(t)}{\mathrm{d}t} &= \sum_{j=1}^{n} \beta_{kj} S_k(t) I_j(t) + \sum_{j=1}^{n} \varepsilon \beta_{kj} V_k(t) I_j(t) - (\mu_k + \gamma_k) I_k(t), \qquad 1 \le k \le n,
\end{aligned} \tag{1}$$

where $S_k(t)$, $V_k(t)$ and $I_k(t)$ denote the density of susceptible, vaccinated and infected individuals in group k at time t, respectively. The effect of imperfect vaccination is described by ε $(0 < \varepsilon < 1)$.

We shall further investigate the global stability of equilibria of an age-structured multigroup SIR epidemic model obtained by discretizing the age variable of an age-structured SIR epidemic model in partial differential equations studied by Inaba (1990) [4]. Mathematically, the model can be regarded as a kind of generalization of vaccination model (1).

References

[1] P. van den Driessche, and J. Watmough, “Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission", Math. Biosci. v. 180, p. 29-48, 2002.
[2] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, “On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations", J. Math. Biol. v. 28, p. 365-382, 1990.
[3] H. Guo, M. Y. Li, and Z. Shuai, “Global stability of the endemic equilibrium of multigroup SIR epidemic models", Canadian Appl. Math. Quart. v. 14, p. 259-284, 2006.
[4] H. Inaba, “Threshold and stability results for an age-structured epidemic model", J. Math. Biol. v. 28, p. 411-434, 1990.

Stability of deterministic and stochastic non linear SIRS Epidemic Model

Adil Lahrouz, Hamid El Maroufy

In this paper we consider a non linear SIRS epidemic model in an open population, in wish we have considered a general infection rate function. The exponential and global stabilities are studied for deterministic model. For the stochastic version we have studied the positivity of the solution, we mainly use the theory of Itô stochastic differential equation to established the mean square and almost surely exponential stability. In addition we illustrate the dynamic behavior of the deterministic and stochastic models via a numerical examples.

STUDY OF THE STABILITY OF A RATIO-DEPENDENT ECO-EPIDEMIOLOGY MODEL OF THE SALTON SEA USING THE OPTIMAL DERIVATIVE

F. Lassouani, **T. Benouaz** and **A. Chikhaoui**

Ecology and epidemiology are major fields of study in their own right. Lotka and Volterra established their original works on the expression of predator-prey and competing species relations in terms of simultaneous non- linear differential equations, making the first break through in modern mathematical ecology. Similarly most models for the transmission of infectious diseases descend from the classical SIR model of Kermack and McKendrick. Eco-epidemiology study is becoming important as it involves persistence- extinction threshold of each population in systems of two or more interacting species subjected to parasitism.

In this paper, we consider, a model of three non-linear differential equations consists of susceptible Tilapia fish, infected Tilapia fish and their predator, the Pelican. In our paper we assume that pelicans will consume whatever is available, be it infected or susceptible fish to describe the natural dynamics. We also consider that the functional response for preying the susceptible fish is ratio-dependent [1]. A problem encountered in the study of the stability at the origin of the ratio dependent predator-prey model is the existence of non-regular functions. The usual approach by linearization fails to infer the structure of a neighborhood of O (0, 0) because the origin is a non-hyperbolic equilibrium for any parameter values (i.e., this point has both eigenvalues equal zero).

The aim of this communication is to present a parameter analysis of the stability and global ratio-dependent predator prey model arising in halieutic, using the optimal derivative. This method has been introduced by Benouaz-Arino (1995-2000) and can be applied when the classical linearization cannot be used at the origin. It was based on the principle of least squares which can associate a linear application. It is designed as an alternative to the derivative in the sense of Frechet, essential to the case of equations involving non-regular functions and, in general, not differentiable [2-3]. We also study the stability of this model in the vicinity of the positive interior equilibrium E* using the same method of the optimal derivative. Some computer simulations are presented to illustrate the possibilities giving by this method.

ON THE BEHAVIOUR OF A RUMOUR PROCESS WITH RANDOM STIFLING

E. Lebensztayn[a], **F. P. Machado**[b] and **P. M. Rodríguez**[c]

Department of Statistics
Institute of Mathemathics and Statistics, University of São Paulo
Rua do Matão 1010, CEP 05508-090, São Paulo, SP, Brazil
[a] elcio@ime.usp.br
[b] fmachado@ime.usp.br
[c] pablor@ime.usp.br

In the past decades, there has been great interest in understanding and modelling different processes for information diffusion in a population. Most of the time, the mathematical theory of epidemics is adapted for this purpose. Indeed, one of the earliest works in this field was motivated by the possible similarity in the spread of physiological and psychological infections.

In the classical rumour model introduced by Maki and Thompson (1973), a closed homogeneously mixing population experiences a rumour process. Three classes of individuals are considered: ignorants, spreaders and stiflers. The rumour is propagated through the population by directed contact between spreaders and other individuals, which are governed by the following set of rules. When a spreader interacts with an ignorant, the ignorant becomes a spreader; whenever a spreader contacts a stifler, the spreader turns into a stifler; and when a spreader meets another spreader, the initiating one becomes a stifler. In the last two cases, we say that the spreader was involved in a stifling experience. Observe that the process eventually finishes (when there are no spreaders in the population).

In this work, we propose a generalization of the Maki-Thompson rumour model by allowing random stifling. That is, we assume that each spreader ceases to propagate the rumour right after being involved in a random number of stifling experiences. We prove a Law of Large Numbers and a Central Limit Theorem for the proportion of the population never hearing the rumour.

References

[1] Daley, D.J.; Kendall, D.G. Stochastic rumours. *J. Inst. Math. Appl.* **1**, p. 42-55, 1965.
[2] Gani, J. The Maki-Thompson rumour model: a detailed analysis. *Environ. Modelling Software* **15**, p. 721-725, 2000.
[3] Lebensztayn, E.; Machado, F. P.; Rodriguez. P. M. Limit theorems for a general stochastic rumour model. *arXiv:1003.4995*, 2010.
[4] Maki, D.P.; Thompson, M. Mathematical Models and Applications. Prentice-Hall, Englewood Cliffs, 1973.

Gene Expression Time Delays and Turing Pattern Formation

S. Seirin Lee[a] and **E.A. Gaffney**[b]

[a]Graduate School of Mathematical Sciences
The University of Tokyo, JAPAN
seirin.lee@gmail.com

[b]Centre for Mathematical Biology
Mathematical Institute, University of Oxford, UK
gaffney@maths.ox.ac.uk

There are numerous examples of morphogen gradients controlling long range signalling in developmental and cellular systems. The prospect of two such interacting morphogens instigating long range self-organisation in biological systems via a Turing bifurcation has been explored, postulated or implicated in the context of numerous developmental processes. However, modelling investigations of cellular systems typically neglect the influence of gene expression on such dynamics, even though transcription and translation are observed to be important in morphogenetic systems. Critically, the previous study [1] observed that even these relatively small gene expression delays substantially amplify patterning lags, thus demonstrating the potential for gene expression dynamics to exert enormous temporal influence on pattern formation.

The investigations of our study demonstrate that the behaviour of Turing models profoundly changes on the inclusion of gene expression dynamics and is sensitive to the sub-cellular details of gene expression [2]. Furthermore, they also highlight that domain growth can no longer ameliorate the excessive sensitivity of Turing's mechanism in the presence of gene expression time delays [3, 4]. These results also indicate that the behaviour of Turing pattern formation systems on the inclusion of gene expression time delays may provide a means of distinguishing between possible forms of interaction kinetics, and also emphasises that sub-cellular and gene expression dynamics should not be simply neglected in models of long range biological pattern formation via morphogens. Finally, our study calls into question the plausibility of the Turing mechanism for pattern formation in biology where robustness is a key requirement. Turing's mechanism would generally require a novel and extensive secondary mechanism to control reaction diffusion patterning.

Keywords Pattern Formation, Cellular Dynamics, Time Delays, Reaction-Diffusion System

References

[1] E.A. Gaffney, N.A.M. Monk, *Gene expression time delays and Turing pattern formation systems*, Bull.Math.Bio. 68 (2006) 99–130.

[2] S. Seirin Lee, E.A. Gaffney, N.A.M. Monk, *The Influence of Gene Expression Time Delays on Gierer-Meinhardt Pattern Formation Systems*, to appear in Bull.Math.Bio. (2010).

[3] S. Seirin Lee, E.A. Gaffney, *Aberrant Behaviours of Reaction Diffusion Self-organisation Models on Growing Domains in The Presence of Gene Expression Time Delays*, to appear in Bull.Math.Bio. (2010).

[4] S. Seirin Lee, E.A. Gaffney, R.E. Baker, *An Investigation of Pattern Formation on Morphogen-controlled Growing Domains with Gene Expression Time Delays and Domain Response Delays*, preprint.

MANIPULATION OF HOST BEHAVIOUR AND VERTICAL TRANSMISSION HAVE ANTAGONISTIC EFFECTS ON THE SPREAD OF *Toxoplasma gondii*

M. Lelu[a,b,c], **E. Perrin**[a], **M. Langlais**[d], **M.-L. Poulle**[b,c] and **E. Gilot-Fromont**[a]

[a]Laboratoire de Biométrie et Biologie Evolutive, UMR CNRS 5588
Université Claude Bernard Lyon1

[b]Centre de Recherche et Formation en Eco-éthologie, Boult aux Bois

[c] Laboratoire de Parasitologie-Mycologie, EA 3800
Université Reims Champagne Ardenne CHU Reims

[d]Institut de Mathématique de Bordeaux, UMR CNRS 5251 et INRIA
Université Victor Segalen Bordeaux 2

In parasites with complex life cycle based on predator-prey relationship, parasite manipulation of host behaviour is thought to facilitate transmission from intermediate host (IH) to definitive host (DH) through predation. However, the importance of this phenomenon in the dynamics of the life cycle, and its interaction with other parameters such as the rate of vertical transmission and virulence is not clear. We analyse this question in *Toxoplasma gondii*, a complex life cycle parasite that is reported to alter its IH behaviour. *T. gondii* may also be transmitted vertically in IH, and infection may alter IH survival. We thus analyse the effects of manipulation combined with vertical transmission under different virulence rates on the transmission of *T. gondii*.

Starting from a deterministic SIR-type model describing the transmission dynamics of *T. gondii*, we add three functions representing vertical transmission, manipulation and virulence. We then analyse the sensitivity of the basic reproductive number R_0 to the corresponding parameters. R_0 and model predictions, i.e. the proportion of environmental contamination and the DH and IH seroprevalences, were used to evaluate the effects of the transmission strategies.

When considered alone, both vertical transmission and manipulation increase parasite spread. However, the impact of vertical transmission is highest when virulence is low, whereas the opposite is observed for manipulation. When both strategies are combined, it is possible to define a threshold of vertical transmission, as a function of virulence. Above this threshold, manipulation gives no advantage for parasite transmission. A possible explanation is that, when vertical transmission is frequent, manipulation may decrease the number of infected intermediate hosts that can reproduce. With our parameters values, this threshold varies from 58 % of vertical transmission when there is no virulence to more than 100 % when IH survival is divided by 6.

These results suggest that manipulation and vertical transmission are antagonistic strategies when vertical transmission is high and virulence is low. Manipulation and vertical transmission are two efficient strategies to compensate the effect of virulence of the parasite. Both strategies are antagonistic at high levels, but may be complementary, due to the high diversity of possible intermediate hosts for this species. The persistence of these two transmission strategies at low level in a parasite life cycle may help to explain the worldwide distribution of *T. gondii*.

A DEMO-GENETIC APPROACH TO THE EVOLUTION OF PEST RESISTANCE IN TOXIC CROPS

V. Lemesle[a] and **L. Mailleret**[b]

[a]UR102
CIRAD, TA B-102/02, F-34398 Montpellier
valerie.lemesle@cirad.fr

[b]URIH UR880
INRA Sophia Antipolis, BP 167, F-06903 Sophia Antipolis
ludovic.mailleret@sophia.inra.fr

Keywords: Agricultural pests, demo-genetic model, impulsive system.

Toxic plants, genetically modified or selected for antibiosis properties, produce toxins that are harmful to agricultural pests [1]. These plants are at the core of some modern pest management strategies. However, pests may evolve resistance to these toxins. In this context, pest resistance becomes a major concern for the sustainability of such pest management strategies. To prevent or delay resistance, non toxic host plants refuges have shown to be useful [2]. In this study, we will consider such a problem on a mixed crop composed of toxic and non toxic plants, taking into account that most agronomic crops are harvested on a regular basis. We moreover consider that some inter-crop pests immigration may occur.
A conceptual model based on a demo-genetic approach [3] and impulsive differential equations [4] is proposed to describe the dynamics of the different genotype densities of a diploid pest population. With T denoting the time length of a cropping season, k the season number, N_{ss}, N_{rs}, N_{rr} the genotype densities of susceptible homozygous, heterozygous and resistant homozygous pests, the model reads:

$$\begin{array}{ll} \dot{N}_{ss} = W_{ss} b g_{ss}(.) - \alpha N_{ss} N - \mu N_{ss} & N_{ss}(kT) = (1-p) N_{ss}(kT^-) + i_{N_{ss}} \\ \dot{N}_{rs} = W_{rs} b g_{rs}(.) - \alpha N_{rs} N - \mu N_{rs} \quad \text{for } t \in [(k-1)T, kT) & N_{rs}(kT) = (1-p) N_{rs}(kT^-) \\ \dot{N}_{rr} = W_{rr} b g_{rr}(.) - \alpha N_{rr} N - \mu N_{rr} & N_{rr}(kT) = (1-p) N_{rr}(kT^-) \end{array}$$

where g_{ij} are derived from Mendel laws [3], α is the competition coefficient, b and μ are the intrinsic birth and death rates, N the total population density. Genotypic fitnesses W_{ij} depend on the proportion of toxic/non toxic plants in the crop (denoted ρ) and is defined such that $W_{ij} = (1-\rho) W_{ij}$(on non toxic plants)
$+\rho W_{ij}$(on toxic plants). As most pest evolved resistance to toxic plants is recessive [2], we assume that only resistant homozygous pests are unaffected by toxic plants. This directly translates into the fitnesses definitions. Seasonal harvests are assumed to remove a proportion p of each sub populations. Moreover we suppose that inter-crop immigration is composed of susceptible homozygous individuals denoted $i_{N_{ss}}$. The mathematical analysis enlightens the influence of cropping season length and identifies threshold values for the proportion of the refuge in the dynamics of resistant pests. We moreover show that immigration favours a bistable behaviour in the model: either resistant pests persist in the system or go extinct. We discuss our results with respect to the use of toxic plants as a sustainable pest management strategy.

References

[1] FS. Betz et al.,"Safety and advantages of Bacillus thuringiensis-protected plants to control insect pests", *Regul Toxicol Pharm*, v. 32, p. 156-173, 2000.
[2] BE. Tabashnik et al., "Resistance to Bt toxins", *Science*, v. 287, p. 42, 2000.
[3] Y. Tyutyunov et al., "Landscape refuges delay resistance of the European corn borer to Bt-maize: A demo-genetic dynamic model", *Theor Popul Biol*, v. 74, p. 138-146, 2008.
[4] L. Mailleret, V. Lemesle, "A note on semi-discrete models in life sciences", *Phil T R Soc A*, v. 367, p. 4779-4799, 2009.

A TWO-STRAIN EPIDEMIC MODEL WITH DIFFERENTIAL SUSCEPTIBILITY AND MUTATION

Xue-Zhi Li[a] and **Sha-Sha Gao**[b]

[a]Department of Mathematics
Xinyang Normal University, Xinyang 464000, P.R. China
xzli66@126.com

[b]Department of Mathematics
Xinyang Normal University, Xinyang 464000, P.R. China
daisyhan@163.com

Keywords: Two-strain epidemic model, differential susceptibility, age-structured, imperfect vaccination, mutation, basic reproduction number; invasion reproduction number, exclusive equilibrium, coexistence equilibrium, stability, persistence.

A two-strain epidemic model with differential susceptibility and mutation is formulated and analyzed in this article. The susceptible population is divided into two subgroups according to the vaccine that provides complete protection against one of the strains (strain two) but only partial against the other (strain one). The explicit formulae for the basic reproduction number and invasion reproduction number corresponding to each strain with and without mutation are derived, respectively. It is shown that there exists exclusive equilibria and coexistence equilibria, even if the reproduction number is below one. The stability of the disease-free equilibrium, strain dominance with or without mutation are investigated. The persistence of the disease is also briefly discussed. Numerical simulations are presented to illustrate the results.

An Ecoepidemiological Model In Two Competing Species

Mats. Gyllenberg, **Xiaoli. Liu**, and **Ping. Yan**

Department of Mathematics and Statistics
University of Helsinki
P.O. Box 68 FI-00014 University of Helsinki
firstname.lastname@helsinki.fi

Abstract. The interaction of disease and competition dynamics is investigated in a system of two competing species in which only one species is susceptible to disease. It is assumed that only susceptibles reproduce and the infected individuals have the same competition strength as the susceptibles but suffer disease induced death rate. It is proved that if in the absence of the disease, there is competitive exclusion between two species, then the introduction of the disease can lead to stable or oscillatory coexistence of both species, which are partially determined by the basic reproduction number $\hat{R}_0$. If in the absence of the disease, the two species can coexist, then the introduction of the disease are determined by another basic reproduction number R_0. More If $R_0 > 1$, then the unique positive equilibrium is globally stable under certain conditions, while if $R_0 < 1$, then the disease will die out eventually.

Key words. Lotka-Volterra Competition, Hopf bifurcation, basic reproduction number, globally stable

Hopf bifurcation in structured population dynamics models

Zhihua Liu

School of Mathematics Sciences
Beijing Normal University
Beijing, 100875,P.R.China
zhihualiu@bnu.edu.cn

Keywords. Hopf bifurcation; structured population dynamics models; non-densely defined Cauchy problem.

Abstract. This talk will focus on our recent results on Hopf bifurcation in structured population dynamics models. Several types of differential equations, such as delay differential equations, age-structure models in population dynamics, some partial differential equations, evolution equations with nonlinear boundary conditions, can be written as semilinear Cauchy problems with non-dense domain. Firstly we will present our new results on Hopf bifurcations for non-densely defined Cauchy problems. Then we will show some applications in structured population dynamics models.

IDENTIFIED PROGENY OF UNKNOWN PARENTS: MATRIX CALIBRATION VIA AN EXTREMAL PRINCIPLE

D. O. Logofet

Laboratory of Mathematical Ecology, Institute of Atmospheric Physics,
Russian Academy of Sciences
Pyzhevskii Pereulok, 3, Moscow, 119017, Russian Federation
daniLaL@postman.ru

A kind of data for a stage-structured population "in which individuals are marked and followed over time" was called "identified individuals" [1, p. 134]. All the transition rates can hereafter be calculated directly from the data, but the birth rates often remains uncertain as far as the parent individuals cannot be identified from the data. For example, monitoring of marked individuals of a herbaceous species on a permanent sample plot does enable the identification of the individual stages, hence calculation of the transition rates, but it fails in doing so for the parent plants without destroying the plot [2]. Therefore, in the standard representation $\boldsymbol{L} = \boldsymbol{T} + \boldsymbol{F}$ of the *projection matrix* $\boldsymbol{L}$ of any matrix population model $\boldsymbol{x}(t+1) = \boldsymbol{L}\,\boldsymbol{x}(t)$, the *transition* matrix $\boldsymbol{T}$ calibrates definitely from data, while (the only nonzero row of) the *fertility* matrix $\boldsymbol{F}$ remains uncertain, though constrained with one (or more) *recruitment balance* equation(s):

$$x_1(t_0+1) = b_1x_1(t_0) + b_2x_2(t_0) + \ldots + b_nx_n(t_0), \qquad (1)$$

where t_0, t_0+1 are any consecutive moments of observation, and $b_j \geq 0$ denote the unknown birth rates. Frobenius eigenvalue $\lambda_1(\boldsymbol{L})$ of the projection matrix, the major model result characterizing the growth potential of a population with given vital rates, is known to be highly sensitive to variations in b_js. Meanwhile, technically simplest or arbitrary assumptions on the b_js that could eliminate the uncertainty would hardly correspond to real biology of the species.

We have broken the deadlock by using an *extremal principle* in the spirit of evolution theory: the values of b_js are supposed to maximize $\lambda_1(b_1, b_2, \ldots, b_n)$ under equation (1) and any other – known or hypothesized – constraints on the stage-specific birth rates. The task of calibrations reduces therefore to the corresponding constraint nonlinear maximization problem, where the eternal issue of local-vs.-global solutions is resolved via the properties of $\lambda_1(\boldsymbol{T}+\boldsymbol{F})$ as an increasing function of $\boldsymbol{F}$ under fixed $\boldsymbol{T}$.

A conjugate linear problem deals with what we call the *potential-growth indicator* (PGI), a calculable scalar function $R(\boldsymbol{L})$ of the vital rates with the following *indicator property*: $R(\boldsymbol{L}) >,=,< 1$ if and only if $\lambda_1(\boldsymbol{L}) > 1,=,< 1$. Proven recently [3], the PGI Theorem states an explicit form of $R(\boldsymbol{L})$ for a wide class of matrix $\boldsymbol{L}$ patterns that covers any *progressive-stage* life cycle graph (*Logofet* matrices). Once matrix $\boldsymbol{T}$ has been calibrated, the PGI becomes a (quantitatively certain) linear function $R(b_1, b_2, \ldots, b_n)$ of the uncertain birth rates. Its maximization over a polygonal defined by (1) plus the biological upper bounds on b_js plus (any number of) further hypothetical expert inequalities like $b_j \geq b_k$ represents a standard *LP-problem*, with an ever-ready solution. The solution verifies, by the PGI Theorem, whether the expert knowledge be compatible with the data and with $\lambda_1(\boldsymbol{L}) > 1$, i.e., with an idea of growing population.

References

[1] H. Caswell, "Matrix Population Models: Construction, Analysis, and Interpretation", 2nd ed., Sinauer, Sunderland, MA, 2001, 722 pp.

[2] D. Logofet, N. Ulanova, I. Klochkova, and A. Demidova, "Structure and dynamics of a clonal plant population: classical model results in a non-classic formulation", *Ecological Modelling*, v. 192 p. 95–106, 2006.

[3] D. Logofet and I. Belova, "Nonnegative matrices as a tool to model population dynamics: classical models and contemporary expansions", *Journal of Mathematical Sciences,* v. 155, No. 6, p. 894–907, 2008.

CONTROLLING A TROPHIC CHAIN TO EQUILIBRIUM

I. Lopez, **M. Gamez**, **S. Molnar** and **Z. Varga**

If an equilibrium state of the population is changed by an abiotic disturbance, a know problem of conservation ecology is to control the system back to the equilibrium. Control theory offers a solution to this problem. We will illustrate this approach on a three-level trophic chain of type resource Ð producer Ð primary consumer. A necessary and sufficient condition for the equilibrium coexistence in such systems can be found in Shamandy (2005), where sufficient conditions for observability in such systems have been obtained. In Varga et al. (2010) observer systems have been constructed for the monitoring of the considered trophic chain. In the present paper, the control of different model parameters (resource supply, recycle rate and Malthus parameters) is considered. First, applying a sufficient condition for local controllability of Lee and Markus (1971) (see also Varga, 2008) we obtain that if a perturbation changes the equilibrium state of the system to a nearby state, then applying a small intervention on one of the mentioned system parameters, the whole trophic chain can be steered back to the equilibrium in given time. Furthermore, the numerical calculation of such control functions is also carried out, solving a corresponding optimal control problem by the use of MatLab toolbox, developed in Banga et al. (2005) and Hirmajer et al. (2009).

An Integrodifference model for the study of long distance dispersal coupled to Allee effect in a biological invasion

S. Lou Vega[a] and **W. Castro Ferreira Jr.**[b]

Department of Applied Mathematics
Instituto de Matemática Estatística e Computação Cinetífica - IMECC- , UNICAMP
Universidade Estadual de Campinas, SP. Brazil
Caixa Postal 6065; CEP 13083-859, Campinas, SP. Brazil.
[a]slouvega@gmail.com
[b]wilson@ime.unicamp.br

A *demographic Allee effect* establishes a positive relationship between the population density and the per capita growth rate. This means that at lower density levels, the per capita growth rate is reduced, sometimes reaching negative growth rates. The Allee effect is important when considering the conservation of endangered or rare species, but recently it has taken relevance in biological invasions and their control. The Allee effect can alter the dynamics of Invasion. It can reduce expansion speeds of invasive organisms, for example.

In Plant invasions, factors affecting seed production can alter the dynamics of the invasion. Pollen limitation is a factor producing Allee effect in plants. Allee effect due to pollen limitation have been detected for some invasive plants such as *Spartina alterniflora*.

It is of great importance that models combine the population growth dynamics with dispersal behavior to have a better understanding of the process of invasion and to contribute to the management of invasive species. Here we develop a model that couples plant growth subject to an Allee effect due to pollen limitation, and dispersal process.

Dispersal is considered to be a multiple scale process. In plant dispersal, two principal scales are commonly defined, local dispersion (LD) and long distance dispersion (LDD). The LDD is considered to be a stochastic or eventual process, although the expansion speed of an invasive species is very sensitive on it.

Using IDE and a multiple scale dispersal (LD and LDD), we analyzed the influence of the Allee effect on the dynamics of the invasion. We also give an analytical approximation for a stationary wave through a piecewise constatn approximation of the travelling wave solution of the model.

KEYWORDS: Plant invasion, Allee effect, integro-difference equations, dispersal kernel.

References

[1] H. Davis, C.M. Taylor, J.G. Lambrinos and D.R Strong, " Pollen limitation causes an Allee effect in a wind pollinated invasive grass (*Spartina alterniflora*)", *Proc. Natl. Acad. Sci.*, **101**, 13804 (2004).
[2] M. Kot, M. Lewis, P. van den Driessch, " Dispersal data and the spread of invading organisms", Ecology, **77**, 2027 (1996).
[4] C.M. Taylor and A. Hastings, " Allee effects in biological invasions ", Ecol. Lett., **8**, 895 (2005).
[6] M.H. Wang, M. Kot, " Integrodifference equations, Allee effects and invasions, J. Math. Biol., **44**, 150 (2002).

EPIDEMIOLOGY OF HIV IN CUBA. MODELING DETECTION WITH VARYING PARAMETERS

R. Lounes[a], **H. de Arazoza**[b], **A. Sanchez**[b] and **Ying-Hen Hsieh**[c]

[a]Laboratoire MAP5, UMR-CNRS 8145
University Paris Descartes
45 rue des Saints-Pères, 75270 Paris CEDEX 06, France
lounes@math-info.univ-paris5.fr

[b]Department of Mathematics
University of Havana
San Lazaro y L, Ciudad de La Habana, Cuba
arazoza@matcom.uh.cu ; asp@matcom.uh.cu

[c]Department of Public Health and Center for Infectious Disease Epidemiology Research China
Medical University Taichung
91 Hsueh-Shih Road,Taichung, Taiwan 40402, R.O.C
hsieh@mail.cmu.edu.tw

Keywords: HIV/AIDS epidemic, contact tracing, epidemic control, deterministic model.

ABSTRACT:
From the beginning of the HIV/AIDS epidemic in Cuba a system for detection of persons living with HIV was put in place, [1].A nonlinear model is developed for the detection system considering different types of searches, some random and others non-random, the model is based on another one studied before by some of the present authors [2], [3]. We study the dynamics of the system. Using the data from the Cuban HIV/AIDS epidemic, we fit the model to the data. For this, as not all types of searches were introduced at the same time, we consider the parameters of the system to be step functions. We obtain estimates for the size of the Cuban HIV epidemic, and for the mean time for detecting a person that is infected with HIV. Also the basic reproduction number is computed for each set of values of the parameters.

References

[1] H. de Arazoza and J. Joanes and R. Lounes and C. Legeai and S. Clémençon and J. Pérez and B. Auvert, "The HIV/AIDS epidemic in Cuba: description and tentative explanation of its low HIV prevalence", *BMC Infectious Diseases*, 7:130 (9 November 2007).

[2] H. de Arazoza R. Lounes, "A non-linear model for a sexually transmitted disease with contact tracing", *IMA J Math Appl Med Biol*, v. 19, p. 221-234, 2002.

[3] Y-H. Hsieh and H. de Arazoza and R. Lounes and J. Joanes, "A Class of Methods for HIV Contact Tracing in Cuba: Implications for Intervention and Treatment", *Deterministic and Stochastic Models for AIDS Epidemics and HIV Infection with Interventions*, W.Y. Tan, editor, Chap. 4, p. 77-92, Singapore: World Scientific., 2005.

SPATIAL DISPERSAL AND PROLONGED DIAPAUSE IN A PATCHY STOCHASTIC ENVIRONMENT

T. Mahdjoub, **S. Gourbière** and **F. Menu**

Spatial dispersal and prolonged diapause are two major traits in evolution and populations dynamics. Often considered as being two alternative strategies, the simultaneous impacts of these two traits on the population growth rate are little studied. The growth rate of a metapopulation, made up of two populations connected by a certain dispersal rate, living on two different qualities patches, is determined by application of the "vec- permutation" method [1]. The growth rate variations are studied in two stages populations, under a constant environment then a stochastic one. Results show that, in the case of a constant environment, the growth rate is optimal when the metapopulation is with simple diapause and the dispersal rate is null. However, under a stochastic environment, the mean stochastic growth rate is optimal for mixed prolonged diapause strategies (bet-hedging) and a non null dispersal rate.

MODELLING VECTOR-BORNE DISEASE: WEST NILE VIRUS

N. A. Maidana[a] and **Y. M. Yang**[b]

[a]Centro de Matemática, Computação e Cognição
UFABC
Rua Catequese 242, CEP:09090-400, Santo Andre, Sp, Brasil.
norberto.maidana@ufabc.edu.br

[b]Departamento de Matemática Aplicada
UNICAMP
Caixa Postal 6065, CEP: 13083-859, Campinas, SP, Brasil.
hyunyang@ime.unicamp.br

West Nile Virus is an arthropod-borne flavivirus that appeared at first time in New York City in the summer of 1999 and then spread prolifically within birds, where over 200 species have been infected. Mammals, as human and horse, do not develop sufficiently high bloodstream titers to play a significant role in transmission, which is the reason to consider the mosquito-bird cycle. We propose a model to study the bird competence to transmit West Nile Virus as a system of ordinary differential equations considering the mosquito and several avian populations. A threshold value R_0 depending on the model's parameters is obtained, which determines the disease level and allows us to propose possible control strategies. We determine the effects regarding to the disease transmission considering the coexisting of two bird species, which are generalized taking into account several bird species. We consider the correspondent reaction diffusion model to study spatial dissemination by traveling wave solutions.

Key-words: West Nile Virus – epidemiological model – control – avian populations – traveling wave – wave speed

References

[1] G. Cruz-Pacheco, L. Esteva, J.A. Montaño-Hirose and C. Vargas , Modelling the dynamics of the West Nile Virus, *Bulletin of Mathematical Biology*, 67, 1157-1172, 2005.
[2] Lewis, M., Renclawowicz, J. and Van den Driessche, P. Travelling Waves and Spread rate for a West Nile Virus Model, *Bulletin of Mathematical Biology*, 68, pp. 3-23, 2006.
[3] DeBiasi, R. L. and Tyler, K. West Nile Virus Meningoencephalitis. *Nature Clinical Pratice Neurology*, Vol. 3 NO 5, pp. 264-275, 2006.
[4] N. A. Maidana and H. M. Yang. Spatial spreading of west nile virus described by traveling waves. *Journal of theoretical biology*, 258(3), 403-417, 2009.

COMPETITION AND DIFFUSIVE INVASION IN A VARIABLE ENVIRONMENT

H. Malchow and **M. Sieber**

Department of Mathematics & Computer Science
Institute of Environmental Systems Research
University of Osnabrück, 49069 Osnabrück, Germany
malchow@uos.de msieber@uos.de

Biological invasions including the spread of infectious diseases have strong ecological and economical impacts. The perception of their often harmful effects has been continuously growing both in sciences and in the public. Mathematical modelling is a suitable method to investigate the dynamics of invasions, both supplementary to and initiating field studies as well as control measures.

A Lotka-Volterra competition-diffusion model is considered for conditions of invasibility of a certain model area occupied by a native species. The variability of the environment due to contingent landslides leads to the temporary extinction of both species at a randomly chosen time and spatial range. The spatiotemporal dimension of these extreme fragmentation events as well as a possible selected harvesting of the invader turn out to be the crucial driving forces of the system dynamics.

MODELLING HOSPITALIZATION, HOME-BASED CARE AND INDIVIDUAL WITHDRAWAL FOR PEOPLE LIVING WITH HIV/AIDS IN HIGH PREVALENCE AREASs

H. Mambili-Mamboundou, **D. Senelani Hove-Musekwa** and **F. Nyabadza**

In sub-Saharan Africa, the model of care of people who living with HIV/AIDS has changed from hospital care to home-based care. In this paper, a mathematical model describing the dynamics of HIV transmission, hospitalization and home-based care is constructed and analyzed. The model reproduction number Re is determined and discussed. The equilibria are determined and analyzed in terms of Re . It is shown that if $\mathrm{Re} < 1$, the disease free equilibrium is both locally and globally asymptotically stable. The model has a unique endemic equilibrium which exists and is locally asymptotically stable whenever $\mathrm{Re} > 1$. The model is fitted to the prevalence data from UNAIDS on Zimbabwe. The implications of some key epidemiological parameters are investigated numerically. Projections are made to determine the possible long term trends of the prevalence of HIV in Zimbabwe. These projections are compared to the HIV/AIDS epidemic trends in Uganda.

SPATIAL PATTERNS OF SOLID TUMOURS IN AVASCULAR STAGE; MODELING AND MATHEMATICAL ANALYSIS

H. Mambili-Mamboundou[a] and **Aziz Ouhinou**[a b]

[a] African Institute for Mathematical Sciences (AIMS),
6 Melrose Road, Muizenberg 7945, Cape Town
South Africa
hermane@aims.ac.za

[b] Department of Mathematical Sciences
University of Stellenbosch
Private Bag X1 7602, Matieland South Africa
aziz@aims.ac.za

In the avascular stage, a solid tumour grows only to a diffusion limited size. For a considerable tumour size, the cells in the middle starve and die creating what is called a necrotic core. Just above the necrotic core, cells have just to survive but will not proliferate, creating a quiescent layer, and the outer layer is a thin rim of proliferating cells.

In the effort of understanding the heterogeneity of a solid tumour, we will look at the production of growth inhibitor factor (GIF) within the spheroid. This aspect has been investigated by many authors [1], [2] and [3]. GIF is believed to reduce the proliferation of tumour cells, and experimental results have shown that there is a threshold value for the concentration of GIF under which mitosis occurs, and if the concentration of GIF is above that value, mitosis is inhibited. In this work, we extend Chaplain's work [1] on avascular tumour growth with spatially dependent diffusion coefficient, by coupling the resulting GIF model with an equation describing the tumour cell density and adding a consumption term of GIF governed by the receptor kinetic law. In addition we include the variation of the spacial domain.

The mathematical model is able to reproduce the experimentally observed spatial pattern of avascular tumour growth. The steady solutions are approximated numerically and the semigroup method is used to look at the stability analysis of stationary solutions.

References

[1] J. A. Adam, A simplified mathematical model of tumor growth, Math. Biosci. 81, 229-244 (1984).
[2] J. A. Adam, A simplified mathematical model of tumor growth : Effect of geometry and spatial nonuniformity on stability, Math. Biosci. 86, 183-211 (1987).
[3] M. A. J. Chaplain, Avascular growth, angiogenesis and vascular growth in solid tumours: the mathematical modelling of the stages of tumour development, Math. Comput. Modelling , 23, No. 6, 47-87 (1996)

THE IMPACT OF VACCINE SIDE EFFECTS ON THE LIFETIME OF IMMUNIZATION PROGRAMMES

P. Manfredi, **A. d'Onofrio** and **P. Poletti**

In regimes of voluntary vaccination at high levels of herd immunity the incidence of vaccine associated side effects might become a central determinant of the vaccine demand, and therefore of the overall infection dynamics [1]. We study a SIR transmission model with dynamic vaccine demand according to an imitation mechanism where the perceived risk of vaccination is modeled as a function of the incidence of vaccine side effects. The model shows some noteworthy theoretical differences compared to the baseline game-dynamic model of vaccination [2]. Moreover its equilibrium results allow to sharply identify the potentially harmful role of economic development and vaccination history on immunization programmes, yielding noteworthy inferences as regards the future lifetime of mass vaccination.

MODELLING VIRUS VARIANTS AND INVASION

J.L. Mann[a] and **M.G. Roberts**[a,b]

[a]Institute of Information and Mathematical Sciences
Massey University
Private Bag 102 904
North Shore Mail Centre 1311
Auckland
New Zealand
j.l.mann@massey.ac.nz

[b]New Zealand Institute for Advanced Study
Massey University
Private Bag 102 904
North Shore Mail Centre 1311
Auckland
New Zealand
m.g.roberts@massey.ac.nz

If a virus is released and then subsequently mutates in an initially naive population, will the mutated virus variant survive in the population and does the distance (in some sense) between the original and the new variants have an affect on the invasibility of the new variant? These are two questions that we will explore using a model based on a virus characterised by a string of epitopes.

We consider a virus expressed as a string of n positions, each of which contains a distinct epitope. An epitope at each position may undergo mutation (thus only one position may vary at each mutation event). At each position in the string there are m possible epitopes available, all of which will be assigned a unique number. That is, in position one of the variant string, epitopes numbered $1 \dots m$ may exist, at position two of the string, epitopes $m+1 \dots 2m$ may exist, and so on. We assume that when a host is infected with a virus variant, it mounts an immune response to one epitope only and will then be immune to all other variants containing that epitope. With an n-position string having m epitopes available at each position, there are a total of m^n possible variants and, at most, $2^{nm}-2$ possible immune states (it is not possible to gain immunity against all epitopes and the naive class is considered separately). In the model, each immune state and each virus variant gives rise to a differential equation. Hence we require $2^{nm}+m^n-2+1$ equations.

Using the method given in [1] to find the next generation matrix with large domain (see [2]), we are able to determine if a variant will be able to produce a large epidemic following an epidemic of another variant.We demonstrate this using a model with a 2-position string with 2 epitopes at each position, hence a system of 19 differential equations. We show that the amount of distance between variants plays a crucial role in the invasibility.

References

[1] A. Hurford, D. Cownden and T. Day, "Next-generation tools for evolutionary invasion analyses", *Journal of The Royal Society Interface*, v. 25, p. 561-571, 2010.
[2] O. Diekmann, J.A.P. Heesterbeek and M.G. Roberts, "The construction of next-generation matrices for compartmental epidemic models" *Journal of The Royal Society Interface* , published online, 2009.

AVIAN INFLUENZA

M. Martcheva and **M. Roy**

The drift and shift mechanisms of genomic evolution jointly allow the influenza A virus to circulate among many animal species and humans worldwide. We develop an influenza A model that involves both the drift evolution of human influenza A, and the shift evolution that allows a novel H5N1 strain to become established in the human population. We parameterize the model so that it faithfully describes the number of human cases of H5N1 in the world. We derive conditions for invasion of the novel pandemic strain. Those suggest that the invasion of the pandemic strain is facilitated by measures that decrease seasonal human influenza, or the presence of highly pathogenic H5N1 in birds. Recently, Magal and Ruan establish that Pease's influenza A drift model, which is a part of our model, can exhibit sustain oscillations. We show that the presence of the avian influenza may stabilize these oscillations.

APPROXIMATE AGGREGATION OF NON-AUTONOMOUS TWO TIME SCALES SPATIALLY DITRIBUTED SYSTEM

M. Marvá[a] and **R. Bravo de la Parra**[a] and **J.C. Poggiale**[b]

[a]Department of Mathematics
Universidad de Alcalá
28871 Alcalá de Henares. Madrid, Spain
marcos.marva@uah.es (corresponding author), rafael.bravo@uah.es

[b]Centre d'Oceanologie de Marseille, UMR CNRS 6117
Laboratoire de Microbiologie, de Geochimie et d'Ecologie Marines
Université de la Méditerranée, Campus de Luminy Case 901
13288 Marseille Cedex 9 - France
jean-christophe.poggiale@univmed.fr

Keywords: approximate aggregation; spatially explicit models; non-autonomous systems; two time scale.

The aim of this work is to present a general class of nonlinear, non-autonomous, ordinary differential equation models with two time scales which dynamics is susceptible of being approached by means of a reduced (less dimensional) system. We couple two different processes assuming that one of them is much faster that the other one. We consider asymptotically autonomous or autonomous equations for the fast part coupled with periodic or asymptotically autonomous equations for the slow terms.

Assuming that fast dynamics instantaneously attains certain equilibrium, we build up and reduced system. It is proved that certain asymptotic behaviors, as the existence of asymptotically stable periodic solutions, to the aggregated system entail that to the original system. The reduction process is included in the so-called *approximate aggregation of variables methods* [1] which consist of describing the dynamics of a complex system involving many coupled variables through the dynamics of a reduced system, so-called *aggregated system*, formulated in terms of a few *global variables*.

A typical application of aggregation techniques to population dynamics models is that of reducing spatially explicit models where migration can be considered a fast process compared to demography processes or community interactions (e.g. prey-predator models). Recent works consider the spread of epidemics within a population as a fast process in front of community dynamics [2] and in front of demographic process [3]. Less usual applications are those coupling the spread of epidemics within a population inhabiting two different patches. We let migrations to be faster than epidemics and conversely.

References

[1] P. Auger, R. Bravo de la Parra, J.-C. Poggiale, E. Sánchez and T. Nguyen-Huu, "Structured Population Models in Biology and Epidemiology", *Lecture Notes in Mathematics*, Springer Berlin/Heidelberg, p. 209–263, 2008.
[2] P. Auger, R. Mchich, T. Chowdhury, G. Sallet, M. Tchuente J. Chattopadhyay. Effects of a disease affecting a predator on the dynamics of a predator-prey system. *Journal of Theoretical Biology* 258, 344–351, 2009.
[3] S. Gaucel, B. Laroche, P. Ezanno, E. Vergu, S. Touzeau. Using singular perturbations to reduce an epidemiological model: Application to bovine viral diarrhoea virus within-herd spread. *Journal of Theoretical Biology* 258, 426–436, 2009.

INDIVIDUAL BEHAVIOR IN A STRUCTURED POPULATION: REDUCTION OF A DISCRETE SYSTEM COUPLING HAWK-DOVE TACTICS AND DEMOGRAPHY

M. Marvá[a] and **R. Bravo de la Parra**[a] and **P. Auger**[b] and **M.A. Zavala**[c]

[a]Department of Mathematics
Universidad de Alcalá
28871 Alcalá de Henares. Madrid, Spain
marcos.marva@uah.es (corresponding author), rafael.bravo@uah.es

[b]IRD UR GEODES Centre IRD de l. Ile de France,
32 Avenue Henri Varagnat, 93143 Bondy Cedex, France
pierre.auger@bondy.ird.fr

[c]Department of Ecology
Universidad de Alcalá
28871 Alcalá de Henares. Madrid, Spain
ma.zavala@uah.es

Keywords: Approximate aggregation; Two time scale models; Game dynamics; Leslie model.

We consider an age-structured population affected by two processes. At the individual level, we consider a classical game model, where adults may behave as aggressive (hawk) or passive (dove) individuals, depending on the average cost or gain of fighting for resources. At the population level, a non-linear Leslie matrix stands for the demographic process. Typically, individual decisions are much faster than demography, which yields a two time scales model. Moreover, we consider a denso-dependent Leslie matrix and we make explicit the game equations following [1], which entails an extension of [2].

This model belongs to a general class of nonlinear discrete models with two time scales which dynamics is susceptible of being approached by means of a reduced system. The reduction process is based on the difference between the time scales and is included in the so-called *approximate aggregation of variables* methods [3]. These techniques consist of describing the dynamics of a complex system involving many coupled variables through the dynamics of a reduced one, the *aggregated system*, formulated in terms of a few *global variables*. Sanz *et al.* [4] have derived general conditions for the approximate aggregation of two time scale discrete models. These results assure that some asymptotic behaviors and hyperbolic asymptotically stable periodic solutions to the aggregated system entail that to the original system.

Regarding our model, from the reduction process we find two different individual strategies merging in the long term. In a poor environment individuals better avoid contests. In a rich environment it seems better to fight for resources. As a result of this analysis we find out some results complementary to those in Sanz *et al.*

References

[1]E. Ahmed, A.S. Hegazi, On some variants of dynamical systems, *Chaos, solitons and fractals*, Vol. 12, p. 2103-2108, 2001.
[2]E. Chambon-Dubreuil, P. Auger, J.M. Gaillard, M. Khaladi, Effect of aggressive behaviour on age-structured population dynamics, *Ecological Modelling*, Vol. 193, No. 3-4, p. 777-786, 2006.
[3] P. Auger, R. Bravo de la Parra, J.-C. Poggiale, E. Sánchez and T. Nguyen-Huu, "Structured Population Models in Biology and Epidemiology", *Lecture Notes in Mathematics*, Springer Berlin/Heidelberg, p. 209-263, 2008.
[4] L. Sanz, R. Bravo de la Parra, E. Sánchez, Approximate reduction of non-linear discrete models with two time scales, *Journal of Difference Equations and Applications*, Vol. 14, No. 6, p. 607-627, 2008.

THE IMPACT OF TEMPERATURE ON THE EVOLUTION OF VIRULENCE

E. Massad[a,b], **L. F. Lopez**[a], **D. R. da Silva**[a] and **C. J. . Struchiner**[c]

[a]School of Medicine, University of Sao Paulo and LIM 01-HCFMUSP
Rua Teodoro Sampaio, 115
CEP: 05405-000 - São Paulo - S.P.- Brazil

[b]London School of Hygiene and Tropical Diseases, U.K.

[c]Program of Scientific Computing, Fundação Oswaldo Cruz, Brazil

Keywords: virulence, temperature, models, basic reproduction number.

Background – It is expected that the increase in global temperature will lead to an expansion of the geographical spread, and an increase in the incidence, of tropical infections. However, the trend in severity of those infections as a function of the increase in temperature is still unknown.

Method - Suppose that two strains of a given parasite are competing for the same host. It is possible to demonstrate that, in general, the strain with an evolutionarily stable strategy, that is, the one that wins the competition, is the one with the highest value of R_0. We want to know which combination of environmental temperature T and virulence ν maximizes R_0(T,υ) . For this we calculate the tangent plane to the maximum point, that is $\frac{\partial R_0}{\partial T} = 0$ and $\frac{\partial R_0}{\partial \nu} = 0$. Now, let us consider the case of a vector-borne infection. In this case we have that:

$$\nu = \frac{\frac{2}{T} + \frac{\kappa_2 e^{-\kappa_2 T} T + 2\kappa_6\kappa_7(1-e^{-\kappa_2 T}) - (1-e^{-\kappa_2 T})}{(1-e^{-\kappa_2 T})T} - 2\gamma_0}{\kappa_4 - \kappa_5}$$

where κ_i $(i = 2, ...7)$ are constants and γ_0 is the basal recovery rate.

Results - We demonstrate that, in this case, the increase in temperature is associated with an increase in parasite virulence.

System Dynamics from Individual Interactions: A Process Algebra Approach to Epidemiology

C. McCaig[a], **S. Benkirane**[a], **R. Norman**[a] and **C. Shankland**[a]

[a]Department of Computing Science and Mathematics
University of Stirling
Stirling, UK, FK9 4LA
cmc,sbk,ran,ces@cs.stir.ac.uk

The problem of changing scale is important in many fields. In epidemiology models can be developed that describe a population in terms of the stochastic behaviour of individuals or in terms of the mean change in the number of infected individuals over time. Although a modeller may design these two models with the same system in mind it is not clear how the behaviour of individuals affects the population dynamics.

In this paper we present process algebra [1] as a novel solution to the problem of changing scale in the context of models of infectious disease spread. The unique benefits of this approach are threefold. Firstly, it is possible to describe the behaviour of individuals directly. Secondly, those individuals can be combined to give the behaviour of the system as a whole. We have developed a rigorous method to make the transition between the individual- and population-level descriptions of the system formal and automatic [2, 3]. Thirdly, the system can be formally investigated to establish features of the system dynamics, allowing us to answer the sort of questions posed above. Individual-based modelling is particularly important because we can more realistically measure facts about individuals, while our questions about epidemics all come from the population level. The ability to move rigorously between different levels of abstraction (individual to population) when describing disease spread gives us completely new ways of thinking about epidemiology.

References

[1] J. Baeten, "A brief history of process algebra", *Theoretical Computer Science*, v. 335, p. 131-146, 2005.
[2] S. Benkirane, J. Hillston, C. McCaig, R. Norman, and C. Shankland, "Improved Continuous Approximation of PEPA Models through Epidemiological Examples", *Proceedings of the Second Workshop From Biology to Concurrency and Back, FBTC 2008*, v. 229, of *Electronic Notes in Theoretical Computer Science* p. 59-74, 2009.
[3] C. McCaig, R. Norman, and C. Shankland, "Process algebra models of population dynamics", *Algebraic Biology, AB2008*, v. 5147, of *Lecture Notes in Computer Science* p. 139-155, 2008.

GLOBAL STABILITY FOR EPIDEMIC MODELS INVOLVING FUNCTIONAL DIFFERENTIAL EQUATIONS

C. Connell McCluskey

Department of Mathematics
Wilfrid Laurier University
Waterloo, Ontario, Canada
ccmcc8@gmail.com

Many ordinary differential equation models of disease spread have been shown to exhibit the traditional threshold behaviour. That is, there is a basic reproduction number R_0 for the disease, and:

if $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable (GAS);
if $R_0 > 1$, then the endemic equilibrium is GAS.

Recently, there has been good progress on this through the use of a Lyapunov function that Goh first used for ecological models.

Separately, there have been many functional differential equation (FDE) models of disease spread that have used delay to account for vector (e.g. mosquito) transmission, or have used integrals to account for infection-age structure. Earlier results on the global dynamics of these models introduce restrictions on the parameters that seem to be an artifact of the method.

I will discuss recent work that uses Goh-type Lyapunov functionals to resolve the global dynamics for some of these FDE models, achieving the traditional threshold result. The following papers [1-4] are related.

References

[1] C. C. McCluskey, "Complete global stability for an SIR epidemic model with delay - distributed or discrete", *Nonlinear Anal. RWA*. 11:55–59, 2010.
[2] C. C. McCluskey, "Global stability for an SEIR epidemiological model with varying infectivity and infinite delay", *Math. Biosci. and Eng.* 6:603–610, 2009.
[3] P. Magal, C. C. McCluskey, and G. Webb, "Liapunov functional and global asymptotic stability for an infection-age model", *Applicable Analysis*. In Press.
[4] C. C. McCluskey, "Global stability for an SIR epidemic model with delay and nonlinear incidence", *Nonlinear Anal. RWA*. doi:10.1016/j.nonrwa.2009.11.005.

CYCLES FROM RESONANT AMPLIFICATION OF DEMOGRAPHIC STOCHASTICITY

Alan McKane

Department of Theoretical Physics
University of Manchester
Manchester, M13 9PL, UK
alan.mckane@manchester.ac.uk

Keywords: Stochastic dynamics, Population cycles

In this talk I will discuss the stochastic dynamics of a number of problems in population biology and biochemistry using the formalism of master equations. When they contain a large number of constituents, the behaviour of these systems may be analysed using an expansion in the system size. To leading order the deterministic analogues of the models can be compared to the equations which are normally written down on phenomenological grounds. At next to leading order a simplified stochastic description is obtained. Attention will focus on systems for which the deterministic description fails to predict cycles, but where large cycles are found at next-to-leading order. These cycles have their origin in fluctuations due to the discrete nature of the system components, and are much larger than would naively be expected because they are amplified by a resonance phenomenon. The generality and applicability of these results will be discussed.

APPLICATION OF AN AGGREGATION METHOD TO THE MANAGEMENT OF MOROCCAN FISHERIES

R. Mchich[a], **P. Auger**[b] and **N. Raïssi**[c]

[a]Department of Managing
Ecole Nationale de Commerce et de Gestion
B.P. 1255, 90000, Tangier, Morocco
racmchich@yahoo.com

[b]UMI 209 IRD & UPMC UMMISCO
Centre de Recherche d'Ile de France
32 avenue Henri Varagnat, 93143, Bondy cedex, France
pierre.auger@ird.fr

[c]Department of Mathematics and Informatics
Faculty of sciences, Ibn Tofaïl University
B.P.133, 14000, Kenitra, Morocco
n.raissi@lycos.com

Mathematical models in ecology are usually nonlinear dynamical systems including a large number of coupled variables (6). These models are then difficult to study analytically. Aggregation of variables, which is based on time separation methods, permits to build, from a complete model involving many variables, a reduced one which governs a few global variables in the long term (1,5,9,10). In this work, we present a review of methods of aggregation of variables and their applications to multi fishery models and Moroccan fisheries management. We particularly study the effects of stock density dependent displacement of the fishing vessels as well as resource price variation on the dynamics of the fishery (2,3,4,7,8). Interpretations of our results can be used by the authority responsible of fishery management, the Moroccan government, as general recommendations for a better management of Moroccan fisheries.

References

[1] P.M. Auger and R. Bravo de la Parra, "Methods of aggregation of variables in population dynamics", Comptes Rendus de l'Académie des Sciences, série III Paris, 323, 665-674, 2000.

[2] P.M. Auger, R. Mchich, N. Raïssi and B. Koii, "Effects of market price on the dynamics of a spatial fishery model : Over-exploited fishery/traditional fishery", Ecological Complexity, Vol. 7, Issue 1, pp 13-20, 2010.

[3] N. Charouki, I.M. Chibani, A. Ramzi and O. Ettahiri "Rapport annuel du projet Natfish, partie Maroc", 2004.

[4] N. Charouki, N. Raïssi, P.M. Auger, R. Mchich and H. Otmani, "A competitive model between two fishing fleets for the sardine fishery on the Moroccan Atlantic coast", Canadian Journal of Fisheries and Aquatic Sciences. Submitted, 2010.

[5] N. Fenichel, "Persistence and Smoothness of Invariant Manifolds for Flows", Indiana University Mathematical Journal 21: 193-226, 1971.

[6] R.M. May, "Stability and complexity in Model Ecosystems", 2nd Edition, Princeton University Press, 1974.

[7] R. Mchich, P.M. Auger, R. Bravo de la Parra and N. Raïssi, "Dynamics of a fishery on two fishing zones with fish stock dependent Migrations: Aggregation and Control", Ecological Modelling, vol. 158, Issue 1-2, pp. 51-62, 2002.

[8] R. Mchich, N. Charouki, P.M. Auger, N. Raïssi and O. Ettahiri, "Optimal spatial distribution of the fishing effort in a multi fishing zone model", Ecological Modelling, vol. 197, Issues 3-4, pp. 274-280, 2006.

[9] J. Michalski, J.C. Poggiale, R. Arditi and P.M. Auger, "Macroscopic Dynamic Effects of Migrations in Patchy Predator-prey Systems", J. Theor. Biol., 185, 459-474, 1997.

[10] J.C. Poggiale, "Applications des Variétés Invariantes à la Modélisation de l'Hétérogénéité en Dynamique des Populations", Thesis, University of Bourgogne, Dijon, 1994.

WKB THEORY OF STOCHASTIC EPIDEMICS IN WELL-MIXED POPULATIONS

B. Meerson

Racah Institute of Physics
Hebrew University of Jerusalem
Jerusalem 91904, Israel
meerson@cc.huji.ac.il

Stochastic effects in disease transmission can eliminate disease from small communities. In one scenario this happens after the disease already reached an endemic state. In another scenario the disease can fade out immediately after an epidemic outbreak. I will review a recent progress [1-3] in theoretical analysis of these classical problems. When the sub-population sizes are sufficiently large, one can use a dissipative version of WKB (Wentzel-Kramers-Brillouin) approximation thus reducing the original master equation to Hamiltonian mechanics. The most probable paths to disease extinction turn out to be describable by heteroclinic trajectories of the underlying Hamiltonian. Further analytical progress in multi-population systems is possible when either disparity of transition rates, or proximity to a bifurcation, causes time scale separation and brings about a small parameter. I will illustrate these points on the examples of the stochastic SI (Susceptible-Infected) and SIS (Susceptible-Infected-Susceptible) models with population turnover: renewal and removal.

References

[1] A. Kamenev and B. Meerson, "Extinction of an infectious disease: A large fluctuation in a nonequilibrium system" *Phys. Rev. E*, v. 77, p. 061107 (1-4), 2008.
[2] B. Meerson and P.V. Sasorov, "WKB theory of epidemic fade-out in stochastic populations", *Phys. Rev. E*, v. 80, p. 041130 (1-4), 2009.
[3] M. Khasin, B. Meerson, and P.V. Sasorov, "Time-resolved extinction rates of stochastic populations", *Phys. Rev. E*, v. 81, p. 031126 (1-8), 2010.

Polymorphic evolution process and evolutionary branching

Sylvie MELEARD, CMAP, Ecole Polytechnique.

We are interested in the study of a polymorphic population with mutation and selection. Our modeling is based on a stochastic individual-based model that details the dynamics of heritable traits characterizing each individual. We consider the specific scales of the biological framework of adaptive dynamics: rare mutations and large population. We prove that under a good combination of these two scales, the population process is approximated in an evolution long time scale by a Markov pure jump process describing successive equilibria of the population. This process generalizes the so-called trait substitution sequence. Then we consider this polymorphic evolution process in the limit of small mutations. From a fine study in the neighborhood of evolutionary singularities, we obtain a full mathematical justification of a heuristic criterion for the phenomenon of evolutionary branching.

A HIERARCHICAL MODEL FOR TUMOR DEVELOPMENT INCLUDING THE TIME-DELAY AND PARAMETERS DEPENDENT ON THE CELL POPULATION

M.P.C. Menezes[a] and **M.E. Gouvêa**[b]

[a]Centro Federal de Educação Tecnológica de Minas Gerais
Curso de Modelagem Matemática e Computacional
Av. Amazonas 7675 , Belo Horizonte, 30510-000, MG, Brazil
profmauriciopessoa@yahoo.com.br

[b]Instituto Federal de Minas Gerais
Campus Formiga
Rua Padre Alberico, s/n, São Luiz, Formiga, 35570-000, MG, Brazil
meg@dppg.cefetmg.br

Keywords: Cancer; Hallmarks of cancer; Stem cell hypothesis; Time delay.

We present a mathematical model to study cancer evolution and its relationship with stem cells. Our model has a hierarchical structure with four compartments: stem cell, early progenitors, late progenitors and specialized cells. We suppose that cancer is due to mutations occurring during mitosis. The parameters related to the cell dynamics, except the death rate, are dependent on all cells populations and, therefore, intend to simulate some of the stimulus present in a real tissue. We also consider the time required, by each cell, to complete its division or, in the case of late progenitors, to maturate. This consideration leads to a model of differential time-delay equations. Our results show that the delay is responsible for oscillations inside each population until an equilibrium value is achieved. However, the equilibrium values attained by each population do not depend on the delay, that is, the same values would be obtained for zero time delay. In the absence of mutations, damages that would cause a sudden raise or reduction on the number of specialized cells will be totally absorbed by the cell system in a short period of time. Therefore, the equilibrium predicted by the model is stable. For simulations including the occurrence of mutations, we consider a mutation rate equal to 10^{-7}. We show that a mutation in the stem cell compartment gives rise to a larger number of abnormal cells in a shorter period of time, confirming the cancer stem cells hypothesis. The model allows us to consider mutations that can modify from 1 to 3 of the features involved in the proposed model: the self-renewal probability, the mitotic fraction and the death rate. Our results show that a tiny increase - just larger than 0,1% - on the stem cell self-renewal probability has a dramatic effect on the abnormal cell population. Although this parameter, the self-renewal probability , is not directly related to a special mechanism - symmetric or asymmetrical cell division - we can suggest that a raise in its value favors the symmetrical stem cells division. However, it must be noted that a mutation causing a significant change (greater than 10%) on the mitotic fraction and/or the death rate can also lead to a tumor development in a short period of time. We also simulate the effect of therapies that kill normal and abnormal cells to show that an increase on the frequency of application of these therapies results in a lowering of the abnormal cell population. At last, we show that the whole abnormal tissue can be driven to death when, somehow, all abnormal stem cells are removed from the tissue.

Acknowledments The authors are grateful to FAPEMIG - (*Fundação de Amparo à Pesquisa do Estado de Minas Gerais - Brasil*) for financial support.

TWO-STAGES POPULATION INVASION SPEED AND ORTHOGONAL POLYNOMIALS

M. Mesk[a] and **T. Mahdjoub**[b]

[a]Département de Biologie
Université Abou-Bekr Belkaid, Tlemcen, BP 119 Imama(Pôle2), Tlemcen 13000, Algeria
m_mesk@mail.univ-tlemcen.dz

[b]Département de Biologie
Université Abou-Bekr Belkaid, Tlemcen, BP 119 Imama (Pôle2), Tlemcen 13000, Algeria
tew.mahdjoub@mail.univ-tlemcen.dz

Abstract. Solutions of a linear two stages population system of integrodifference equations describing both demography and dispersal processes are expressed by using orthogonal polynomials. From the asymptotic behavior of these polynomials and the saddle point method [1], the asymptotic behavior of solutions is analyzed.

Keywords : Biological invasions ; Invasion speed; Integrodifference equations ; Method of steepest descent; orthogonal polynomials.

References

[1] M. Kot, and M. G. Neubert, "Saddle-Point Approximations, Integrodifference Equations, and Invasions**",** *Bulletin of Mathematical Biology*, 70, 1790–1826, 2008.

EFFECTIVE POPULATION SIZES AND THE CANONICAL EQUATION OF ADAPTIVE DYNAMICS

J.A.J. Metz[a,b,c]

[a]Mathematical Instittute
Leiden University
Niels Bohrweg 1, NL-2333 CA Leiden, Netherlands

[b]Institute of Biology
Leiden University
Sylviusweg 72, NL-2333BE Leiden, Netherlands
j.a.j.metz@biology.leidenuniv.nl

[c]Evolution and Ecology Program
International Institute for Applied Systems Analysis
A-2361 Laxenburg, Austria

Deterministic population dynamical models connect to reality through their interpretation as limits for systems size going to infinity of stochastic processes in which individuals are represented as discrete entities. In structured population models individuals may be born in different states (e.g. locations in space) after which they proceed through their h(eterogeneity)-state space, e.g. spanned by their i(dividual)-state and location. On such models one can graft evolutionary processes like random genetic drift or adaptive evolution by rare repeated substitutions of mutants in heritable traits affecting the state transition and reproduction processes of individuals. From this general perspective I will derive the so-called Canonical Equation of adaptive dynamics, a differential equation for evolutionary trait change derived under the additional assumption that mutations have small effect. In the CE approximation the rate of evolution is found to correspond to the product of a parameter $n_{\mathrm{e,A}}$, equal to the population size times a dimensionless product of life history parameters (including spatial movements), times the gradient of the invasion fitness of potential mutants with respect to their trait vector. From a heuristic connection with the diffusion approximation for genetic drift it follows that $n_{\mathrm{e,A}} = n_{\mathrm{e,D}}$, the effective population size from population genetics.

MUTATIONS ARISING DURING INVASIONS

J. Miller[a]

[a]Department of Mathematics and Statistics
Georgetown University
Washington DC USA 20057
jrm32@georgetown.edu

When a neutral mutation arises in an invading population, it quickly either dies out or "surfs", i.e. it comes to occupy almost all the habitat available at its time of origin. Beneficial mutations can also surf, as can deleterious mutations over finite time spans. We quantify the relationship between the probability that a mutation will surf and demographic parameters for a cellular automaton model of surfing. We also provide a simple mechanistic model that performs well at predicting the probability of surfing for neutral and beneficial mutations in one dimension. The results suggest that factors—possibly including even abiotic factors—that promote invasion success may also increase the probability of surfing and associated adaptive genetic change, conditioned on such success. Finally, we discuss extensions to the two-dimensional case.

A NEW MATHEMATICAL MODEL OF SYPHILIS

F. Milner[a] and **R. Zhao**[b]

[a]School of Mathematical and Statistical Sciences
Arizona State University
P.O. Box 871804
Tempe, AZ 85287-1804, USA
milner@asu.edu

[b]Bionumerics Research Group
Department of Computer Science
Purdue University
305 N. University Street
West Lafayette, Indiana, 47907-2107, USA
rzhao@cs.purdue.edu

The CDC launched the National Plan to Eliminate Syphilis from the USA in October 1999. In order to reach this goal, a good understanding of the transmission dynamics of the disease is necessary. Based on a SIRS model Breban et al. provided some evidence that supports the feasibility of the plan proving that no recurring outbreaks should occur for syphilis. We study in this work a syphilis model that includes partial immunity and vaccination. This model suggests that a backward bifurcation very likely occurs for the real-life estimated epidemiological parameters for syphilis. This may explain the resurgence of syphilis after mass treatment. Occurrence of backward bifurcation brings a new challenge for the plan of the CDC's striking a balance between treatment of early infection, vaccination development and health education. Our models suggest that the development of an effective vaccine, as well as health education that leads to enhanced biological and behavioral protection against infection in high-risk populations, are among the best ways to achieve the goal of elimination of syphilis in the USA.

MODELLING DYNAMICS AND EVOLUTION OF INFLUENZA A (INDIVIDUALLY BASED APPROACH)

P. Minayev[a] and **N. Ferguson**[a]

[a]MRC Centre for Outbreak Analysis and Modelling
Imperial College London
St.Mary's Campus, Norfolk Place, London W2 1PG, UK
p.minayev@imperial.ac.uk

The evolution and dynamics of influenza A have been a subject of mathematical research for a few decades now, however various aspects of modelling such as immunity acquisition, role of non-specific immune response and, most importantly, correspondence between virus genotype and phenotype still remain debatable.

We study two classes of individually based models characterized by different approaches to modelling antigenic space and acquisition of immunity. In the first class, antigenic changes are directly determined by genetic changes, i.e. there exists an almost direct (up to synonymous mutations) correspondence between genotype and phenotype. An individual's cross-immune response in this case is dependent on Hamming distance in the genotype space from an attacking strain to the immune history of that individual. Transient nonspecific immunity is necessary in this case to eliminate explosive growth of strain diversity and obtain influenza-like evolutionary dynamics. The second class uses a neutralized fitness landscape model to map virus genotypes to an appropriate phenotype in a "many-to-one manner" and the virus evolution mainly occurs along neutral networks, whereas major antigenic changes are fairly infrequent.

Besides antigenic space, an important component of any multistrain model is the host immune response. It can be modelled in many different ways. One of the simplest ones can be called "maximum-per-epitope", where, based on the whole host's immune history, maximum response is chosen for each epitope and the overall response is calculated as an average. Our simulations prove that, in conjunction with transient nonspecific response, it leads to realistic results in terms of evolutionary patterns and temporal dynamics. A more complicated example is an analog of "polarised immunity". We show that it is generally inadequate for modelling influenza in large closed populations as it tends to lead to poor strain diversity and wrong periodicity of seasonal epidemics.

We also use individually based models to demonstrate the importance of spatial heterogeneity on the global evolution of influenza A.

ANALYSIS OF THE BLOOD PRODUCTION STRUCTURED MODEL WITH DELAY FEEDBACK

P.J. Mitkowski[a] and **W. Mitkowski**[b]

[a]Faculty of EAIiE, PhD student
AGH-University of Science and Technology
Al. Mickiewicza 30, 30-059 Kraków, Poland
pawel.mitkowski@agh.edu.pl

[b]Department of Automatics
AGH-University of Science and Technology
Al. Mickiewicza 30, 30-059 Kraków, Poland
wojciech.mitkowski@agh.edu.pl

We consider the following age-structured model of the red blood cell production and destruction system with delay feedback formulated by M. Ważewska-Czyżewska and A. Lasota in [7]

$$\begin{cases} \dfrac{\partial n(t,a)}{\partial t} + \dfrac{\partial n(t,a)}{\partial a} = -\lambda(t,a) \cdot n(t,a), \\ \\ n(t,0) = p(t), \\ \\ p(t) = \rho \cdot e^{-\gamma \int_0^\infty n(t-h,\ a)da}, \end{cases} \tag{1}$$

where $n(t,a)$ is the density of the age distribution of blood cells. The model (1) is known for interesting biological interpretations [7], [6], [4] as well as mathematical consequences [3], [1], [2], [5]. The main goal is to carry out numerical analysis in order to investigate some earlier research hypotheses concerning the system (1).

References

[1] Z. Brzeźniak, and A.L. Dawidowicz, "On periodic solutions to the von Foerster-Lasota equation, *Semigroup Forum* 78, p. 118-137, 2009.
[2] A.L. Dawidowicz, and A. Poskrobko, "On the age-dependet population dynamics with delayed dependence of the structure, *Nonlinear Analysis* 71, p. 2657-2664, 2009.
[3] A. Lasota, "Invariant measures and a linera model of turbulence", *Rendiconti del Semianrio Matematico della Univesità di Padova*, tome 61, p. 39-48, 1979.
[4] A. Lasota, and M.C. Mackey, and M. Ważewska-Czyżewska "Minimizing Therapeutically Induced Anemia", *J. Math. Biology*, 13, p. 149-158, 1981.
[5] R. Rudnicki, "Chaoticity of the blood cell production system", *Chaos* 19, 6 pages, 2009.
[6] M. Ważewska-Czyżewska, "*Erythrokinetics. Radioisotopic Methods of Investigation and Mathematical Approach*", Foreign Scientific Publications Department of the National Center for Scientific, Technical and Economic Information, Warsaw, Poland, 1983.
[7] M. Ważewska-Czyżewska, and A. Lasota "Matematyczne problemy dynamiki układu krwinek czerwonych (Mathematical problems of the dynamics of red blood cell population)", (in Polish), *Matematyka Stosowana* VI, p. 23-40, 1976.

A MODEL FOR AN INFECTIOUS DISEASE WITH TIME DEPENDENT PARAMETERS

M. R. Moalei

In this talk we are going to consider infectious diseases in an open society from the mathematical point of view. An open society means a society which passengers enter to it or leave it. We will pay more attention to the societies which the entrance parameters and leaving parameters are time dependent. The model is an extension of the SIR models. Our approach to this model is an O.D.E. approach. We are also going to speak on the new mathematical methods of the edge group population dynamics to present a new research topic.

EMERGENCE OF HOLLING TYPE III FUNCTIONAL RESPONSE IN ECOSYSTEMS WITH SPATIAL HETEROGENEITY: PLANKTON COMMUNITY AS A CASE STUDY

A. Morozov[a]

[a]Department of Mathematics
University of Leicester, UK
University Road, Leicester, UK LE1 7RH
am379@le.ac.uk

Parameterization of predation terms plays a crucial role in dynamics of food-web models. Theoretical studies predict enhancing of ecosystems' stability in case the functional response is of sigmoid type (Holling type III). In mean-field models, an ecosystem is described in terms of the average over space biomass of species. In this case, the functional response of a predator is understood as the rate of consumption of prey biomass (per predator biomass per unit of time) as a function of average prey biomass. Such functional response can be referred to as an overall functional response opposite to a local functional response, describing consumption of food by an individual predator at a given location. Interestingly enough, the difference between the local the overall functional responses is not only quantitative. It might include as well alteration of type of response. In particular, an overall response of sigmoid type can emerge even in the case the local response is non-sigmoid

As a case study, I consider plankton communities with vertical spatial heterogeneity. In those communities, the local response of herbivorous zooplankton is non-sigmoid, which has been confirmed by extensive experiments in laboratories. I show that despite the non-sigmoid nature of local functional response, the overall response of zooplankton can be of Holling type III. My conclusions are based on large amount of field data on plankton feeding in situ. Also, I suggest several mathematical models based on explicit vertical resolution to describe the observed alteration of types. The suggested mechanism of emergence of sigmoid overall functional response is the interplay between vertical heterogeneity in food distribution and active foraging behaviour of predator (zooplankton) along vertical direction. Thus, I challenge the common opinion in the literature that the implementation of sigmoid functional response in plankton models is meaningless.

References

[1] A. Morozov, and E. Arashkevich, "Patterns of zooplankton functional response in communities with vertical heterogeneity: a model study", *Math. Model. Nat. Phenom.* v. 3, p. 131-148, 2008.
[2] A. Morozov, E. Arashkevich, M. Reigstad, S. Falk-Petersen. "Influence of spatial heterogeneity on the type of zooplankton functional response:a study based on field observations",*Deep-Sea Res. II* v. 55, p. 2285-2291, 2008.
[3] A. Morozov, and E. Arashkevich, "Towards a correct description of zooplankton feeding in models: Taking into account food-mediated unsynchronized vertical migration", *Journal of Theortical Biology*, v. 262, p. 346-360, 2010.
[4] A. Morozov, "Emergence of Holling type III zooplankton functional response: bringing together field evidence and mathematical modelling", *Journal of Theortical Biology* , 2010, *in press.*

PATTERNS OF PATCHY SPREAD IN MODELS OF BIOINVASIONS AND BIOCONTROL

A. Morozov[a]

[a]Department of Mathematics
University of Leicester, UK
University Road, Leicester, UK LE1 7RH
am379@le.ac.uk

In real ecosystems, spread of populations in space often occurs via interaction and propagation of patches of high species density, separated from each other by areas where the species density is close to zero. Such type of spread is called a 'patchy spread' (opposite to one having a continuous population front). In earlier models, the patchy spread was considered to be a result of a pronounced environmental or/and demographic stochasticity. Later, it was found that a patchy spread can arise in a fully deterministic predator-prey system and in models of infectious diseases [1, 2]. In the current study, I continue development of deterministic models of patchy spread. The framework implemented is the reaction-diffusion approach in two spatial dimensions. I demonstrate a number of models of biological invasion and biological control which exhibit patchy spread in a fully homogeneous environment. In particular, a patchy spread arises both in mutualism and competition models influenced by predation. I show as well a pattern of patchy spread having significantly different speeds in different spatial directions in a fully homogeneous space. Earlier such phenomenon was attributed to strong environmental heterogeneity. The basic properties of spatiotemporal dynamics of patchy spread common for the given approach are formulated. Finally, I mark out properties of the 'reaction' terms which are necessary to obtain patchy spread based on the reaction-diffusion modelling framework.

References

[1] S. Petrovskii, H., Malchow, F., Hilker, E. Venturino. "Patterns of patchy spread in deterministic and stochastic models of biological invasion and biological control", *Biological Invasions*, v. 7, p. 771-793, 2005.

[2] A. Morozov, S. Petrovskii, B.L. Li. "Spatiotemporal complexity of the patchy invasion in a predator-prey system with the Allee effect", *Journal of Theoretical Biology*, v. 238, p. 18-35, 2006.

[3] A. Morozov, S. Ruan, and Bai-Lian Li, "Patterns of Patchy Spread in Multi-Species Reaction-Diffusion Models", *Ecological Complexity*, v. 5, p. 313-328, 2008

Optimal Control of Chikungunya Disease: Larvae Reduction, Treatment and Prevention

D. Moulay[a] and **M.A. Aziz-Alaoui**[a]

[a]Laboratoire de Mathématique Appliquées du Havre (LMAH)
University of Le Havre
25 rue Philippe Lebon, BP540, 76058 Le Havre Cedex
djamila.moulay@univ-lehavre.fr

Since the 1980s, there has been a worldwide re-emergence of vector-borne diseases including Malaria, Dengue, Yellow fever, or more recently chikungunya. These viruses are arthropod-borne viruses (arboviruses) transmitted by arthropods like mosquitoes of Aedes genus. The nature of these arboviruses is complex since it conjugates human, environmental, biological and geographical factors. Recent research [1] has suggested, in particular during the Reunion Island epidemic in 2006, that the transmission by Aedes Albopictus has been facilitated by genetic mutations of the virus.

Models describing the Aedes Albopictus mosquito population dynamics, vector of chikungunya virus and transmission to human population are discussed. On the one hand, we propose model using stage structured model based on the biological mosquito life cycle (eggs, larvae, pupae and adults). On the other hand the transmission to human population is described with SIR type model [2]. First we establish global stability results based on the reproductive number like in [3].

Then, based on the observation during one of the last and most important chikungunya epidemic, three main efforts are considered in order to limit the virus transmission [4]. Indeed, there is not vaccine or specific treatment against chikungunya, that's why the main measures includes:

- individual protection against mosquito bites, like the use of insect repellents or mosquito nets, wearing appropriate clothing, etc;
- symptomatic treatment of infected patient;
- control of the proliferation of adult mosquito mainly by reducing the number of larvae in breeding sites which was one of the vector control tools.

Therefore, we look at time dependent breeding sites destruction, prevention and treatment efforts, where optimal control theory is applied. Using analytical and numerical techniques, it is shown that there are cost effective control efforts.

References

[1] M. Enserink, "Epidemiology: Tropical Disease Follows Mosquitoes to Europe", *Science*, v. 317, 2007.
[2] D. Moulay, M.A. Aziz-Alaoui, and M. Cadivel,"The Chikungunya Disease : Modeling, Vector and Transmission Global Dynamics", *submitted to Mathematical Biosciences (2010).*
[3] L. Esteva, and C. Vargas, "Analysis of a dengue disease transmission model", *Mathematical Biosciences*, v. 150,p. 131-151,1998.
[4] D. Moulay, H.D. Kwon M.A. Aziz-Alaoui,"Optimal Control of Chikungunya Disease", *in preparation.*

DYNAMICS OF IMMUNE CELL DIVISION AND DIFFERENTIATION

S. Nakaoka and **K. Aihara**

An immune system protects the host against diseases by detecting and eliminating antigens, including viruses, tumors and so on. Despite of its protective activity, the immune system sometimes incorrectly attacks self-tissues (autoimmunity) or over-react to harmless environmental substances (allergy). In general, these disorders occur as a consequence of dynamical interactions between immune cells and pathogens. Therefore, it is important to understand the dynamics of an immunological network for prediction and control of immune responses. In this talk, we introduce a theoretical study inspired by quantitative measurement of immune cell growth. We construct a mathematical model which describes division/death processes of immune cells. It is shown that a modified birth-death (stochastic) process well describes *in vitro* cell proliferation measurement [1]. Further, the model is modified to incorporate differentiation processes of naive CD4 T cells into either effector Th1 or Th2 subsets. It is shown that heterogeneity among naive CD4 T cells in the amount of gene expression in response to antigenic stimulation can be an important factor to determine the balance of the Th1/Th2 ratio.

References

[1] Hawkins, E.D., Markham, J.F., McGuinness, L.P. and Hodgkin, P.D. “A single-cell pedigree analysis of alternative stochastic lymphocyte fates", *Proc Natl Acad Sci USA* , v. 106, p. 13457-13462, 2009.

Aggregation of variables in a time discrete model and application to a spatially explicit model

T. Nguyen-Huu[a]

[a]UMMISCO
IRD
32 av. Henri Varagnat, 93143 Bondy Cedex
tri.nguyen-huu@ird.fr

Aggregation methods allow to reduce the complexity of models involving different time scales in more mathematically tractable model. In population dynamics spatial models, demography is often considered as a slow time scale process, while dispersal is considered as fast time scale. It is clear that within the demographic processes reproduction should be actually described at a slow time scale, there are nevertheless some examples like survival that are treated or monitored at a fast time scale while they are actually slow time processes.

In this work, we present a class of models of metapopulations connected with dispersal taking the form $R\left((MS^{1/k})^k\right)$, where the dispersal process is represented by a migration matrix M, the survival is represented by a diagonal matrix S and the reproduction process by a function R. The parameter k represents the number of occurence (per unit of time) of the dispersal.

We will present an aggregation method which allows to reduce such a kind of model and then describe an application of this model to the dynamics of a beetle called *abax parallelepipedus* living in Britany. This insect is a very good representation of the biodiversity of the region and is very sensitive to the fragmentation of the landscape. The local model is derived from a Leslie model. The analysis of the aggregated model will entail a good description of the effect of fragmentation and habitat distribution on the long term dynamics of this population.

References

[1] T. Nguyen-huu, R. Bravo de la Parra and P. Auger, "Approximate aggregation of linear discrete models with two time-scales: re-scaling slow processes to the fast scale", *Journal of Difference Equations and applications*, in press.

[2] J.B. Pichancourt, F. Burel, P. Auger, "Assessing the effect of habitat fragmentation on population dynamics: An implicit modelling approach", *Ecological modelling*, v. 192, p. 543-556, 2006.

AGE-DEPENDENT RESPONSE TO THE INFLUENZA A (H1N1) 2009 VIRUS INFECTION

H. Nishiura[ab]

[a]PRESTO
Japan Science and Technology Agency
Kawaguchi-shi, Saitama, Japan

[b]Theoretical Epidemiology
University of Utrecht
Yalelaan 7, Utrecht, 3584 CL, The Netherlands
h.nishiura@uu.nl

Epidemiological observations of the 2009 influenza A (H1N1) pandemic have consistently shown that the infection is most frequent among children. In contrast to interpandemic influenza, the age distribution of the infection has shifted toward children and young adults, because of the age specificities in immunological reactions and transmission characteristics. Although seroepidemiological studies have proven that older people may have been protected by preexisting immunity due to past exposures to similar viruses, the level of immunity and its extent can only be explicitly estimated by means of epidemiological methods.

Three different modelling approaches were taken to estimate the age-dependent immunity. First, a multivariate renewal process was employed to estimate the age-dependent next-generation matrix from the early epidemic growth data in Japan. Despite no strong signature of age-dependent infectiousness, relative susceptibility differed with age. Using those aged from 20-39 years to define the baseline, the age-groups 0-5, 6-12 and 13-19 years appeared to be 2.77 (95% CI: 2.35, 3.24), 2.67 (95% CI: 2.41, 2.95) and 2.76 (95% CI: 2.55, 2.98) times more susceptible than adults aged 20-39 years. Those aged from 40-59 years and 60 years and older were 0.56 (95% CI: 0.45, 0.68) and 0.17 (95% CI: 0.09, 0.28) times as susceptible than those aged 20-39 years [1].

Second, differences in genetic sequence were sought between the 2009 influenza and other H1N1 viruses that have circulated in the 20th century. The sequence data indicated that 2009 H1N1 virus was most similar to H1N1 viruses that circulated before 1943. The 2009 H1N1 lacked glycosylation sites on the globular head of hemagglutinin (HA1) near antigenic regions, a pattern shared with the 1918 pandemic strain and H1N1 viruses that circulated until the early 1940s. Later H1N1 viruses progressively added new glycosylation sites likely to shield antigenic epitopes [2].

Third, household data were analyzed to examine the age-dependency in the frequency of secondary transmissions within the household. Again, there was no strong signature of age-dependent infectiousness, but the relative susceptibility sharply decreased as a function of age. The estimates were quite consistent with those obtained from the next-generation matrix which rests on community transmission data.

In conclusion, the preexisting immunity mainly reduced susceptibility among previously exposed individuals.

References

[1] H. Nishiura, C. Chowell, M. Safan, C. Castillo-Chavez, "Pros and cons of estimating the reproduction number from early epidemic growth rate of influenza A (H1N1) 2009", Theor Biol Med Model, 2010; 7: 1. [2] T. Reichert, G. Chowell, H. Nishiura et al, "Does Glycosylation as a modifier of Original Antigenic Sin explain the case age distribution and unusual toxicity in pandemic novel H1N1 influenza?", BMC Infect Dis, 2010; 10: 5.

A HYBRID MODEL OF WOODPIGEON POPULATION DYNAMICS IN IRELAND

Suzanne M. O'Regan,[a] **Denis Flynn, Thomas C. Kelly, Michael J. A. O'Callaghan, Alexei Pokrovskii,** and **Dmitrii Rachinskii**

[a]Department of Applied Mathematics
University College Cork
Western Gateway Building, Western Road, University College Cork, Cork, Ireland.
s.m.oregan@student.ucc.ie

Keywords: Bird strike; Hybrid system; Competition model; Pest; Woodpigeon.

The woodpigeon *Columba palumbus* is the largest and most abundant pigeon in Ireland. Woodpigeons are herbivores and their diet consists mainly of clover, cereal grains, tree buds, flower seeds and brassicae. Due to their generalist eating habits, it has always been considered a major agricultural pest and has greatly benefited from changing agricultural practices. The recent explosion of the woodpigeon population in Ireland has become a cause for concern as woodpigeons have caused multiple bird strike incidents at Dublin airport.

There are several hypotheses for the cause of the woodpigeon population increase. In the 1960s, the woodpigeon population was fairly stable. Regulation of the woodpigeon population occurred during long, cold winters, through elimination of the "doomed surplus", i.e., those doomed to die. Weaker, subordinate individuals, usually in their first year of life, died mainly from starvation that was most likely caused by intra-specific competition for food. Winters are milder now compared to forty years ago and consequently, the length of the growing season has increased. Therefore, there is decreased competition among woodpigeons for food. In addition, woodpigeons have the potential to breed multiple times per year if conditions are suitable. Furthermore, managing the woodpigeon population in Ireland would require achieving additive mortality through heavy shooting, a difficult and expensive task.

The objective of this research is to investigate the mechanisms behind the woodpigeon population increase using a dynamical model. I will describe the development of a model of the baseline 1960s scenario of a stable population regulated by intra-specific competition. The basic model is a hybrid between a discrete matrix model and a continuous system of ordinary differential equations. The continuous system assumes that regulation is caused by intra-specific competition. Numerical simulations of the hybrid model will be discussed and an algorithm for finding its parameters will be described. Numerical experiments indicate that the hybrid system exhibits varied and interesting dynamics. In addition, the effect of perturbations such as increased fecundity and decreased competition will be examined. Although the growth of the woodpigeon population is considered to be a serious problem, to our knowledge, no dynamical models of woodpigeon population dynamics have been developed.

SINGULAR INFINITE HORIZON CALCULUS OF VARIATIONS: NON UNIQUENESS OF THE OPTIMAL MANAGEMENT OF A FISHERY

E. Ocana[a] and **P. Cartigny**[b]

[a]Department of Mathematics
IMCA-UNI
Lima Perou
eocana@uni.edu.pe

[b]Department of Economie
GREQAM Universit Mditerrane
Marseille France
pierre.cartigny@univmed.fr

Key words: Infinite horizon, calculus of variation, MRAP, transversality condition, Turnpike.

We consider an optimal infinite horizon calculus of variations problem linear with respect to the velocities:

$$\max_{x(.)} \int_{t=0}^{+\infty} e^{-\delta t}[A(x(t)) + B(x(t))\dot{x}(t)]dt\,,$$

where the state variable x is scalar and the velocities belong to a closed set.
In this case the Euler-lagrange equation is algebraic (in general it is a differential equation) and the interior solutions are given by constant values. The question is thus to determine the optimal solutions from initial conditions. The curves that connect as quickly as possible the initial conditions with the solutions of the Euler-Lagrange equation, called MRAP for Most Rapid Approach Pathes, are candidates. We don't consider any concavity assumption, and therefore we have to solve straightforwardly the question of the optimality of these particular solutions.
We present a direct proof of the optimality of the MRAPs when the Euler-Lagrange possesses a finite number of solutions, the case with only one solution being well-known. We underline in particular the role of the transversality condition.
It is well-known that the optimal management of a fishery can be modelised in this way. A main result : the same optimal revenue for the fishery can be obtained with different policies of efforts, corresponding to different levels of the stock of biomass.

SEARCHING FOR THE MOST COST-EFFECTIVE STRATEGY OF CONTROLLING EPIDEMICS

Katarzyna Oleś [a] and **Dr Adam Kleczkowski**[b]

[a]Faculty of Physics, Astronomy and Applied Computer Science
M. Smoluchowski Institute of Physics, Mark Kac Center for Complex Systems Research
ul. Reymonta 4, 30-059 Kraków, Poland

[a]Department of Computing Science and Mathematics
University of Stirling
Stirling FK9 4LA, United Kingdom
kas@cs.stir.ac.uk

[b]Department of Computing Science and Mathematics
University of Stirling
Stirling FK9 4LA, United Kingdom
ak@cs.stir.ac.uk

Models for control of spread of an infectious disease on local, small-world, and random networks are considered. We assume that only partial information is available about the status of individuals as they can be infectious before showing symptoms and thus can avoid detection. We consider various control strategies and search for the most cost-effective one. Traditionally, epidemiology concentrated purely on a reduction of number of cases without considering economic and social costs. If costs are associated with both reactive (hospitalisation) and preventive (vaccination or culling) treatment, three main strategies emerge, with treating all individuals indiscriminately (global strategy), treating only individuals in a well-defined neighbourhood of a detected case (local strategy) and allowing the disease to spread unchecked (null strategy). The choice of the optimal strategy is governed mainly by a relative cost of preventive and reactive treatments. If the disease spreads locally, the local strategy is optimal unless the cost of a single vaccine is much higher than the cost associated with hospitalisation. In the latter case, it is most cost-effective to refrain from prevention. Destruction of local correlations, either by long-range (small-world) links or by inclusion of many initial foci, expands the range of costs for which the null strategy is most cost-effective. The global strategy emerges for the case when the cost of prevention is much lower than the cost of treatment and there is a substantial non-local component in the disease spread. The details of the local control strategy, and in particular the optimal radius of control, are determined by the epidemiology of the disease. The properties of the pathogen might not be known in advance for emerging diseases, but the choice of the strategy can be made based on economic analysis only.

Keywords: epidemiological modelling, disease spread, stochastic modelling, epidemiological control, dispersal patterns.

OPTIMAL CONTROL FOR POPULATION DYNAMICS OF INCOMPLETE DATA

A. Omrane and **B. Jacob**

We are concerned with the control question for linear age- structured population dynamics of incomplete initial data. More precisely, the initial population age distribution is supposed to be unknown. We here generalize the notion of no-regret control of J.-L. Lions to such singular population dynamics, following the method by Nakoulima, Omrane and Velin (C.R.A.S 2000). We prove that the problem we are considering has a unique no-regret control that we characterize by a singular optimality system.

GLOBAL STABILITY ANALYSIS OF A SYSTEM MODELING CELL DYNAMICS IN ACUTE MYELOGENOUS LEUKEMIA

H. Özbay[a], **C. Bonnet**[b], **H. Benjelloun**[b] and **J. Clairambault**[b],

[a]Department of Electrical and Electronics Eng.
Bilkent University, Bilkent, Ankara, 06800 Turkey
currently, on leave at[b]
hitay@bilkent.edu.tr; Hitay.Ozbay@inria.fr

[b]INRIA Paris-Rocquencourt,
Domaine de Voluceau, B.P. 105
78153 Le Cesney, Cedex, France
{Catherine.Bonnet,Houda.Benjelloun,Jean.Clairambault}@inria.fr

Keywords: distributed delays, stability, nonlinear small gain, hematopoietic cell maturation, AML

We investigate stability of a system modeling the cell dynamics in Acute Myelogenous Leukemia (AML), originally proposed in [1]; see also [2,3,4]. This system can be seen as a cascade connection of sub-systems consisting of distributed delays and static nonlinear feedbacks.

Local asymptotic stability were obtained in [1] and [4]. Later these results were improved by the Nyquist stability analysis of the linearized system around the positive equilibrium in [5] and [6]. From the nonlinear system theoretic point of view, an analysis is made in [7] by using the circle and Popov criteria for absolute stability. In this paper, we derive a global stability condition by using a nonlinear small gain argument. We also illustrate the results with numerical examples and simulations.

References

[1] Adimy, M., Crauste, F., El Abdllaoui, A., "Discrete maturity-structured model of cell differentiation with applications to acute myelogenous leukemia", *J. Biological Systems*, vol. 16 No. 3 (2008), pp. 395–424.
[2] Adimy, M., Crauste, F., "Modeling and Asymptotic Stability of a Growth Factor-Dependent Stem Cell Dynamics Model with Distributed Delay," *Discrete and Continuous Dynamical Systems - Series B*, vol. 8 (2007), pp. 19–38.
[3] Adimy, M., Crauste, F., El Abdllaoui, A., "Asymptotic behavior of a discrete maturity structured system of hematopoietic stem cell dynamics with several delays" *Mathematical Modelling and Natural Phenomena* vol. 1, No. 2, (2006), pp. 1-19.
[4] Adimy, M., Crauste, F., Ruan, S., "A Mathematical Study of the Hematopoiesis Process with Applications to Chronic Myelogenous Leukemia," *SIAM J. Appl. Math.*, vol. 65 (2005), pp. 1328–1352.
[5] Ozbay, H., Bonnet, C., Clairambault, J., "Stability Analysis of Systems with Distributed Delays and Application to Hematopoietic Cell Maturation Dynamics," in *Proc. of the 47th IEEE Conference on Decision and Control*, Cancun, Mexico, December 2008, pp. 2050-2055.
[6] Ozbay, H., Benjelloun, H., Bonnet, C., Clairambault, J., "Stability Conditions for a System Modeling Cell Dynamics in Leukemia," to appear in the *Proceedings of the 9th IFAC Workshop on Time Delay Systems, TDS2010*, Prague, Czech Republic, June 2010.
[7] Ozbay, H., Benjelloun, H., Bonnet, C., Clairambault, J., "Absolute Stability of a System with Distributed Delays Modeling Cell Dynamics in Leukemia," to appear in the *Proceedings of the 19th International Symposium on Mathematical Theory of Networks and Systems* (MTNS 2010), Budapest, Hungary, July 2010.

DYNAMIC OF NEURONS POPULATION

K. Pakdaman[a], **B. Perthame**[b] and **D. Salort**[c]

[a] Institut J. Monod
Universite Paris-Diderot
Batiment Buffon - 15 rue Helene Brion - 75013 Paris - France
pakdaman@ijm.univ-paris-diderot.fr

[b] Laboratoire Jacques-Louis Lions
UPMC Univ Paris 06
175 rue du Chevaleret 75013 Paaris - France
benoit.perthame@upmc.fr

[c] Institut J. Monod
Universite Paris-Diderot
Batiment Buffon - 15 rue Helene Brion - 75013 Paris - France
salort.delphine@ijm.univ-paris-diderot.fr

This talk is devoted to the mathematical study of an homogenous network of neurons, that is to say that we assume that each neuron of the network is governed by the same dynamic. Moreover all the neurons are excitatory. To do this, we introduce an age-structured model given by

$$\begin{cases} \frac{\partial n(s,t)}{\partial t} + \frac{\partial n(s,t)}{\partial s} + p(s, X(t))\ n(s,t) = 0, \\ N(t) := n(s=0,t) = \int_0^{+\infty} p(s, X(t))\ n(s,t) ds, \end{cases} \tag{1}$$

completed with an initial probability density $n^0(s)$ that satisfies

$$0 \leq n^0(s) \leq 1, \qquad \int_0^{\infty} n^0(s) ds = 1. \tag{2}$$

The coefficient $p(s, X)$ represents the firing rate of neurons in 'state s' and in an environment X resulting from the global neural activity. The function $n(s,t)$ models the probability density of finding a neuron in 'state' s at time t where s represents the time elapsed since the last discharge.

The aim is to understand the link between the strengh of the interconnections of neurons in the network and the appareance of synchronous rhythmic activity. To do this, in a first step, we consider the case where the neurons are not interconnected and we prove that, in our model, total asynchronous firing of neurons assymptotically appears. In a second step, we consider the case with interconnections. In particular, we give some conditions on the assumptions concerning the strengh of interconnections to obtain a resynchronization of neural activity in our network.

Age-dependent immune response and antigenic drift in influenza.

Andrea Parisi[a], **Ana Nunes**[a] and **M. Gabriela M. Gomes**[b]

[a]Centro de Fsica da Matria Condensada and Departamento de Fsica
Complexo Interdisciplinar da Universidade de Lisboa
Av. Prof. Gama Pinto 2, 1649-003 Lisboa, Portugal
parisia@ptmat.fc.ul.pt

[b]Instituto Gulbenkian de Cincia
Apartado 14, 2781-901 Oeiras, Portugal

Human Influenza A is characterized by different subtypes, defined by the antigenicity of the corresponding Hemmaglutinin protein which is recognized by the host immune system. When a human host immunized by a certain strain of influenza is infected by a new influenza virus, the immune system is able to promptly respond to the aggression provided the virus presents only a few mutations with respect to the previous immunizing strain. In general a number of mutations are necessary in more than one antigenic site in order for the immune system not to be able to recognize the virus. So, on the one hand immune escape is possible when a number of mutations occur; on the other hand, any new mutation of an infecting virus will still be recognized by the immune system and will hardly survive in the host. Hence the mechanism through which new influenza strains can escape the immune response and spread, leading to the occurrence of an antigenic drift, is still not understood. Here we propose a possible origin for such mechanism. Some studies [1,2] have underlined the fact that the immune response of adult individuals is more developed than that of young individuals. In particular, young humans with age up to a few years show a high degree of specificity in their immune response. Hence, we explore how an age-dependent immune response in humans can lead to interesting epidemic dynamics with synchronized subsequent epidemics of mutants in both the adult's and children populations.

ADAPTIVE DYNAMICS OF COOPERATION MAY PREVENT THE COEXISTENCE OF DEFECTORS AND COOPERATORS AND EVEN CAUSE EXTINCTION

Kalle Parvinen[a]

[a]Department of Mathematics
FI-20014 University of Turku
Finland
kalle.parvinen@utu.fi

It has recently been demonstrated [1,2] that ecological feedback mechanisms can facilitate the emergence and maintenance of cooperation in public goods interactions: the replicator dynamics of defectors and cooperators can result, for example, in the ecological coexistence of cooperators and defectors. Here [3] we show that these results change dramatically, if cooperation strategy is not fixed but instead is a continuously varying trait under natural selection. For low values of the factor with which the value of resources is multiplied before they are shared among all participants, evolution will always favour lower cooperation strategies, until the population falls below an Allee threshold and goes extinct, thus evolutionary suicide [4,5] occurs. For higher values of the factor there exists a unique evolutionarily singular strategy, which is convergence stable. Because the fitness function is linear with respect to the strategy of the mutant, this singular strategy is neutral against mutant invasions. This neutrality disappears if a non-linear functional response in receiving benefits is assumed. For strictly concave functional responses singular strategies become uninvadable. Evolutionary branching, which could result in the evolutionary emergence of cooperators and defectors, can occur only with locally convex functional responses, but we illustrate that it can result also in coevolutionary extinction.

References

[1] C. Hauert, M. Holmes, and M. Doebeli "Evolutionary games and population dynamics: maintenance of cooperation in public goods games." *Proceedings of the Royal Society of London B: Biological Sciences* **273**, 2565–2570, (2006)
[2] C. Hauert, M. Holmes, and M. Doebeli "Corrigendum: Evolutionary games and population dynamics: maintenance of cooperation in public goods games." *Proceedings of the Royal Society of London B: Biological Sciences* **273**, 3131–3132, (2006)
[3] K. Parvinen, "Adaptive dynamics of cooperation may prevent the coexistence of defectors and cooperators and even cause extinction", *Proceedings of the Royal Society of London B: Biological Sciences*, Published online before print April 7, 2010, doi: 10.1098/rspb.2010.0191
[4] R. Ferrière, "Adaptive responses to environmental threats: evolutionary suicide, insurance, and rescue." *Options* Spring 2000, IIASA, Laxenburg, Austria, 12–16.
[5] K. Parvinen, "Evolutionary suicide." *Acta Biotheoretica* **53**, 241–264 (2005)

REAL-TIME GROWTH RATE FOR SIR EPIDEMICS IN SOCIALLY STRUCTURED POPULATIONS

L. Pellis, **N. M. Ferguson** and **C. Fraser**

Department of Infectious Disease Epidemiology
Imperial College London
St. Mary's Hospital, Norfolk Place, London, W2 1PG
l.pellis05@imperial.ac.uk

Keywords: real-time growth rate, Malthusian parameter, SIR model, stochastic epidemic, epidemic dynamics, households, workplaces, social structure, household reproduction number

SIR epidemic models for socially structured human populations (e.g. including households, workplaces, schools...) naturally suggest a stochastic modelling approach to deal satisfactorily with frequent mixing in small groups. In particular, such models have found a florid ground in the so-called standard stochastic SIR model.

Unfortunately, such a model is somewhat not designed to focus on the dynamics of infection spread. Therefore, despite being of great practical interest, results about the epidemic dynamics in socially structured populations are very limited (usually restricted only to the unrealistic Markovian case of constant recovery rate).

Here I present a refinement of the standard stochastic model that is more suitable to realistically characterise the dynamics of the infection spread and suggest a novel and coherent framework for the numerical computation of the real-time growth rate (i.e. the simplest piece of information concerning the epidemic dynamics in a large and fully susceptible population) in a model with households and (time permitting) with households and workplaces.

Because the real-time growth rate is often one of the first pieces of information readily available during an emerging outbreak, a practically useful application consists in reversing the methodology to allow the estimation of the household reproduction number from the real-time growth rate.

References

[1] L. Pellis, "Mathematical models for emerging infections in socially structured populations: the presence of households and workplaces, *PhD Thesis*, 2008, http://hdl.handle.net/10044/1/4696
[2] C. Fraser, "Estimating individual and household reproduction numbers in an emerging epidemic", *PLoS ONE*, 2(8), e758, 2007, doi:10.1371/journal.pone.0000758

THE COARSE MESH PROBLEM IN ECOLOGICAL MONITORING

N. Petrovskaya

School of Mathematics
University of Birmingham
Birmingham, B15 2TT, The United Kingdom
n.b.petrovskaya@bham.ac.uk

Monitoring of pest insects is an essential part of the integrated pest control and management program. Its purpose is to provide information about the population size of the pest species in a given agricultural field or area in order to avoid unjustified pesticides application and yet to prevent pest outbreaks. Depending on the pest biology, the information about pest population size is usually collected through sampling or trapping. The traps are exposed for a certain time, after that they are emptied and the caught insects are counted. The trap counts obtained in this way provide information about the pest population density at the position of the traps. In order to derive an estimate of the population size, i.e. the total number of the insects in the field, the space-discrete function (the population density) should be integrated over the whole monitored area by a chosen method of numerical integration.

From a mathematical viewpoint installing traps in a domain where sampled data are collected means that the discrete integrand function is defined on a (uniform) computational mesh. This is a conventional problem of numerical integration, as integration of sampled data frequently arises in experimental work as well as in computational applications. However, the integration of a discrete function usually implies that the mesh over the domain where the integration is to be taken can be made sufficiently fine to provide required accuracy, while the situation in the pest monitoring problem is essentially different. The matter is that, in routine monitoring, the number of traps installed in a field cannot be made large. Although this is not a principal limitation of the method, its practical reasons are important and cannot be neglected. First, installment of many traps per a unit agricultural area would by itself bring a considerable damage to the agricultural product and hence would make the whole procedure rather senseless. The second important factor that must be taken into account is that the financial and labor resources available for monitoring are always limited. Thus the number of traps installed over a typical agricultural field and/or the number of samples taken very rarely exceed a few dozens and the problem of pest monitoring requires numerical integration of a discrete function obtained on a very coarse mesh.

In our talk we introduce a method to perform accurate numerical integration of sparse data. A discussion of the method will mainly be focused on the question of how to obtain the integral evaluate with the maximum accuracy possible on a given coarse mesh with a fixed number of mesh nodes, especially in case when the integrand may have a nontrivial spatial structure such as regions with a large solution gradient, irregular spatial oscillations, etc. For the validation of our approach we consider a generic population dynamics model to generate different spatial population distributions and to show that implementation of our method allows one to achieve reasonable accuracy on a coarse mesh.

NOISE CAN PREVENT ONSET OF CHAOS IN SPATIOTEMPORAL POPULATION DYNAMICS

S. Petrovskii

Department of Mathematics
University of Leicester
Leicester, LE1 7RH, U.K.
sp237@le.ac.uk

Many theoretical approaches predict the dynamics of interacting populations to be chaotic but that has very rarely been observed in ecological data. It has therefore risen a question about factors that can prevent the onset of chaos by making, in a spatially explicit case, the population fluctuations synchronized over the whole habitat. One such factor is stochasticity. The so-called Moran effect predicts that a spatially correlated noise can synchronize the local population dynamics in a spatially discrete system, thus preventing the onset of spatiotemporal chaos. On the whole, however, the issue of noise has remained controversial and insufficiently understood. In particular, a well-built nonspatial theory infers that noise enhances chaos by making the system more sensitive to the initial conditions. In this paper, we address the problem of the interplay between deterministic dynamics and noise by considering a spatially explicit predator-prey system where some parameters are affected by noise. Our findings are rather counter-intuitive. We show that a small noise (i.e. preserving the deterministic skeleton) can indeed synchronize the population oscillations throughout space and hence keep the dynamics regular, but the dependence of the chaos prevention probability on the noise intensity is of resonance type. Once chaos has developed, it appears to be stable with respect to a small noise but it can be suppressed by a large noise. Finally, we show that our results are in a good qualitative agreement with available field data.

STATISTICAL MECHANICS OF POPULATION DYNAMICS AND ANIMAL MOVEMENT

S. Petrovskii

Department of Mathematics
University of Leicester
Leicester, LE1 7RH, U.K.
sp237@le.ac.uk

Populations of ecological species do not remain fixed in space. Instead, their distribution evolves due to motion of individuals. A cornerstone for understanding mechanisms of dispersal is identification of factors affecting the shape of the dispersal curve, in particular, its rate of decay at large distance. The standard random walk approach is widely believed to result in a dispersal curve with a 'thin' Gaussian tail, which is often at variance with field data. Here we show that the thin tail is more an artifact of an over-simplified description of the dispersing population rather than immanent property of the random walk diffusion. Specifically, we show that a fat-tailed dispersal curve with either exponential or power law rate of decay arises naturally in a population of non-identical individuals, i.e. in a population with some inherent statistical structure [1]. Our findings prove that, contrary to a widely spread opinion, a thick dispersal tail is not necessarily a fingerprint of Levy flights or "superdiffusion" as it is usually believed. Our theoretical results appear to be in a very good agreement with some available field data.

A good understanding of population dispersal is hardly possible without knowing what happens on the "microscale", i.e. on the spatial/temporal scales of the individual movement. Correspondingly, we then proceed to the analysis of animal's individual paths. Movement paths are characterized by the distribution of the length and duration of bouts of continuous movements. Studies of different species have revealed that the distribution of bout durations often has a "fat tail" which is well described by a power law, truncated at high values so that the distribution decays fast for long bouts. The relation between this pattern and the processes that cause it has remained poorly explored. Our attempt to reveal this relation is based on some tools statistical mechanics. Basing on the concept of "statistically structured populations" [1], here we formulate an approach that allows us to describe data on bout duration within a unified framework and show that a truncated fat-tail in the bout distribution of animal movement is an immediate consequence of the inherent statistical variation of individual traits.

References

[1] S. Petrovskii, and A. Morozov, "Dispersal in a statistically structured population: Fat tails revisited", *American Naturalist*, v. 173, p. 278-289, 2009.

March 4, 2010 18:23

Can a mathematical model be helpful to determine optimal control procedures for grapevine pest populations ?

Delphine PICART[1], Bedr'Eddine AINSEBA[2], Fabio MILNER[1], Denis THIERY[3]

[1] *Arizona State University, School of Mathematical and Statistical Sciences, Tempe, AZ 85287-1804, United States,*
delphine.picart@asu.edu

[2] *INRIA Bordeaux Anubis, UFR Sciences et Modélisation, Université Victor Segalen Bordeaux 2-3, Place de la Victoire Bâtiment Leyteire 33076 Bordeaux Cedex, France,*

[3] *UMR 1065 INRA-ENITAB en Santé Végétale, Institut des Sciences de la Vigne et du Vin, centre de recherches de Bordeaux, B.P.81, F-33883 Villenave d'Ornon Cedex, France.*

Lobesia botrana, the European Grapevine moth (EGVM) has been the most serious wine pest in Europe, North Africa and in many Asian countries since the end of the XVIII century. The larvae spark off important economic damages. By feeding the grapes, not only the number of fruits is reduced but also their quality by favoring indirectly the development of pathogens on the fruits, as for example the grey mold or the black rots. Several families of pesticides and control techniques are used to decrease the population size of the moth, e.g. insecticides, insect growth regulators, Bt toxins and mating disruption. But their extensive use leads today to the existence of serious environmental damages that are expected to increase in the future due to climatic changes. Therefore, the main objective in agricultural research is to reduce the use of these products and to develop new methods more Eco-aware.
A solution, to optimize the number of treatments and their efficient, is to determine the best period during the pest life cycle to apply the products, and dynamic models should be helpful in this way. In this talk, we present an age and stage structured model that describes the insect population dynamic in its environment. After a short description on their functioning, the different control techniques just cited above are then add in this model. We finally determinate, through the study of an optimal control problem, the better control procedure to fight this insect population.

Keywords : population dynamics ; age structured population ; Optimal control.

Modelling the dynamics of dengue real epidemics

S.T.R. Pinho[a], **C.P. Ferreira**[b], **L. Esteva**[c], **F.R. Barreto**[d], **V.C.M. Silva**[e] and **M.G.L. Teixeira**[f]

[a]Instituto de Física
Universidade Federal da Bahia
40210-340, Salvador, BA, Brazil
suani@ufba.br

[b]Depto de Bioestatística
Instituto de Biociências, Univ. Estadual Paulista
18618-000, Botucatu, SP, Brazil
pio@ibb.unesp.br

[c]Facultad de Ciencias
Universidad Nacional Autónoma de México
04510, México, D.F., Mexico
esteva@lya.fciencias.unam.mx

[d,e,f]Instituto de Saúde Coletiva
Universidade Federal da Bahia
0110-040, Salvador, BA, Brazil
[d]florisneide@ufba.br, [e]morato@gmail.com, [f]magloria@ufba.br

The infectious diseases are still a relevant problem for human life. Nowadays, due to the intense flow of people around the world and within the cities, the understanding of their complex dynamics is a multi-disciplinary issue. Concerning dengue, a vector transmitted disease, there is no vaccine against any of the four serotypes of the virus, although many efforts have been done in that direction. As a result, dengue transmission control is based on the control of the aquatic and adult mosquito forms. So far, the modelling of the dynamics of dengue may be very helpful for testing both the adopted vector control strategies and the action of future vaccines.

In South and North America, there are records of occurrence of all serotypes of dengue virus, while in Brazil, until now, only 3 serotypes (DENV1, DENV2 and DENV3) have been reported. However, Brazil is responsible for 80% of dengue cases in South America and 60% of notified cases around the world. The circulation of the three serotypes represent an important risk factor for the occurrence of dengue hemorrhagic fever (DHF). Although all the efforts applied by the Brazilian dengue control program to stop dengue transmission, it is still a relevant problem in the first decade of this century. Two factors had been associated to the failure of dengue control: the vector's adaptive capacity and the occurrence of new virus strains.

In this work we use a mathematical model for dengue transmission with the aim to analyze and compare two dengue epidemics that occurred at Salvador, Brazil in 1995-1996 and 2002. Using real data, we obtain the force of infection, Λ, and the basic reproductive number,R_0 for both epidemics. We also obtain the time evolution of the effective reproduction number, $R(t)$, which result to be a very asuitable measure to comparing the patterns of both epidemics. Based on the estimations of R_0 and $R(t)$ we show that control applied only on the adult stage of the mosquito population is not sufficient to stop dengue transmission, emphasizing the importance of the control applied on the aquatic mosquito phase.

ENRICHMENT PARADOX INDUCED BY SPATIAL HETEROGENEITY IN A PHYTOPLANKTON - ZOOPLANKTON SYSTEM

J. -C. Poggiale[a], **M. Gauduchon**[a] and **P. Auger**[b]

[a]Centre d'Oceanologie de Marseille
Universite de la Mediterranee - UMR CNRS 6117
Case 901 - Campus de Luminy - 13288 Marseille Cedex - France
jean-christophe.poggiale@univmed.fr
mathias.gauduchon@univmed.fr

[b]UMI 209 UMMISCO
Institut de Recherche pour le Developpement
32 Avenue Henri Varagnat - 93143 Bondy Cedex - France
Pierre.Auger@bondy.ird.fr

This talk deals with the study of a predator - prey model in a patchy environment. The model represents the interactions between phytoplankton and zooplankton in the column water. Two patches are considered with respect to light availability: one patch is associated to the surface layer, the second patch describes the bottom layer. We show that this spatial heterogeneity may destabilize the predator - prey system, even in oligotrophic system where the nutrient is low enough to avoid "paradox-enrichment" phenomenon. Indeed, in this case, an heterogeneity index can be used as a bifurcation parameter, leading to a Hopf bifurcation. Moreover, we assume that individuals can be dispersed in both patches via hydrodynamism processes, like in a mixed layer. The effect of mixing intensity is analyzed as well as interactions between dispersion and enrichment. We also show that, in some cases, spatial heterogeneity has a stabilizing effect. These contradictory results are examined by considering the non linear interaction between heterogeneity, dispersal and enrichment and some mechanisms leading to stabilization/destabilization are provided.

Modeling the MAPK Pathway in Starfish Oocytes

Ioana Policeanu[1],

[1]Department of Computer Science
Florida Institute of Technology
Melbourne, FL 32901 USA

Semen Koksal[2]

[2]Dept. of Mathematical Sciences
Florida Institute of Technology
Melbourne, FL 32901 USA

and

David Carroll[3]

[3]Dept. of Biological Sciences
Florida Institute of Technology
Melbourne, FL 32901, USA

Abstract

In this talk, we will introduce a mathematical model, a system of nonlinear ordinary differential equations, which simulates the MAPK pathway, one of the most important biological cascades within the cell. Although this cascade contains numerous components, not all are accounted for in this model. Instead, we focus on the last and most important biological enzymes, Raf*, MEK and MAPK, whose input is directly derived from the initial hormone, 1-MA, which is added to the cell in order to start the cascade. This is the first model that analyzes the activation of three kinesis to 1-MA. In addition, model set up, parametric estimations and numerical simulations will be given. Comparison between the experimental and mathematical results will be discussed.

Key Words: 1-MA, MAPK pathway, nonlinear ordinary differential equations.

MODELING THE ASSEMBLY OF INTERMEDIATE FILAMENTS

S. Portet[a]

[a]Department of Mathematics
University of Manitoba
342 Machray Hall, Winnipeg, MB, R3T 3N2, Canada
portets@cc.umanitoba.ca

The cytoskeleton, a complex arrangement of structural proteins organized in networks, is involved in many major cellular functions; its organization is the main determinant of its function, and its misorganization is the cytological signature of some human diseases. One of the three main components of the cytoskeleton are intermediate filaments. Mutations on their proteins can lead to several diseases such as Alexander disease, cardiomyopathy, and amyotrophic lateral sclerosis. Here, models to study the dynamics of the assembly of filaments and networks will be presented.

ON THE EVOLUTION OF MAGIC TRAITS AND SPECIATION

Tadeas Priklopil

Department of Mathematics and Statistics
University of Helsinki
P.O. Box 68 FI-00014 University of Helsinki
tadeas.priklopil@helsinki.fi

We study the adaptive dynamics of a so-called magic trait, which is under natural selection and which also serves as a cue for mating. We derive general results on the monomorphic evolutionary singularities. Next, we study the long-term evolution of single-locus genetic polymorphisms under different forms and strengths of mating preferences in a version of Levene's soft selection model, where natural selection favours different values of a continuous trait within two habitats. If adaptive dynamics leads to a polymorphism with sufficiently different alleles, then the corresponding homozygotes cease to interbreed so that speciation occurs.

References [1] E. Kisdi, and T. Priklopil, "Evolutionary branching of a magic trait", Submitted.

MODELLING DRUG RESISTANCE IN HIV TYPE 1: ASSESSING THE IMPACT OF ANTIRETROVIRAL THERAPY

Silvia Martorano Raimundo[a,b], **Ezio Venturino**[c] and **Hyun Mo Yang**[d]

[a]Department of Patology, Faculdade de Medicina, Universidade de São Paulo
Rua Teodoro Sampaio, 115, Instituto Oscar Freire, CEP: 05405-000, São Paulo, S.P., Brazil
[b]Fondazione ISI, Villa Gualino, viale Settimio Severo 65, 10133, Torino - Italia
corresponding author: silviamr@dim.fm.usp.br

[c]Dipartimento di Matematica "Giuseppe Peano", Universita' di Torino
via Carlo Alberto,10, 10123, Torino, Italia
ezio.venturino@unito.it

[d]Department of Mathematics, Universidade de Campinas
IMECC, DMA, Campinas, S.P., Brasil
hyunyang@ime.unicamp.br

keywords: HIV infection, drug resistant, fitness, epidemic models.

This work proposes a mathematical model to describe the dynamic of both drug-sensitive and drug-resistant HIV-1 infected patients that are submitted to antiretroviral therapies (ART). In our model, drug-resistant strains can evolve during treatment of drug-sensitive cases. We accounted for the fact that after the beginning of the treatment the patient may evolve to success (satisfactory virus suppression) or virological fail, and infected individuals who have treatment successfully for a period of time may also present a secondary virological fail. Treatment is changed for drug intolerance (substitution) or virologic failure (selection by resistance tests). We determine an analytical expression for R_S^* and R_R^* which are defined, respectively, as the average number of new sensitive and resistant HIV-infections that one infected individual will generate during his/her lifetime in a community where ART are available. Due to the fact that drug-resistant virus is less susceptible to ART, the mutant variant is more fit than the wild-type virus (sensitive) in the presence of drug, although resistance mutations may decrease the intrinsic capacity of the virus to replicate. The basic reproductive number is a commonly used measure of the absolute fitness of a virus within a host [1]. We examine the effect of ART on the HIV-1 fitness resistant virus by analyzing the reproductive number in the presence of ART. In this way, the goal is to assess the impact of drug-resistance on "transmission fitness" (i.e., the relative ability of viruses to infect susceptible hosts, guaranteeing the continued transmission dynamics of drug-resistant strains) maintaining these basic reproductive numbers (R_S^* and R_R^*) greater than one, and to establish whether a person who harboured a resistant strain would cause the same number of secondary cases as a person with a sensitive strain.
We conclude that if drug-resistant HIV is transmitted substantially less frequently than wild-type HIV from individuals who have acquired drug-resistant strains while receiving ART then there is coexistence of both resistant and sensitive strains. However, if drug-resistant HIV is transmitted substantially more frequently than wild-type HIV then there is competion between both resistant and sensitive strains, and the resistant strains emerge while the sensitive strains will go to extinction.

References

[1] M. A. Gilchrist, D. Coombs, A.S. Perelson Optimizing whitin-host viral fitness: infected cell lifespan and virion production rate. J. Theor. Biol. 229, 281-288, 2004.

A CONTINUOUS SIZE STRUCTURED MODEL APPLIED TO ZOOPLANKTON COMMUNITY

Jonathan Rault[a], **Eric Benoit**[b], **Pieter Vandromme**[c]

[a]INRIA - COMORE Project Team
2004 Route des Lucioles - BP93, F-06902 Sophia Antipolis Cedex, France
jonathan.rault@sophia.inria.fr

[b]Laboratoire de Mathématiques
Université de La Rochelle
Avenue Michel Crépeau, 17042 La rochelle, France
ebenoit@univ-lr.fr

[c]Laboratoire d'Océanographie de Villefranche
CNRS & Universite Paris VI
Station Zoologique, B.P. 28, F-06230 Villefranche-Sur-Mer, France
pvand@obs-vlfr.fr

Size appears to be a very good indicator of physiological behaviour in the zooplankton community, like predation or even detritus production. In order to get a better understanding of planktonic ecosystem dynamics, we build a continuous size-structured model. This approach is very popular in fish studies (Benoit and Rochet,2004; Andersen and Pedersen, 2009). Our model incorporates predation on phytoplankton and cannibalism within the zooplankton community, and various predation behaviours are tested.

Mathematical results are in the most cases hard to get in such models. Nevertheless for some choice of predation function, and external mortality, we can obtain mathematically equilibria. Numerical simulations show that these equilibria are not always stable, and Hopf bifurcations appear when model parameters vary.

Keywords: Size-structured model, Allometry, Predation, Cannibalism, Dynamic energy budget theory

References

[1] E. Benoit, and M.J. Rochet, "A continuous model of biomass size spectra governed by predation and the effects of fishing on them", *Journal of Theoretical Biology 226, 9-21, 2004*.
[2] O. Maury, B. Faugeras, Y.J. Shin, J.-C. Poggiale, T. Ben Ari, and F. Marsac, "Modeling environmental effects on the size-structured energy flow through marine ecosystems. part 1: The model", *Progress In Oceanography 74, 479-499, 2007*
[3] K.H. Andersen and M. Pedersen, "Damped trophic cascades driven by fishing in model marine ecosystems", *Proc. R. Soc. B 2010 277, 795-802, 2009*
[4] R.W. Sheldon, A. Prakash and W.H.J. Sutcliffe, "The size distribution of particles in the ocean", *Limnol. Oceanogr. 17 (3), 327-340, 1972*
[5] A.M. De Roos, "A gentle introduction to physiologically structured population models", *Structured-population models in marine, terrestrial, and freshwater systems, Tuljapurkar & Caswell editors, Chapman & hall, 1997*

A MODEL FOR AN INFECTIOUS DISEASE WITH TIME DEPENDENT PARAMETERS

Mohammad Reza Molaei

In this talk we are going to consider infectious diseases in an open society from the mathematical point of view. An open society means a society which passengers enter to it or leave it. We will pay more attention to the societies which the entrance parameters and leaving parameters are time dependent. The model is an extension of the SIR models. Our approach to this model is an O.D.E. approach. We are also going to speak on the new mathematical methods of the edge group population dynamics to present a new research topic.

STOCHASTIC ANIMAL MOVEMENT MODELS WICH GENERATE ANGULAR DISTRIBUTIONS

William Reed

A number of stochastic movement models, leading to circular (angular) distributions are discussed. They can be divided into two categories: (i) models in which the state of the system is an angle (direction or orientation); and (ii) models in which the state of the system is a point in the plane. Various stopping rules are considered. Resulting angular distributions include the wrapped versions of the normal, stable, Laplace and normal-Laplace (NL) distributions; the von Mises and the projected (offset) normal distributions and a new skewed form of the von Mises distribution. The fit of some of the models to angular data will also be discussed.

ON THE SPATIO-TEMPORAL DISEASE DYNAMICS IN POPULATIONS WITH DISCRETE-TIME EPIDEMICS IN ONE AND TWO SPATIAL DIMENSIONS

Karen Rios-Soto[a], **Carlos Castillo-Chavez**[b]

The use of integrodifference equations in the study of the role of dispersal on populations with discrete generations has generated interesting mathematical problems and expanded our understanding of their spatio-temporal dynamics. Here, we use discrete-time epidemic models that can be reduced to a single map for the infectious class, $I_{t+1} = g(I_t)$, where g may or may not be monotone. We use new theoretical work, modeling, analysis and simulations to illustrate the role of g on disease dynamics in one and two spatial dimensions.

COUPLING AN AGE-STRUCTURED POPULATION MODEL FOR FISH DYNAMICS WITH HYDRODYNAMIC INDIVIDUAL-BASED MODEL FOR LARVAL DISPERSAL WITHIN A BAYESIAN STATE-SPACE MODELING FRAMEWORK

Sebastien Rochette, **Olivier Le Pape**, **Martin Huret, Etienne Rivot**

Bayesian state-space models coupled with sequential Monte Carlo methods provide a general framework for quantitative ecological modeling, statistical inference and predictions. They provide a support for embedding high dimensional models for biological processes within statistical inferences accounting for multiple sources of variability and uncertainty. Diagnostics and predictions in the Bayesian framework are derived on a probability based rationale and are easily embedded in a formal decision analysis. In particular, they provide a framework for building stage-structured population models with multiple forms of uncertainty and variability, while accounting for various sources of field observations.

In this work, the value of the framework is considered in the field of fisheries sciences. Assessment of harvested marine fish populations is usually conducted using statistical catch-at-age analysis that uses age or stage-structured population models fitted on commercial catch data, using scientific surveys and other estimates of age or stage-structured densities as additional information. Spatially explicit models are key issues for improving our understanding of population dynamics and enhancing fisheries management. For instance, many exploited fish populations directly depend upon spatially restricted habitats such as nurseries for juveniles. Although the annual catches are usually reported with a (relatively coarse) spatial information, spatially explicit models are seldom considered because of the lack of reliable information about the spatial distribution of young stages, from eggs on spawning grounds, larvae during their drift to juveniles on their nursery grounds.

In the present approach, we used the Bayesian framework to build a spatially explicit population model for the common sole (Solea solea) at the scale of the Eastern English Channel. A state-space model is built for an age-structured population model. The demographic model was coupled with an hydrodynamical model of the Eastern Channel (MARS 3D) that provides prior information about the dispersion of sole larvae, from spawning grounds overseas to the coastal nurseries. Previous work to map nurseries habitats was then used to account for the habitat capacity in coastal nurseries [1]. The whole model was fitted to 20 years series of spatially explicit catches and abundances indices. The fitted model was then used to explore the possible trajectories of the sole population during the last 30 years under different scenarios with regards to habitat degradation of nurseries and fishing pressure.

PATTERN FORMATION, LONG-TERM TRANSIENTS, AND TURING-HOPF BIFURCATION IN A SPACE- AND TIME-DISCRETE PREDATOR-PREY SYSTEM

L.A.D. Rodrigues[a], **D.C. Mistro**[a] and **S. Pretovskii**[b]

[a]Department of Mathematics
Federal University of Santa Maria
Santa Maria, Brazil
ladiazrodrigues@gmail.com

[b]Department of Mathematics
University of Leicester
Leicester, UK
sp237@le.ac.uk

Populations living in their natural habitat are often heterogeneously distributed in space, with high population density alternate with areas where the individuals are virtually absent. Sometimes, this heterogeneity can be linked to the properties of the environment. However, in many cases, there is no external force linked to the spatial pattern. Understanding this phenomenon is an issue of crucial importance since ecological patchiness has implications for population dynamics and ecosystem functioning (Petrovskii et al., 2004). Over the last three decades there has been a significant progress in understanding pattern formation in predator-prey and resource-consumer systems (Segel and Jackson, 1972; Petrovskii and Malchow, 1999). While scenarios and mechanisms of patter formation in spatially-continous system are studied relatively well (Malchow et al., 2008; Neubert et al., 1995), scenarios of pattern formation in discrete system are not not well studied yet.
In this work, we consider a predator-prey system that is discrete both in space and time, and is described by a Coupled Map Lattices. We assume that the prey growth is affected by a weak Allee effect and predator dynamics present intraspecific competition. We focus on the system's properties in the vicinity of a discrete Turing-Hopf bifurcation. We perform extensively numerical simulations and reveal a rich variety of spatiotemporal patterns. In particular, we found spike, labyrinthine and checkboard-like patterns. For some parameters values, the system's dynamics is dominated by long-term transients, so that the asymptotical stationary pattern arise as a sudden transition between two different patters. Finally, in the discrete system studied, we observed that pattern formation is possible even outside the Turing instability parameters domain.

References

[1] H. Malchow, S. Petrovskii and E. Venturino, "Spatiotemporal Patterns in Ecology and Epidemiology: Theory, Models and Simulations." Chapman & Hall / CRC Press, London, 2008.

[2] M. G. Neubert, M. Kot and M.A. Lewis, "Dispersal and pattern formation in a discrete-time predator-prey model", *Theor. Pop. Biol.*, v. 48, p. 7-43, 1995.

[3] S. Petrovskii, and H. Malchow, "A minima model of pattern formation in a prey-predator system", *Math. Comp. Modelling*, v. 29, p. 49-63, 1999.

[4] S. Petrovskii, B. -L. Li, and H. Malchow, "Transition to spatiotemporal chaos can resolve the paradox of enrichment", *Ecological Complexity*, v. 1(1), p. 37-47, 2004.

[5] L. A. Segel, and J. L. Jackson,"Dissipative structure: An explanation and an ecological example", *J. Theor. Biol.*, v. 37, p. 545-559, 1972.

STOCHASTIC SENSITIVITY FUNCTION TECHNIQUE FOR ANALYSIS OF THE NOISE-INDUCED PHENOMENA IN THE POPULATION DYNAMICS

L. Ryashko and **I. Bashkirtseva**

Department of Mathematics
Ural State University
Lenina, 51, 620083, Ekaterinburg, Russia
lev.ryashko@usu.ru

Stochastic fluctuations in nonlinear models of population dynamics can cause various unexpected phenomena such as stochastic resonance, noise-induced transitions, noise-induced order and chaos. The sensitivity analysis of corresponding attractors is a key for the investigation of these phenomena. Now a research interest is moved from a finding of qualitative variety for the noise-induced phenomena to a quantitative analysis of underlying probabilistic mechanisms. Kolmogorov-Fokker-Planck equation is a basic mathematical tool for the theoretical analysis of the stochastic dynamics. However, a direct using of this equation is very difficult even for simplest situations.
We suggest a new technique for the approximation and computational analysis of the forced attractors (equilibria and limit cycles) of the population dynamics models. This technique is based on the stochastic sensitivity function (SSF) [1-3]. This function describes a dispersion of random trajectories in the stochastic attractor and adequately reflects probabilistic peculiarities of dynamics. We apply this technique to the analysis of stochastic cycles of 2D and 3D-models of population dynamics. SSF analysis allows us to find examples of high sensitive 2D-cycles where very small disturbances transfer a system to the chaotic regime. For 3D-models, we suggest a universal theoretical approach to the quantitative and geometrical analysis of stochastic cycles in a period-doubling bifurcations zone. An effectiveness of this approach is demonstrated for the study of noise-induced transitions and stochastic bifurcations.

This work was partially supported by grants RFBR09-01-00026, 09-08-00048, 10-01-96022ural, Federal Education Agency 2.1.1/2571, Federal Target Program N 02.740.11.0202.

References

[1] I. A. Bashkirtseva, and L. B. Ryashko, "Stochastic sensitivity of 3D-cycles", *Mathematics and Computers in Simulation*, v. 66, p. 55-67, 2004.
[2] I. A. Bashkirtseva, L. V. Karpenko, and L. B. Ryashko, "Analysis of attractors for stochastically forced "predator-prey" model", *Applied Nonlinear Dynamics*, v. 17, No. 2, p. 37-53, 2009.
[3] I. Bashkirtseva, L. Ryashko, and P. Stikhin, "Noise-induced backward bifurcations of stochastic 3D-cycles", *Fluctuation and Noise Letters*, v. 9, No. 1, p. 89-106, 2010.

NON-DIFFUSIVE MIGRATION MODELS BASED ON HALDANE PRINCIPLE

Michael Sadovsky

Standard approach to model a spatially distributed community is to based on the implementation of partially differential equations of "reaction-diffusion" type. This approach faces a serious problem: to be feasible, one must suppose that the beings under consideration transfer themselves in space randomly and aimlessly. This assumption is absolutely inapplicable to the greatest majority of real biological situations. Alternatively, a migration must yield some adaptive value, providing a being with the improvement of its welfare. More exactly, the adaptive value of migration means that a migration must run in the way to increase the net reproduction $k(\rho(\overrightarrow{r}), \overrightarrow{r})$, with respect to transfer cost; here $\overrightarrow{r}$ is a space point, and ρ is the (local) density of a population (maybe, depending on $\overrightarrow{r}$ explicitly) [1].

A set of models has been developed implementing this principle into the practice of mathematical model of dynamics of biological communities. Firstly, a model of the development of clusters in (originally) homogeneous in space population resulted from Alle's effect is implemented [1, 3]. The second model describes the dynamics of a population inhabiting two stations (box model) so that the transfer from station to station (migration, in proper sense) increases an average net reproduction, over the stations. This model implies that the beings are globally informed: they know the environmental conditions (expressed in equation parameters), the density of each subpopulation, and the transfer cost. This is the basic model.

The third model describes the dynamics of *prey-predator* community that inhabits two stations; both species are supposed to be globally informed [2]. Also, this model takes into account the reflexive strategies of spatial distribution; a comparative study of regular and reflexive strategies is provided.

The situation of local information access is provided by the fourth model. This model resembles the basic one, while the beings under consideration know nothing towards the environmental conditions and subpopulation density in the station of immigration. It yields a threshold pattern for the migration rule: the emigration starts, as soon as the abundance exceeds some critical figure. It should be noticed, that the model with targeted migration of locally informed being exhibits the reciprocal migrant fluxes. Similar model of locally informed beings describing the dynamics of two-species community of *prey-predator* type was implemented. The effect of information access level on the dynamics of spatially distributed communities with targeted migration has been studied.

Finally, the model of a population inhabiting an areal divided into a number of stations was implemented. A space (habitation area) is presented as a lattice, or a set of stations; obviously, there is an arbitrariness in the connectivity graph choice. We have implemented two versions of the model; the former implies a full connectivity of the graph, and the latter is a lattice model. The problem of a share of an emigration flux is the key one here. Two versions of the rule have been implemented: a majorization rule implies that the flux immigrate into a station with better living conditions (expressed in the parts of net reproduction); equality rule implies that the flux is shared equally among all possible stations of immigration.

Further explorations in the non-diffusive modelling of spatially distributed communities implementing the adaptive mechanisms of migration are discussed.

References

[1] A. Gorban, "Selection Theorem for Systems with Inheritance", *Math. Model. Nat. Phenom*, v. 2, # 4, p. 1-45, 2007. [2] M. Sadovsky, "*Prey-predator* model with purposeful space migration of individuals", *J. Gen. Biol. (Russia)*, v. 63, # 3, p. 239-245, 2001. [3] A. Gorban, M. Sadovsky, "Optimization models of spatial distribution: Alle's effect", *J. Gen. Biol. (Russia)*, v. 50, # 1, p. 66-72, 1989.

Effect of community size distribution on R_0 in a multi-community system with common infection place

Yasuhisa Saito[a] and **Hiromi Seno**[b]

[a]Department of Mathematics
Pusan National University
Pusan 609-735, Republic of Korea
saito.yasuhisa@gmail.com

[b]Department of Mathematical and Life Sciences
Graduate School of Science, Hiroshima University
Kagamiyama 1-3-1, Higashi-hiroshima, 739-8526 Japan
seno@math.sci.hiroshima-u.ac.jp

Transportation, a common phenomenon in human society, is considered as one of main factors that could cause the outbreak of some diseases such as influenza and SARS. SARS's impact caused by transport-related infection is still fresh in our memory and the influenza pandemic has nowadays threatened human society.

The first step to model the transport-related infection is to use a disease transmission model based on the well-known patch models described by ordinary differential equations with a geographically divided population, where the transport-related infection has usually been modeled as an instantaneous event. However, during transportation, one shares specific time and place with other people, which would essentially make diseases spread. In this talk, to have modeling on such a situation, we propose an epidemical model of multi-community system with taking into account infection in a common (public) place, besides geographically divided compartments. We derive the basic reproduction ratio R_0 of the model and discuss the effect of community size distribution on R_0.

Key words multi-community system, common infection place, the basic reproduction ratio, community size distribution

UNIFORM PERSISTENCE IN DISCRETE AND CONTINUOUS NON-AUTONOMOUS DYNAMICAL SYSTEMS WITH AN APPLICATION TO AN EPIDEMIC MODEL OF AN AMPHIBIAN POPULATION

Paul Leonard Salceanu

Mathematics Department
University of Louisiana at Lafayette
Lafayette, LA 70504, U.S.A.
pls8855@louisiana.edu

Keywords: Uniform persistence, non-autonomous dynamical systems, disease persistence.

This is an extension of the work of Salceanu and Smith [1], where boundary attractors for autonomous, dissipative dynamical systems on the positive orthant of R^m, generated by maps, were characterized as *uniformly weak repellers*, in order to obtain conditions for uniform persistence of a subpopulation vector. Here we take an unified approach, for both discrete and continuous time non-autonomous systems. The main assumption is that a nontrivial compact positively invariant set M exists on a bounding hyperplane. We show that when this boundary set has certain repelling properties, uniform persistence for the complementary dynamics is obtained. When the system is periodic, and every solution in M is attracted (in forward time) to a periodic orbit, the repelling properties of M are expressed in terms of spectral radii. We apply these results to an SI (susceptible and infected) discrete-time model of an amphibian population with periodic coefficients [2], and obtain conditions for uniform persistence of the disease.

References

[1] P.L. Salceanu, H.L. Smith, "Lyapunov exponents and persistence in discrete dynamical systems", *Discrete Cont. Dyn-B.*, v. 12, p. 187-203, 2009.

[2] K.E. Emmert, L.J. Allen, "Population exctinction in deterministic and stochastic discrete-time epidemic models with periodic coefficients with applications to amphibian populations", *Nat. Resour. Model.*, v. 19, p. 177-164, 2006.

viability Analysis of a Multi-fishery Based on Continuous fishing Effort

C. Sanogo [a],**S. Ben Miled**[b] and **N. Raissi**[a]

[a]Department of Mathematics
Ibn Tofail University, Faculty of sciences
BP 133, Kenitra Morocco
csanogo@yahoo.com
n.raissi@lycos.com

[b]ENIT-LAMSIN and the Pasteur Institute of Tunis
BP 37, 1002 Tunis Belvedere, Tunisia
slimane@ipeit.rnu.tn

The goal of this work is to study the viability domain corresponding to fishing model population. The biological constraint ensures the stock durability and the economic constraint ensures a minimum income for fleets.
The classic theory of dynamic optimization does not include the possibility of several decision-makers. It is usually in the games theory that such situations arise. In management of fishing the hypothesis of only one decision-maker is not appropriated. The analysis of the model of C. Clark in [1] shows that the less efficient fleet ended up leaving the fishing. N.Raissi in [2] has considered a regulator which aimed to maintain the two fleets as a long as possible in the fishing. The analysis of these models led to optimal discontinuous fishing efforts which would present a difficulty in the application. In this work, we consider that the control is the variation of the effort of fishing and we suppose that the fish population is harvested by two fleets which a priori do not cooperate. We used the viability analysis to build domain of viability which allows both fleets to exploit the resource simultaneously and ensures a minimum income for fleets with stock durability.

References

[1] C.W. Clark , " Mathematical Bioeconomics : the Optimal Management of Renewable Resources.", *2nded : A Wiley-Interscience.* 1990.

[2] N. Raissi , " features of bioeconomics models for the optimal management of a fishery exploited by two fleets", *Natural resources modelling*, volume 14, number 2, 2001.

[3] C. Béné, L. Doyen, and D. Gabay, " A viability analysis for a bio-economic model", *Ecological Economics*, volume 36, 2001.

DYNAMICS OF AGE STRUCTURED POPULATIONS IN RANDOM ENVIRONMENTS

L. Sanz[a] and **J.A. Alonso**[b]

[a]Department of Mathematics
E.T.S.I.I, Universidad Politécnica de Madrid
José Gutierrez Abascal, 2, 28006, Madrid, Spain
lsanz@etsii.upm.es

[b]Department of Mathematics
E.T.S.I.I, Universidad Politécnica de Madrid
José Gutierrez Abascal, 2, 28006, Madrid, Spain
jalonso@etsii.upm.es

In this work we study some features of the behavior of Leslie type matrix models for age structured populations subjected to environmental stochasticity [1]. We think of a population described at time n by vector $\mathbf{X}_n = (x_n^1, ..., x_n^N)^T$ and living in an ambient in which there are s different environmental states. The vital rates corresponding to each one of these environments are given by the Leslie matrices $\mathbf{L}_\alpha \in \mathbb{R}^{N\times N}$, $\alpha = 1, ..., s$ in such a way that, for each α, $\mathbf{L}_\alpha$ contains the fertility and survival rates of the population in environment α. The environmental variation is characterized by a sequence of random variables τ_n, that we will consider to be an irreducible and aperiodic Markov chain, with state space $\{1, ..., s\}$ in such a way that τ_{n+1} describes for the environmental condition for the system between times n and $n+1$. Thus, the model reads

$$\mathbf{X}_{n+1} = \mathbf{L}_{\tau_{n+1}} \mathbf{X}_n \tag{15}$$

where $\mathbf{X}_0 \geq \mathbf{0}$ is a fixed (non random) non-zero vector. Moreover, we assume that the set of matrices of vital rates meets a certain technical condition (ergodic set).

The most important parameter concerning the behavior of (15) is the so called stochastic growth rate (s.g.r.) defined as $a := \lim_{n\to\infty} \log \|\mathbf{X}_n\| / n$, with probability one [2]. Therefore, $a > 0$ implies that every realization grows asymptotically with rate e^a, and $a < 0$ implies that the population goes extinct with probability one. Moreover, the vector of population structure converges to a limit distribution F which is independent of the initial conditions, and the total population is asymptotically lognormal, where the asymptotic distribution is characterized by a and a certain positive parameter σ^2. The difficulty with model (15) is that, even in very simple situations, it is not possible to calculate a, σ^2 or F and one must resort to numerical simulations. Moreover, it is not possible to ascertain whether F is absolutely continuous or singular [2].

In this work we use different approaches to give some insight into the dynamics of models of this kind. In the first place we deal with the scalar case ($N = 1$) giving exact results for a and σ^2 and necessary/sufficient conditions for the growth/extinction of the population. Then we proceed to the case $N = 2$, where we make use of perturbation techniques to estimate a, σ^2 and F. In particular, we obtain results that explain the behavior observed in numerical simulations and that suggest that the character of the limit distribution F is very sensitive to the parameters of the model.

References

[1] H. Caswell. Matrix Population Models (2^{nd} ed.), Sinauer Associates Inc., Sunderland, 2001.
[2] S. Tuljapurkar. Population Dynamics in Variable Environments, Springer-Verlag, 1990.

Modelling pathogen spread over genetically and spatially heterogeneous landscape

N. Sapoukhina[a] , **Ch. E. Durel**[b] and **B. Le Cam**[a]

[a] UMR077 Plant Pathology-PaVé
INRA Angers-Nantes
BP 60057, F-49071 Beaucouzé, France
[a]natalia.sapoukhina@angers.inra.fr
[a]Bruno.Lecam@angers.inra.fr

[b] UMR1259 Genetics and Horticulture-GenHort
INRA Angers-Nantes
BP 60057, F-49071 Beaucouzé, France
[b]Charles-Eric.Durel@angers.inra.fr

Abstract
Biological invasions originated by rapid evolutionary changes constitute a serious threat to ecosystems. A representative example is the breakdown of gene-for-gene plant resistance by a novel virulent pathogen. The emergence of a highly virulent pathogen after release of a plant carrying a new combination of resistance genes challenges to develop a generic modeling framework that accounts for evolutionary processes underlying pathogen invasions and spatial/genetic heterogeneity of the invaded crops.

We formulate a spatially realistic population-genetic model for ascertaining the synergetic effect between genetic and spatial composition of the host population on the pathogen spread reinforced by evolutionary processes [1]. The model is developed in the context of the reaction-diffusion models with integration of sexual and asexual reproduction for a pathogen population and multilocus gene-for-gene interactions. We show that spatial arrangement of host genotypes is crucial to the efficacy of host genetic diversification. In particular, the reductive effect of multigenic resistance on the pathogen density can be produced by a random patterning of monogenic resistances. Random patterns can reduce both density and genetic diversity of the pathogen population and delay invasion promoted by sexual recombination. By contrast, patchy distributions diversify pathogen population and, hence, reduce the efficacy of resistance genes.

The proposed approach provides theoretical support for studying fast emergence and spread of novel pathogen genotypes carrying multiple virulence genes. It has a practical applicability to design innovative strategies for the most appropriate deployment of plant resistance genes.

Key words : Population-genetic model. Gene-for-gene resistance. Heterogeneity.

References

[1] N. Sapoukhina, Ch. E. Durel and B. Le Cam, "Spatial deployment of gene-for-gene resistance governs evolution and spread of pathogen populations", *Theoretical Ecology*, v. 2, p. 229-238, 2009.

A MATHEMATICAL MODEL OF POPULATION DYNAMICS WITH PREDATOR'S BEHAVIORAL CHANGE INDUCED BY PREY'S BATESIAN MIMICRY

H. Seno[a] and **T. Kohno**[b]

[a]Department of Mathematical and Life Sciences
Graduate School of Science, Hiroshima University
Kagamiyama 1-3-1, Higashi-hiroshima, 739-8526 Japan
seno@math.sci.hiroshima-u.ac.jp

[b]Department of Mathematics
Faculty of Science, Hiroshima University
Kagamiyama 1-3-1, Higashi-hiroshima, 739-8526 Japan

In this work, we analyze a mathematical model of the population dynamics among a mimic, corresponding model, and their predator populations. The predator changes its search-and-attack probability by forming and losing its search image. We analyze a mathematical model consisting of the daily population dynamics with ordinary differential equations, the seasonal population dynamics with difference equations, and the annual population dynamics with difference equations.

We construct a mathematical model consisting of the daily population dynamics with ordinary differential equations, the seasonal population dynamics with difference equations, and the annual population dynamics with difference equations. Each predation season is composed with the daily dynamics repeated day by day in T days. The predator population size is assumed to be kept constant, given by P, independently of the model and the mimic population sizes. This means such an assumption that the predator is a generalist and has some other preys to keep the stationary population size, so that it can survive and sustain its population even if the model and the mimic population go extinct.

The predator cannot distinguish the mimic from the model, so that each predator searches and attacks them with common probability. Once a predator predates a model individual, it comes to omit both the model and the mimic species from its diet menu, and then not to search nor attack them in the same day. If a predator predates a mimic individual, it comes to increase the search-and-attack probability for the model and the mimic. The frequency of predators with higher search-and-attack probability and that with zero search-and-attack probability decreases by a rate between the subsequent days, because of the predator's losing (i.e., forgetting) the search image.

The reproductions in model, mimic, and predator populations are assumed to occur between the subsequent predation seasons. In other words, there is no reproduction of model, mimic or predator within the predation season, so that the model and the mimic populations monotonically decrease due to the predation during the predation season.

Analyzing our model system, we can get the result such that the condition for the persistence of model population does not depend on the mimic population size, while the condition for the persistence of mimic population does depend on the predator's ability of the search image formation.

A MATHEMATICAL MODEL FOR A GROUP WAVE EMERGENCE WITH WAVING BEHAVIOR OF OCYPODID CRAB *Ilyoplax pusillus*

H. Seno[a] and **K. Tsutamura**[b]

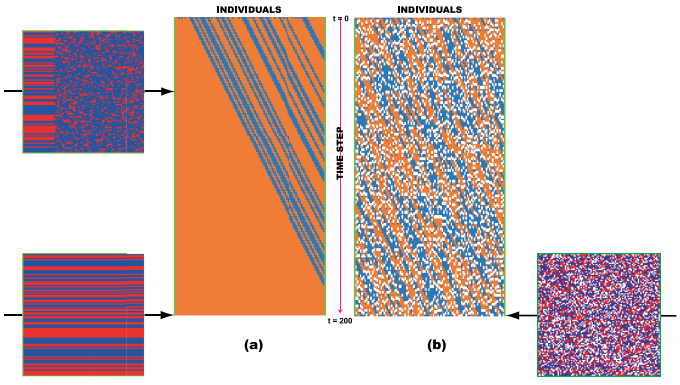
[a]Department of Mathematical and Life Sciences
Graduate School of Science, Hiroshima University
Kagamiyama 1-3-1, Higashi-hiroshima, 739-8526 Japan
seno@math.sci.hiroshima-u.ac.jp

[b]Department of Mathematics
Faculty of Science, Hiroshima University
Kagamiyama 1-3-1, Higashi-hiroshima, 739-8526 Japan

Ocypodid crab *Ilyoplax pusillus* inhabits intertidal sandy-mud flats along the coast. Males show their chelipeds' "waving" behavior in the breeding season. Waving is regarded as an aggressive display against other males and an attractive display against females. *Ilyoplax pusillus* has been known from its globally quasi-synchronous waving pattern, that is, a spatial group wave emerged by an interaction between wavings of different males in space. As for such a group wave of *Ilyoplax pusillus,* no study has yet been conducted. In the present study, we try to get some theoretical insights about the mechanism of group wave's emergence, making use of a mathematical model with a cellular automaton.

In our model, each individual is located on the lattice point in the 2-dimensional square lattice space. In the initial condition, we give a "direction" and a state of waving to each individual: the chelipeds are raised or not. Each individual does not move. No individual exists at the boundary of the lattice space. One effective neighbor individual is chosen from the Moore type neighborhood, depending on the "direction" of each individual. When the effective neighbor's chelipeds are raised, the individual tends to synchronize its own waving, going to raise its own chelipeds simultaneously. With no effective neighbor, the waving is a perfectly periodic oscillation. Each individual has the "scooping" behavior, too. The individual stops the waving during scooping.

From numerical calculations of our model, the group wave does not emerge if any individual never performs scooping or if each individual perform scooping and changing its "direction" at random at each time step. The group wave emerges only when each individual performs scooping with a biased distribution of the "direction". From our results, we give a conjecture that some appropriate breaks of the interaction of waving due to scooping with a biased "direction" of individuals would be necessary for the group wave emergence in case of *Ilyoplax pusillus*.



Numerical calculations of our cellular automaton model. The direction of every individual is fixed to the left. (a) With no scooping; (b) With scooping. The temporal variation of the waving state is plotted for the individuals of the 50 th row in the 2-dimensional lattice space.

EVOLUTION OF DISPERSAL IN AMERICAN PIKA METAPOPULATIONS

Anne Seppänen[a], **Kalle Parvinen**[a] and **John Nagy**[b]

[a]Department of Mathematics
University of Turku
Department of Mathematics, FI-20014 Turku, Finland
anne.seppanen@utu.fi

[b]Department of Life Sciences
Scottsdale Community College
Scottsdale, AZ 85256-2626, USA

The motivation for this work is driven by a desire to understand the evolution of dispersal in populations of American pikas (*Ochotona princeps*) and especially how global warming will affect their survival. American pikas are small, diurnal herbivores, whose physiology limits their range to areas with cold climates, primarily on mountains near or above treeline. Precisely these regions are expected to experience the greatest impact of global climate change (GCC). Therefore, intuition suggests that pikas are exposed to great risk from GCC. However, field data are ambiguous at best. How GCC is affecting pika populations, and if this species will be able to adapt to what changes are occurring are still open questions.

We derive a mechanistic dispersal model in semi-discrete time, meaning continuous within-phases dynamics (e.g. emigration, immigration, chasing territory) connected with discrete between-phases dynamics (e.g. winter survival, breeding). Our metapopulation model contains infinitely many patches with different local population sizes. Local catastrophes occur with a rate which can depend on the local population size.

We derive (a proxy for) the invasion fitness with which we are able to investigate the adaptive dynamics of dispersal in this model. We have considered GCC as a change in model parameters, e.g. catastrophes, dispersal cost, mortality and survival rate. In this presentation we will explore how these potential effects of GCC will affect the evolutionarily stable dispersal strategies.

Keywords: dispersal, adaptive dynamics, american pika

Stabilization by the Oxygen Control in the Wastewater Treatment Model

M. Serhani[a], **H. Labbardi**[b] and **N. Raissi**[c]

[a]Department of Economy
FSJES, University My Ismail, Meknes, Morocco
mserhani@hotmail.com

[b]Department of Biology
FST, University Hassan II-Mohammedia, Morocco
h_labbardi@yahoo.fr

[b] EIMA Team, Department of Mathematics
FS, University Ibn Tofail, Kenitra, Morocco
n.raissi@mailcity.com

In this work we study a model of wastewater treatment. The model describes an activated sludge process in which the substrate is removed by bacterial population. the phenomena considered are: The reaction kinetics in the aerator linked to microbial growth, The substrate degradation, the recycle of the biomass from the settler and the dissolved oxygen consumption.
The mass balance of the various constituents gives the following set of equations:

$$(S) \quad \begin{cases} \dot{s} = -k_s\mu(s,c)x - (1+r)Ds + Ds_{in} \\ \dot{x} = \mu(s,c)x - (1+r)Dx + rDx_r \\ \dot{x}_r = \nu(1+r)Dx - \nu(w+r)Dx_r \\ \dot{c} = -k_c\mu(s,c)x - D(1+r)c + Dc_{in} + k_a(c_s - s); \end{cases}$$

with

$$s(0) = s_0 \ ; \ x(0) = x_0 \ ; \ x_r(0) = x_{r0} \ ; \ c(0) = c_0.$$

Where s, x, x_r and c are the states variables representing the substrate, the biomass, the recycled biomass and the dissolved oxygen concentrations, respectively. D is the dilution rate, r is the recycle rate and w is the waste rate. s_{in} and c_{in} corresponds to the substrate and dissolved oxygen concentrations in the feed stream. k_s and k_c refer to the yield coefficient of the growth of biomass on substrate and dissolved oxygen respectively. k_a is the oxygen mass transfer coefficient and c_s is the saturation constant. $\mu(s,c)$ is the specific growth function of bacteria.
Due to fluctuations of D and s_{in}, the substrate (pollutant) s fluctuates and hence exceeds the tolerated level. Our goal, is to use the online measurements of the dissolved oxygen c to build a feedback control in term of c, in order to keep the substrate below a suitable level s_d fixed by the environmental considerations. On the other hand, in the case where the substrate concentration in the feed stream and the coefficient of oxygen are not well known, we use the asymptotic observers to have an accurate idea of the variables x, x_r and s. By the way, we build a feedback control using those observers to stabilize the substrate s below the level s_d.

References

[1] M. Serhani, N. Raissi and P. Cartigny , Robust Feedback Control Design for a Nonlinear Wastewater Treatment Model. J. of Math. Model. Nat. Phenom. Vol. 4, No. 5, pp. 128-143, 2009.

[2] M. Serhani, J.L. Gouze and N. Raissi, Dynamical study and robustness for a nonlinear wastewater treatment model. Proceeding book "Systems Theory : Modeling, Analysis and Control, FES2009", Eds A. EL Jai, L. Afifi and E. Zerrik, PUP, ISBN 978-2-35412-043-6, pp. 571-578.

ERGODICITY IN PRACTICE

R. Service

Department of Mathematics and Statistics
University of Helsinki
PO box 68, 00014 University of Helsinki
robert.service@helsinki.fi

Ergodicity was originally formulated as an heuristic concept in statistical mechanics in the nineteenth century. Around 1930 the concept of ergodicity was defined rigorously in its modern form in connection with the discovery of the ergodic theorems of von Neumann (mean ergodic theorem) and Birkhoff (pointwise ergodic theorem). However, the concept is still a source of confusion in the applied mathematics literature and in the field of mathematical biology in particular. The intention of the talk is to present a review of the most important mathematical concepts and results in ergodic theory from an applied point of view, while drawing attention to how muddled or incorrect reasoning based on ergodicity is found even in peer reviewed journals.

VEGETATION PATTERNS IN SEMI-ARID ENVIRONMENTS

J. Sherratt

In many semi-desert environments, rainfall is insufficient to support the uniform vegetation that occurs in temperate regions. Rather, vegetation is self-organised into spatial patterns; on slopes, these have the characteristic form of stripes running parallel to the contours, separated by regions of bare ground. I will describe a partial differential equation model for the formation of these stripes. Mathematically, the striped patterns are periodic travelling waves, and I will discuss bifurcation analysis of the travelling wave equations, which reveals the existence of a family of patterns, with a range of wavelengths. In particular, this enables a specific prediction of the critical level of rainfall below which patterns cannot be sustained, so that full-blown desert forms; this is an issue of major environmental significance at the present time. I will also show that for many ecological parameters, the model predicts a range of stable patterns, raising the possibility of hysteresis in properties such as wavelength, as the annual rainfall varies. Finally, I will combine the various results to derive a simple formula that predicts the wavelength that will develop as a semi-desert forms via a decrease in annual rainfall. I will end with a discussion of the ecological implications of the work, and a summary of the key open mathematical problems.

MODELLING ADHESION IN CELL POPULATIONS AND ITS ROLE IN CANCER INVASION

J. Sherratt, **K. Painter** and **N. Armstrong**

Adhesion of cells to one another and their environment is an important regulator of many biological processes, but has proved difficult to incorporate into continuum mathematical models.
I will describe a new approach to the mathematical modelling of adhesion in cell populations, based on an integro-partial differential equation for cell density, in which the integral represents the sensing by cells of their local environment.
This enables an effective representation of cell-cell adhesion, as well as random cell movement, and cell proliferation. I will show how this modelling approach can be applied to cancer growth. In this context, the model is capable of supporting both benign (non-invasive) and invasive growth, according to the relative strengths of cell-cell and cell-matrix adhesion. I will go on to describe the use of the model to investigate the criticality of matrix heterogeneity in shaping invasion, making the testable prediction that highly heterogeneous extracellular matrix can result in a f́ingering ó́f the tumour front, which is a hallmark of invasive cancers

OPTIMIZING METAPOPULATION SUSTAINABILITY THROUGH A CHECKERBOARD STRATEGY

N. Shnerb and **Y. Ben-Zion**

The persistence of a spatially structured population is determined by the rate of dispersal among patches. If the local dynamic at the subpopulation level is extinction-prone, the system viability is maximal at intermediate connectivity, where recolonization is allowed, but full synchronization that allows global extinction is forbidden.

We developed and used an algorithm for agent-based simulations in order to study the persistence of a stochastic metapopulation. The effect of noise is shown to be dramatic, as the dynamics of the spatial population has nothing to do with the predictions of deterministic models like the logistic map, Ricker map and Nicholson-Bailey host-parasitoid equations [1].

To analyze the possibility of extinction, previous studies, based on deterministic models, were focused on the attractiveness (Lyapunov exponent) of stable solutions and the structure of their basin of attraction (dependence on initial population size). Our results suggest that these features are of secondary importance in the presence of feasible stochasticity. Instead, optimal sustainability is achieved when decoherence is maximal.
Accordingly, agent-based simulations of metapopulation of different sizes, dimensions and noise types, show that the system's lifetime peaked when it displays checkerboard spatial patterns. Our conclusions are supported by the results of recently published laboratory experiments. We show how one should manipulate the migration rates (e.g., by constructing corridors) between habitats to minimize the risk of extinction.

NOVEL EXPONENTS CONTROL THE QUASI-DETERMINISTIC LIMIT OF THE EXTINCTION TRANSITION

N. Shnerb and **D. Kessler**

The quasi-deterministic limit of the generic extinction transition is considered within the framework of standard epidemiological models. The susceptible-infected- susceptible (SIS) model is known to exhibit a transition from extinction to spreading, as the infectivity is increased, described by the directed percolation equivalence class. We find [1] that the distance from the transition point, and the prefactor controlling the divergence of the (perpendicular) correlation length, both scale with the local population size, N, with two novel universal exponents. Different exponents characterize the large N behavior of the susceptible-infected-recovered (SIR) model, which belongs to the dynamic percolation class. Extensive numerical studies in a range of systems lead to the conjecture that these characteristics are generic and may be used in order to classify the high density limit of any stochastic process on the edge of extinction.

SURNAMES AND SPECIES, GENERA AND GENOMES: THE ULTIMATE NEUTRAL MODEL EXPLAINS (ALMOST) EVERYTHING

N. Shnerb, **D. Kessler** and **Y. Marukva**

Deducing quality (e.g., fitness) from success (large abundance, long lifetime, high returns on investments) is a common feature of many eco, econo and bio-theories; in particular it sets the conceptual framework for the theory of evolution by means of natural selection. Huge differences in success among species, with an uneven, fat-tailed distribution of abundance, seems to support these claims. Many heretics, however, suggest neutral models in which the main factor beyond success is merely luck. The uneven statistics is attributed to the effect of multiplicative stochastic process (drift). We show that the Kummer statistic, emerges from the neutral birth-death- mutation process, fits marvelously many empirical distributions in complex systems (e.g., species within genera, species abundance ratio, surname frequencies, cluster statistics of trees in the tropical forest and much more).

While previous neutral theories have been focused on the power-law tail, Our method allows explains not only the (noisy) tail but also the typical "shoulder" at small families. Scale-free networks are subject to slightly different underlying process but the resulting statistics is identical with that obtained for BDM. We thus suggest the BDM distribution as a standard neutral model: effect of fitness and selection are to be identified by substantial deviations from it.

NOISE MAY SUPPRESS PERIODIC TRAVELLING WAVES IN OSCILLATORY POPULATION MODELS

M. Sieber, **H. Malchow** and **S. V. Petrovskii**

Ecological field data suggests, that some species show periodic changes in abundance not only over time, but also in a specific spatial direction. The spatiotemporal pattern of population dynamics may then resemble a periodic travelling wave. Oscillatory reaction-diffusion equations have helped to identify possible scenarios, by which such periodic travelling waves may arise.

Now, any natural population can be expected to be subject to erratic fluctuations imposed by the environment. It is known, that such noise may induce shifts between different dynamical regimes in models of population dynamics and significantly alter the shape of spatiotemporal patterns found in these models.

This raises the question, whether periodic travelling waves as solutions to oscillatory reaction-diffusion systems are robust with respect to noise. It is shown, that small spatially homogeneous noise may suppress periodic travelling waves in generic oscillatory reaction-diffusion systems. Irregular spatiotemporal oscillations however appear to be more robust and persist under the same stochastic forcing. The implications of these findings for the modelling of travelling waves in species abundance are discussed.

A method for constructing deterministic individual-based epidemic models on arbitrary contact networks

Kieran Sharkey

A novel deterministic individual-based approach to modelling the spread of infectious diseases across contact networks is discussed[1]. This provides a limited but viable deterministic alternative to stochastic simulation for modelling realistic heterogeneous systems. The methodology sits in the large divide between individual-based stochastic simulation and population-level deterministic models and enables some theoretical insights to be gained. In particular, we demonstrate that the correlations between populations in mean-field and pair-approximation models can be partly attributed to previously unidentified anomalous terms. These terms describe unbiological currents of infection through a contact network and enable us to gain a more detailed understanding of the networks in which the mean-field and pair-approximation models perform well.

References

[1] Sharkey, KJ: Deterministic epidemiological models at the individual level. J. Math. Biol. 57, 311-331. (2008).

MODELING APPROACH FOR THE PHYSIOLOGICAL TICK LIFE-CYCLE

Dania Sheaib[a], **Slimane Ben Miled**[b] and **Nabil Nassif**[c]

[a]Department of Mathematics
American University of Beirut
Beirut, Lebanon
dws02@aub.edu.lb

ENIT-LAMSIN & Institut Pasteur de Tunis
Université de Tunis el Manar
BP 37, 1002 Tunis, Tunisia
slimane.benmiled@pasteur.rns.tn

[c]Department of Mathematics
American University of Beirut
Beirut, Lebanon
nn12@aub.edu.lb

Tick-borne diseases (theleriosis, rickettsiosis, Lyme disease, Ehrlichiosis, relapsing fever, tick-borne encephalitis) are serious health problems affecting humans as well as domestic animals in many parts of the world. These infections are generally transmitted through a bite of an infected tick, and it appears that most of these infections are widely present in some wildlife species; hence, an understanding of tick population dynamics and its interaction with hosts is essential to understand and control such diseases.

In this presentation, we first intend to describe the different evolution stages associated with the tick life-cycle as well as the tick-host epidemiological interaction level. This allows us to identify and clarify all the physiological parameters affecting the development of the tick population. Thus, we are able to describe the tick life-cycle model formulated to study the effect of temperature and seasonality on the density of ticks. The model used here is a system of partial differential equations, (PDE). This model will be the foundation of a later epidemiological model which describes the tick-host interactions.

To obtain a numerical solution of the tick life-cycle model, Petrov-Galerkin approximations based on variational formulations are derived using Legendre polynomials. This leads to a system of ordinary differential equations which computations are carried in view of investigating and understanding the tick population dynamics. Numerical results will be presented to illustrate basic features of the mathematical model and its solution.

References

[1] W. P. Gardiner, G. Gettinby, and J. S. Gray,"Modes Based on Weather For The Development Phases of the Sheep Tick, Ixodes-Ricinus", *Veterinary Parasitology*, 9(1):75 – 86, 1981.
[2] S. Randolph, "Epidemiological uses of a population model for the tick Rhipicephalus appendiculatus", *Trop Med Int Health*, 4(9):$A34 - A42$, septembre 1999.
[3] S. E. Randolph, "Abiotic and biotic determinants of the seasonal dynamics of the tick Rhipicephalus appendiculatus in South Africa", *Medical and Veterinary Entomology*, 11(1):25 – 37, 1997.
[4] R. Rosà and A. Pugliese, "Effects of tick population dynamics and host densities on the persistence of tick-borne infections", *Mathematical Biosciences*, 208(1):216 – 240, 2007.

PARAMETERS ESTIMATION IN SMITH-MARTIN MODEL OF CELLS DIVISION DYNAMICS OF OT1 AND F5 T-CELLS

Andrey Shuvaev[a], **Daniel Commenges**[a], **Thea Hogan**[b,c], **Robin Callard**[b]
Ben Seddon[c], **Andrew Yates**[d], **Rodolphe Thiebaut**[a]

[a]INSERM U897
ISPED, Université Bordeaux 2, France
Andrey.Shuvaev@isped.u-bordeaux2.fr

[b]Institute of Child Health
University College London,UK

[c]National Institute of Medical research
Medical research Council, UK

[d]Albert Einstein Institute, New York, USA

We investigated the division dynamics of two types of immune T-cells (in transgenic mice having F5 and OT1 TCR receptors) in lymphopenic conditions. A modified Smith-Martin model of cell cycle was used [1, 2]. This model assumes two parts: A-state with stochastic duration (representing a G_0 period of the cycle) and B-phase with fixed duration (representing of G_1, M and S periods of the cycle). We estimated the following parameters: a transfer rate from A-state to B-phase λ, duration of B-phase Δ and time of trigerring to division T_0. We used four types of λ parameter: constant and dependend on time, number of divisions and total number of cells. Estimations were done using weighted sums of squared residuals routine (WSSR) and likelihood approach.
We had a best fit (according to the lower AIC criterion) with WSSR routine for both types of cells. Also, for both F5 and OT1 cells, a better fit was achieved when allowing λ vary over time. OT1 was shown divide more intensively than F5 (having higher transfer rate from A state to B and earlier trigerring to division).
Also, we explored a modification of the model allowing some of the cells to go from previous B-phase to the next B-phase without entering A-state.

References

[1] J. Smith, and L. Martin, "Do cells cycle?", *PNAS*, v. 70, p. 1263-1267, 1963.
[2] A. Yates, and M. Saini, A. Mathiot, B. Seddon, "Mathematical Modeling Reveals the Biological Program Regulating Lymphopenia-Induced Proliferation", *Journal of Immunology*, v. 1800, p. 1414-1422, 2008.

Exact epidemic models on graphs using graph-automorphism driven lumping

Péter L. Simon[a], **Michael Taylor**[b] and **Istvan Z. Kiss**[b]

[a]Institute of Mathematics
Eötvös Loránd University
Budapest, Hungary
simonp@cs.elte.hu

[b]Department of Mathematics
School of Mathematical and Physical Sciences, University of Sussex
Falmer, Brighton BN1 9RF, UK
mt264@sussex.ac.uk (Michael Taylor)
i.z.kiss@sussex.ac.uk (Istvan Z Kiss)

The dynamics of disease transmission strongly depends on the properties of the population contact network [1, 3]. Pair-approximation models and individual-based network simulation have been used extensively to model contact networks with non-trivial properties [2-6]. In this paper, using a continuous time Markov Chain [4, 7, 8], we start from the exact formulation of a simple epidemic model on an arbitrary contact network and rigorously derive and prove some known results that were previously mainly justified based on some biological hypotheses. The main result of the paper is the illustration of the link between graph automorphisms and the process of lumping whereby the number of equations in a system of linear differential equations can be significantly reduced. The main advantage of lumping is that the simplified lumped system is not an approximation of the original system but rather an exact version of this. For a special class of graphs, we show how the lumped system can be obtained by using graph automorphisms. Finally, we discuss the advantages and possible applications of exact epidemic models and lumping.

Keywords: network, epidemic, Markov Chain, lumping, graph automorphism.

References

[1] Diekmann, O. & Heesterbeek, J.A.P. 2000 Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. John Wiley & Sons Ltd, Chichester, UK.
[2] House, T., Davies, G., Danon, L. & Keeling, M.J. 2009 A Motif-Based Approach to Network Epidemics. Bull. of Math. Biol. 71, 1693 – 1706.
[3] Keeling, M.J. & Eames, K.T.D. 2005 Networks and epidemic models. J. R. Soc. Interface 2, 295 – 307.
[4] Keeling, M.J. & Ross, J.V. 2008 On methods for studying stochastic disease dynamics. J. R. Soc. Interface 5, 171 – 181.
[5] Kiss, I.Z., Green, D.M. & Kao, R.R. 2006b. The effect of network heterogeneity and multiple routes of transmission on final epidemic size. Math. Biosci. 203, 124 – 136.
[6] Kiss, I.Z., Green, D.M. & Kao, R.R. 2008 The effect of network mixing patterns on epidemic dynamics and the efficacy of disease contact tracing. J. R. Soc. Interface 5, 791 – 799.
[7] Kurtz, T.G. 1970 Solutions of ordinary differential equations as limits of pure jump Markov processes. J. Appl. Prob. 7, 49 – 58.
[8] Kurtz, T.G. 1971 Limit theorems for sequences of jump Markov processes approximating ordinary differential processes. J. Appl. Prob. 8, 344 – 356.

ANALYSIS OF STABILITY AND HOPF BIFURCATION IN LESLIE-GOWER MODEL WITH DISCRETE DELAYS

A. Singh[a] and **S. Gakkhar**[b]

[a]Department of Mathematics, Indian Institute of Technology Roorkee,
Roorkee (U.K.) 247667, India
anurajiitr@gmail.com

[b]Department of Mathematics, Indian Institute of Technology Roorkee,
Roorkee (U.K.) 247667, India
sungkfma@iitr.ernet.in

The dynamical analysis of two-dimensional Leslie-Gower model with double delays is analyzed here. It is shown that the time delay can destabilize the system and periodic solution is obtained after demonstration of Hopf bifurcation. By using normal form theory and center manifold argument, direction and stability of periodic solutions are determined. Numerical simulations substantiate the analytical results including appearance of chaos.

References

[1] K. Lai and J. Wei, "Stability and Hopf bifurcation analysis of a prey-predator system with two delays", Chaos, Solitons and Fractlas, v. 42, p. 2606-2613, 2009.
[2] B. Hassard, D. Kazarino and Y. Wan, "Theory and applications of Hopf bifurcation", Cambridge University Press, Cambridge, 1981.
[3] Y. Song and J. Wei, "Bifurcation analysis for Chen's system with delayed feedback and its application to control of chaos", Chaos, Solitons and Fractlas, v. 22, p. 75-91, 2004

MODELLING OF THE POPULATION DYNAMICS TAKING INTO ACCOUNT CHILD CARE

V. Skakauskas[a] and **S. Repsys**[b]

[a]Faculty of Mathematics and Informatics
Vilnius University
Naugarduko 24, LT-03225 Vilnius, Lithuania
vladas.skakauskas@maf.vu.lt

[b]Faculty of Mathematics and Informatics
Vilnius University
Naugarduko 24, LT-03225 Vilnius, Lithuania
sarunas.repsys1@mif.vu.lt

Modelling of the age-structured population dynamics taking into account child care and nonlinear diffusion will be considered. The model consists of a system of integro-PDEs subject to conditions of integral type. Number of equations depends on the biologically possible maximal number of offspring of the same generation produced be an individual. Numerical results will be demonstrated.

Key words: age-structured populations, child care.

References

[1] V. Skakauskas, “A one-sex population dynamics model with discrete set of offsprings and child care", *Nonlinear Analysis: Modelling and Control*, v. 13(4), p. 525-552, 2008.

EFFECT OF DIFFUSION FLUXES ON PROTECTIVE PROPERTIES OF ANTIBODIES

A. Skvortsov, P. Gray

HPP Division,
Defence Science and Technology Organisation
506 Lorimer Street, Fishermans Bend, Vic 3207, Australia
alex.skvortosv@dsto.defence.gov.au
peter.gray@dsto.defence.gov.au

The aim of this study was to evaluate the influence of diffusion fluxes on the potential protective effect of antibodies. To simulate this we propose a simple, kinetic based model that rigourously captures dynamics of receptor-toxin-antibody interactions, the toxin-receptor complex formation and toxin internalisation (for details see [1])

$$\frac{d[RT]}{dt} = k_1[R][T]_s - k_{-1}[RT] - k_3[RT], \quad (1)$$

$$\frac{d[AT]}{dt} = k_2[A][T] - k_{-2}[AT], \quad (2)$$

$$\frac{d[T_i]}{dt} = k_3[RT], \quad (3)$$

where $[R]$, $[T]$ and $[A]$ are the concentrations of receptor, toxin and antibody respectively, $[T]_s$ is the concentration of the toxin on the surface of a cell, $[T_i]$ is the concentration of the internalised toxin and $[RT]$ and $[AT]$ are the concentrations of the receptor-toxin and antibody-toxin complexes. The k_i values are the rate constants. This system should be supplemented with conservation laws for $[R], [T]$ and $[A]$.
The distribution of toxin around a cell is driven by the standard diffusion equation

$$\frac{\partial[T]}{\partial t} = D\triangle[T], \quad (4)$$

where D is the diffusion coefficient of toxin in the extracellular space. The boundary condition for (4) is discontinuity of the diffusion flux $D\frac{\partial[T]}{\partial r} = \frac{d[RT]}{dt}$ at the surface of a "spherical" cell (i.e. at $r = a$).
The system (1) - (4) describes the interplay of toxin depletion (caused by formation of the toxin-receptor complexes) and its diffusion influx from extracellular space towards the cell surface. To simplify analysis of the system we applied the method of the "diffusion equivalent capacitance" proposed in [2] and derived the "simplified diffusion equation"

$$\frac{d[RT]}{dt} = CS([T] - [T]_s), \quad (5)$$

where $S = 4\pi a^2$ is the surface area of the cell and the expression for the equivalent capacitance $C \equiv C(t)$ is given in [2].
The simplified system (1) - (2), (5) is much easier to solve numerically and to treat analytically (since it becomes a system of ODE). With the diffusion fluxes taken into account we found that antibody may significantly affect the toxin-receptor binding and toxin internalisation, so the main results and conclusions of the "well-mixed" approximation [1] are still valid.

References

[1] A. Skvortsov and P. Gray. "Modeling and simulation of receptor-toxin-antibody interaction", *MODSIM 2010: Proc. 18th World IMACS / MODSIM Congress*, pp.185-191, 2009.
[2] P.R. Naira and M. A. Alam. Performance limits of nanobiosensors. *Applied Physics Letters*, **88**, pp. 233120-03, 2006.

PREDICTION AN EPIDEMIC OUTBREAK BASED ON DATA STREAMS FROM SYNDROMIC SURVEILLANCE

A. Skvortsov[a], **B. Ristic**[b] , **C. Woodruff** [a]

[a]HPP Division, [b]ISR Division
Defence Science and Technology Organisation
506 Lorimer Street, Fishermans Bend, Vic 3207, Australia
alex.skvortosv@dsto.defence.gov.au
branko.ristic@dsto.defence.gov.au
chris.woodruff@dsto.defence.gov.au

Early detection and prediction of an epidemic outbreak (malicious or natural) is of crucial importance for a timely medical response (quarantine, vaccination, etc). A conventional approach to this problem often involves large scale computer simulations (agent-based) which are resources demanding (see [1] and refs). This paper proposes an alternative framework formulated in the context of stochastic nonlinear filtering. This framework does not require any advanced computer facility to implement and to run.
The framework is based on the stochastic SIR epidemiological with inhomogeneous mixing [1],

$$\frac{ds}{dt} = -\alpha\, i\, s^{\nu} + \sigma_{\alpha}\xi, \tag{1}$$

$$\frac{di}{dt} = \alpha\, i\, s^{\nu} - \beta i - \sigma_{\alpha}\xi + \sigma_{\beta}\zeta, \tag{2}$$

and $r = 1 - s - i$. Here ξ, ζ are two uncorrelated white Gaussian noise processes (both with zero mean and unity variance); parameter ν, describes a mixing inhomogeneity (with a particular value $\nu = 1$ corresponding to the uniform mixing scenario), $\sigma_{\alpha}, \sigma_{\beta}$ are amplitudes of noise associated with inhomogeneities in contract rate α and recovery rate β. In general, ν can be treated as another fitting parameter of the model.
For syndromic observations we employ a power-law model. This means that each syndrome (number of visits to pharmacies, calls to "hot lines", sales of a particular product, visits of particular web sites, etc) is a power-law function of the number of infected people. The observation model is then

$$z^{j} = b^{j}\, i^{\theta_j} + \sigma_j \eta^{j}, \tag{3}$$

where z^j is the observable syndrome index $j = 1, \ldots, N_z$; $b^j, \sigma_j, \theta_j = const$ (different for different syndromes); η^j is zero-mean, unit variance white Gaussian noise, associated with syndrome j; η^j is assumed to be uncorrelated to other syndromes and noises ξ and ζ.
The problem of early detection and prediction of an epidemic is formulated in the framework of sequential Bayesian estimation for stochastic dynamic systems (1, 2). We adopt the state-space approach and for the purpose of estimation define the state vector and its initial (prior) PDF. Finally using the time-discretised dynamic model, we estimate sequentially the state vector, as the measurements become available. The estimated state vector is eventually predicted for future times using the dynamic model (1, 2). For further details see [2,3].

References

[1] P. Stroud *et al.* "Semi-empirical power-law scaling of new infection rate to model epidemic dynamics with inhomogeneous mixing", *Math. Biosciences*, v. 203, p. 301-318, 2006.
[2] B. Ristic *et al.* "Predicting the progress and the peak of an epidemic", *ICASSP 2009: Proceedings IEEE Inter. Conf. Acoustics, Speech and Signal Processing*, p. 513-516, 2009.
[3] A. Skvortsov *et al.* "Predicting an epidemic based on syndromic surveillance", *FUSION 2010: Proceedings 13th International Conference on Information Fusion*, 2010 (submitted).

MATHEMATICAL MODELLING OF INFECTIOUS DISEASES CONSIDERING AGE-STRUCTURED CONTACT RATE

Cintia Soares[a] and **Hyun Mo Yang**[b]

Department of Applied Mathematics
IMECC/UNICAMP
Rua Srgio Buarque de Holanda, 651 CEP: 13083-859; Campinas, S.P., Brasil
[a]cintiasoares@ime.unicamp.br
[b]hyunyang@ime.unicamp.br

The main goal of mathematical epidemiology of directly transmitted diseases is the development of a deterministic model to evaluate the disease control through vaccination. In order to be useful, the models need to describe the medical phenomenon with a certain degree of realism. This can be done by adjusting the model to the field data. Then, predictions can be done with a relative accuracy when epidemiologic control interventions are introduced, such as vaccination.

We propose a model that describes directly transmitted diseases, such as measles and rubella. The heterogeneity in age is considered in the model, by the reason that these infections usually occur during the childhood. The control of these diseases is performed through vaccination campaign which is incorporated in the model.

Based on the model elaborated in [2], we yield a function $Y(a)$ which represents the infectious individuals, at age a, at steady state. So we can define the operator T:

$$TY(a) = N^* \frac{\sigma}{(\gamma-\sigma)} e^{-\mu a} \int_0^L Y(x) \int_0^a \beta(\zeta, x) e^{-\int_0^L Y(y) \int_0^\zeta \beta(x,y)dxdy - \int_0^\zeta \nu(t)dt} \left(e^{\sigma(\zeta-a)} - e^{\gamma(\zeta-a)} \right) d\zeta dx, \tag{16}$$

where $\beta(a, a')$ is the contact rate among susceptible and infectious individuals, at age a and a' respectively; ν is the vaccination rate; μ is the natural mortality rate; N^* is the natality rate; σ^{-1} and γ^{-1} are, respectively, the mean periods of incubation and recovery.

We defined the reproduction number as the spectral radius of Frchet derivative of the operator T at 0. The existence and the stability of the fixed point of the operator T have been studied. The model was tested using the seroprevalence data found in the literature [3]. In addition it will be tested with Mexico incidence data [1], and the results will be presented in this conference.

Keywords: Epidemiology, Mathematical Models, Age-structure, Population dynamics, Vaccination

References

[1] J.L. Daz-Ortega, C.D. Meneses-Reyes, and M. Palacios-Martnez, “Incidencia y Patrones de Transmisin de Rubeola en Mxico", *Salud Pblica de Mxico*, v. 49, p. 337-344, 2007.

[2] H.M. Yang, “Directly Transmitted Infections Modeling Considering Age-structured Contact Rate", *Math. Comput. Modelling* v. 29, p. 39-48, 1999.

[3] R.S. Azevedo Neto, A.S.B Silveira, D.J. Nokes, H.M. Yang, S.D. Passos, M.R.A. Cardoso, and E. Massad, “ Rubella Seroepidemiology in a Non-immunized Population of So Paulo State, Brazil", *Epidemiology and Infection* , v. 113, p. 161-173, 1994.

STABILITY AND HOPF BIFURCATION FOR A VIRAL INFECTION MODEL WITH NON-LYTIC IMMUNE RESPONSE

Xinyu Song[a] and **Xia Wang**[b]

[a]Department of Mathematics
Xinyang Normal University
Xinyang 464000, Henan, P.R. China
xysong88@163.com

[b]Department of Mathematics
Xinyang Normal University
Xinyang 464000, Henan, P.R. China
xywangxia@163.com

Mathematical modelling has been proven to be valuable in understanding the dynamics of HIV infection [1-4]. By direct application of models to data obtained from experiments in which antiretroviral drugs were given to perturb the dynamical state of infection in HIV-1 infected patients, minimal estimates of the death rate of productively infected cells, the rate of viral clearance and the viral production rate have been obtained [1-3]. Those models gave so accurate depiction of the virus load that they are almost consistent with the actual data. The research of mathematical models is very helpful for the clinical treatment. Especially, the models of combination therapy provide very important meaning for the cure of HIV.

A class of more general viral infection model with non-lytic immune response is proposed based on some important biological meanings. By means of Lyapunov functions, the global properties of model are obtained [5]. For delayed non-lytic immune response, the sufficient criteria for local and global asymptotic stabilities of the viral free equilibrium are given. And the stability and Hopf bifurcation of the infected equilibrium have been studied. The effects of the birth rate of susceptible T cells and the efficacy of the non-lytic component on the stabilities of the positive equilibrium E are also studied by numerical simulations [6].

References

[1] A. Neumann, N. Lam, H. Dahari, D. Gretch, T. Wiley, T. Layden, A. Perelson, Hepatitis C viral dynamics in vivo and antiviral efficacy of the interferon-α therapy, Science, v. 282, p. 103-107, 1998.
[2] A. Perelson, P. Essunger, Y. Cao, M. Vesanen, A. Hurley, K. Saksela, M. Markowitz, D. Ho, Decay characteristics of HIV-1-infected compartments during combination therapy, Nature, v. 387, p. 188, 1997.
[3] A. Perelson, P. Nelson, Mathematical analysis of HIV-1 dynamics in vivo, SIAM Rev. v. 41(1), p. 3-44, 1999.
[4] JE Mittler, B. Sulzer, AU Neumann, AS Perelson, Influence of delayed viral production on viral dynamics in HIV-1 infected patients, Math. Biosci. v. 152, p. 143-163, 1998.
[5] X. Wang and X.Y. Song, Global properties of model of immune effector responses to viral infections, Advances in Complex Systems, v. 10(4), p. 495-503, 2007.
[6] X.Y. Song, S.L. Wang and X.Y. Zhou, Stability and Hopf bifurcation for a viral infection model with delayed non-lytic immune response, Journal of Applied Mathematics and Computing, In press.

Savanna-Fire Model: Combined effects of tree-tree establishment competition and spatially explicit fire on the spatial pattern of trees in savannas

Flora S. Bacelar[a], **Justin M. Calabrese**[b], **Volker Grimm**[b] and **Emílio Hernández-García**[a]

[a]IFISC(CSIC-UIB) Instituto de Física Interdisciplinar y Sistemas Complejos
Campus Universitat Illes Balears, E-07122 Palma de Mallorca, Spain
florabacelar@ifisc.uib-csic.es

[b]Dept. of Ecological Modelling
Helmholtz Centre for Environmental Research (UFZ)
Leipzig, Germany
justin.calabrese@ufz.de

Savannas are characterized by robust coexistence of a discontinuous tree layer superimposed on a continuous grass layer. Savannas occur across a wide range of climatic, edaphic, and ecological conditions covering approximately one fifth of the earth's land area. In some countries these grass- dominated ecosystems are a principal biotic resource playing important roles in both the configuration of natural landscapes and in local economies. Identifying the mechanisms that facilitate tree-grass coexistence in savannas has remained a persistent challenge in ecology and is known as the "savanna problem", [1].

Several studies have suggested that adult trees can protect vulnerable juveniles from fire, thus increasing their chances of survival. Exactly how such protection works has not been intensively studied. However, given the frequent occurrence of fires in many savannas, it seems likely that the protection effect may be one of the most common forms of positive facilitation among savanna trees. Alternatively, many studies have documented the importance of competition among trees in diverse savannas. In a previous paper, Calabrese et al., [3], studied the interaction between competition and fire in a highly simplified savanna model. They showed that these two forces interact non-linearly with sometimes surprising consequences for tree population density and spatial pattern. However, because Calabrese et al., [3], treated fire in a non-spatially explicit manner, they did not study the protection effect, and thus could not fully tease apart how these contrasting of local interactions function in combination.

In the present work, a model is proposed to combine the previous savanna model, [3], with the Drossel-Schwabl forest fire model [4], therefore representing fire in a spatially explicit manner. We use the model to explore how the pattern of fire spread, coupled with an explicit, fire-vulnerable tree life stage and the negative and positive effects of adult trees on juvenile trees affects tree density and spatial pattern in savannas. Tree density depended strongly on both fire frequency and fire front patterns. Furthermore, the relationship between the post-fire grass recovery rate and the tree growth rate was a key factor in mediating tree-grass coexistence and its robustness. Different spatial patterns can be found depending on the parameters of the system: regular, random, and clumped. The last one, in contrast with the previous model, appears in two different forms, namely open and closed clusters, due to contrasts between the local interactions (protection and competition). When changing recovery rate or sparkling frequency, transitions from exponential cluster-size to power-law behavior can be found in the tree canopy distributions.

References

[1] Sarmiento, G. The ecology of neotropical savannas. Harvard University Press, Cambridge, MA, 1984.

[2] Michael E. Hochberg, Jean Claude Menaut, and Jacques Gignoux. The influences of tree biology and fire in the spatial structure of the West African savannah. *Journal of Ecology*, 82:217–226, 1994.

[3] Justin M. Calabrese, Federico Vázquez, Cristóbal López, Maxi San Miguel, and Volker Grimm. The interactive effects of tree-tree establishment competition and fire on savanna structure and dynamics. *the american naturalist*, 175(3):45-65, 2010.

[4] B. Drossel and F. Schwabl. Self-organized critical forest-fire model. *Physical Review Letters*, 69(11):1629–1632, 1992.

A COMPUTATIONAL MODEL FOR THE MATURATION OF FOCAL ADHESIONS AND STRESS FIBRES

Application to cell motility on a discrete adhesive environment

A. Stéphanou[a,b], **C. Franco**[a] and **T. Tzvetkova-Chevolleau**[b]

[a]Lab. TIMC-IMAG, UMR CNRS 5525
In3S - Faculté de Médecine de Grenoble
38706 La Tronche cedex, France
Angelique.Stephanou@imag.fr

[b]LTM, UMR CNRS 5129
c/o CEA Grenoble, 17 rue des Martyrs
38054 Grenoble cedex 9, France

We propose a computational model to investigate the coupling between cell's adhesions and actin fibres and how this coupling affects cell shape and stability. To accomplish that, we take into account the successive stages of adhesion maturation from adhesion precursors to focal complexes and ultimately to focal adhesions, as well as the actin fibres evolution from growing filaments, to bundles and finally contractile stress fibres.

We use substrates with discrete patterns of adhesive patches. The inter-patches distance can be modulated in order to control the location of the adhesions and the resulting fibres architecture. We then investigate the emergence of stable cell morphologies as a function of the inter-patches distance, for two different cell phenotypes generated from the model. Force generated by the stress fibres on the focal adhesions and specifically the influence of the cell contractility are considered.

Our results suggest that adhesion lifetime and fibre growing rate are key parameters in the emergence of stable cell morphologies and limiting factors for the magnitude of the mean tension force from the fibres on the focal adhesions.

Keywords: motility, stress fibres, focal adhesions, micropatterned substrates, integrative modelling.

References

[1] C. Franco C, T. Tzvetkova-Chevolleau and A. Stéphanou. "On the influence of discrete adhesive patterns for cell shape and motility: a computational approach", *Mathematical Modelling of Natural Phenomena, Cell Migration*, v. 5(1), p. 56-83, 2010.

FOOD QUALITY IN PRODUCER-GRAZER MODELS

D. Stiefs[a,b], **G.A.K. van Voorn**[c], **B.W. Kooi**[d], **U. Feudel**[e] and **T. Gross**[a]

[a]Max Planck Institute for the Physics of Complex Systems
Nöthnitzer Str. 38, 01187 Dresden, Germany
[b]stiefs@pks.mpg.de

[c]Wageningen University and Research Centre, Wageningen, The Netherlands

[d]Theoretical Biology, Vrije Universiteit, Amsterdam, The Netherlands

[e]ICBM, Carl von Ossietzky Universität, Oldenburg, Germany

Keywords: food quality, variable efficiency, stoichiometry, generalized model, paradox of enrichment.

We use the approach of generalized modeling [1] to investigate the effects of stoichiometric constraints on producer-grazer systems. The stability of steady states can be analyzed by using a normalization technique to plot 3-dimensional bifurcation diagrams [2]. Because we do not specify the functional form of the processes in the generalized model our results hold for a whole class of stoichiometric producer-grazer systems.

To understand the differences and commonalities between specific stoichiometric models we map the specific bifurcation diagrams into the generalized parameter space. On the one hand, these combined bifurcation diagrams show how the generic results of the generalized analysis are represented in the specific model. On the other hand, it becomes clear that some model features like the sequence of bifurcations observed during enrichment scenarios can be tied to specific modeling assumptions and are hence not structurally stable. In conclusion, we believe that our study paves the way for the unification of insights in stoichiometric population dynamics.

References

[1] T. Gross, and U. Feudel, "Generalized models as a universal approach to nonlinear dynamical systems", *Physical Review E*, v. 73, Article Number 016205, 2006.
[2] D. Stiefs, T. Gross, R. Steuer, and U. Feudel, "Computation and Visualization of Bifurcation Surfaces", *International Journal of Bifurcation and Chaos*, v. 18, p. 2191-2206, 2008.

THE INFLUENCE OF DYNAMIC NOISE ON SYSTEMS WITH CO-EXISTING ATTRACTORS, INCLUDING DETERMINISTICALLY CHAOTIC, IN APPLICATION TO EPIDEMIOLOGY AND ECOLOGY

Nico Stollenwerk and **Maíra Aguiar**

Centro de Matemática e Aplicações Fundamentais da Universidade de Lisboa,
Avenida Prof. Gama Pinto 2,
1649-003 Lisboa, Portugal,
maira@ptmat.fc.ul.pt and nico@ptmat.fc.ul.pt

In epidemiological and ecological systems one frequently encounters in biologically relevant parameter regions dynamics with co-existing attractors, including deterministically chaotic attractors. Boundary crises often lead to breakdown of chaotic attractors and result in deterministic ODE models in periodic solutions. However the transients and noisy realizations still show complex behaviour. Such scenarios can be detected by investigation of co-existences of attractors before the crisis in the deterministic model, then analysed deeper in stochastic versions, either stochastic ODEs or master equations. Applications reach from epidemiological models for childhood diseases, dengue fever and influenza to ecological predator prey models, with consequences for parameter estimation from empirical data time series.

References

[1] Drepper, F.R., Engbert, R., & Stollenwerk, N. (1994) Nonlinear time series analysis of empirical population dynamics, *Ecological Modelling* **75**/**76**, 171–181.

[2] Aguiar, M., Kooi, B., & Stollenwerk, N. (2008) Epidemiology of dengue fever: A model with temporary cross-immunity and possible secondary infection shows bifurcations and chaotic behaviour in wide parameter regions, *Math. Model. Nat. Phenom.* **3**, 48–70.

[3] Aguiar, M., Stollenwerk, N., & Kooi, B. (2009) Torus bifurcations, isolas and chaotic attractors in a simple dengue fever model with ADE and temporary cross immunity, *Intern. Journal of Computer Mathematics* **86**, 1867–77.

[3] S. van Noort, N. Stollenwerk and L. Stone, "Analytic likelihood function for data analysis in the starting phase of an influenza outbreak", *Proceedings of 9th Conference on Computational and Mathematical Methods in Science and Engineering, CMMSE 2009*, ISBN 978-84-612-9727-6, edited by Jesus Vigo Aguiar *et al.*, Salamanca, 2009, pp. 1072–1080.

GENERALISTS ENEMIES CAN STOP THE SPREAD OF INVASIVE SPECIES

C. Suppo[a], **C. Cosner**[b], **E. McCauley**[c] and **J. Casas**[a]

[a] IRBI UMR CNRS 6035
Faculté des Sciences, Université de Tours, 37200 Tours, France
christelle.suppo@univ-tours.fr, jerome.casas@univ-tours.fr

[b] Department of Mathematics
University of Miami, P. O. Box 249085, Coral Gables, FL 33124-4250, USA
gcc@math.miami.edu

[c] National Center for Ecological Analysis and Synthesis
735 State Street, Suite 300, Santa Barbara, CA 93101, USA
mccauley@nceas.ucsb.edu

Keywords: PDE, invasion process, biological control, host-parasitoid system, generalist parasitoid.

Abstract: Biological invasions are a major contemporary problem and the solutions are very few and terribly expensive. Biological control of invading insects with natural enemies is one of the most promising possibilities, but the focus has been invariably on specialised enemies used for inundating biological control, in order to avoid spill over in the ecological community. By contrast, little work has been done of the potential of generalist predators to slow down, stop or reverse the spread of invasive pests in the context of conservation biological control. We use the following system of partial differential equations for a host and generalist parasitoid:

$$\begin{cases} u_t = D_u u_{xx} + r_1 u\left(1 - \dfrac{u}{K_1}\right) - v\dfrac{Eu}{1+Ehu}, \\ v_t = D_v v_{xx} + \gamma v\dfrac{Eu}{1+Ehu} + r_2 v\left(1 - \dfrac{v}{K_2}\right) \end{cases}$$

Where u (x,t) = hosts density at point x and at time t; v (x,t) = parasitoids density at point x and at time t; D_u, D_v = diffusion rate of hosts (respectively parasitoids); r_1, r_2 = growth rate of hosts (respectively parasitoids); K_1, K_2 = carrying capacity of hosts (respectively parasitoids) in absence of focal hosts, E = encounter time, h = harvesting time, γ = conversion efficiency.

Numerical simulations and a travelling wave approach are used for analysing the spread of the hosts in space. We observe that the inclusion of space in the models increases the range of parameters for which a control of the pest (i.e. a reversal of the advancing waves) is possible. Indeed, generalists, being already present in the entire domain before the arrival of the pest thanks alternative hosts, might control it. They may provoke the disappearance of the small pockets of hosts at the very front of the wave. In a first part of the presentation, we study the relationship between the dispersal ability of the host and the parasitoid. We estimate the maximal parasitoids diffusion rate necessary to control host's invasion in function of the host's diffusion rate. We find that the efficiency of parasitoids decreases when they move much faster than their host. Parasitoids must be concentrated at the location of hosts to control them. The speed of the travelling wave is studied in the second part of the study. We analyse the travelling wave connecting the non trivial equilibrium (u*,v*), which corresponds to the invasion of host, and the semi trivial equilibrium (0,v*) which corresponds to the control of host. We find the existence's condition of such a travelling wave in function of the model's parameters. The spatial dynamics of generalist predators and their prey are strikingly different from the ones obtained for specialists. Given the overriding majority of generalists in insect communities, our work will also lay the foundations for these interactions in space.

COMMUNITY-BASED MEASURES FOR MITIGATING THE 2009 H1N1 PANDEMIC IN CHINA

S.Y. Tang[a] **Y.N. Xiao**[b] **Y.P. Yang**[b] **Y.C. Zhou**[b] **J.H. Wu**[c] **Z.E. Ma**[b]

[a] College of Mathematics and Information Science, Shaanxi Normal University, Xi'an, 710062, P.R.China
sytang@snnu.edu.cn & sanyitang219@hotmail.com
[b] Department of Applied Mathematics, Xi'an Jiaotong University, Xi'an, 710049, P.R.China
[c] Centre for Disease Modeling, York University Toronto, Ontario M3J 1P3, Canada

Since the emergence of influenza A/H1N1 pandemic virus in March-April 2009, stringent interventions were implemented to prevent importation of infected cases and decelerate the disease spread in mainland China. Surveillance data show large scale outbreak did not happen until the early September when the fall semester started. We sought to investigate the effectiveness of the interventions (quarantine, isolation, hygiene precaution and *Fengxiao-a tightly monitored measure of movement restriction, was also put in place to proscribe college and university students, faculty, and staff members to leave their campuses, and to disallow on-campus visits while maintaining essential services and normal scientific activities*), try to inform decisions whether there interventions should be enforced and/or improved local control measures to manage the on-going outbreak, expected to be complicated by the substantial nationwide travel during the Spring Festival in the middle of February 2010. On the basis of spatially stratified compartmental model we addressed the impact of interventions including *Fengxiao* and travel reduction on pandemic mitigation during and following the Spring Festival holiday. Main results include

- Using data from initial laboratory-confirmed cases in the province of Shaanxi, we estimated the mean reproduction number as 1.68 (95\% CI 1.45-1.92) and other parameters, and analyzed their sensitivity.
- *Fengxiao*, if implemented early, can delay the epidemic peak significantly and obviously prevent the disease spread to the general population though it may cause more severe outbreak due to the aggregate outbreaks within the universities/college. Late implementation of *Fengxiao* has little impact to reduce the magnitude of the outbreak or delay the epidemic peak, behaves like not implementation of *Fengxiao*. Strengthening local control strategies (quarantine and hygiene precaution) is much more effective to mitigate outbreaks and inhibit the successive waves than implementing *Fengxiao*.
- In absence of transport-related transmission, reduction of control measures (quarantine or precaution) during Spring Festival holiday (Middle of February 2010) has more potential risk to cause a new wave than increasing dispersal or the size of susceptible population. Strengthening hygiene precaution is more effective in reducing attack rate than strengthening quarantine rate. Massive travel is inevitable during Spring Festival holiday, if without significant improvement of the transportation system it will likely increase the infection during the travel. It is necessary to consider transport-related transmission. Either strong mobility or high transport-related transmission rate will hardly reverse the overall declining trend, but both will result in a large new wave. So, travel precautions should not be relaxed unless strict measures of quarantine, isolation, and hygiene precaution practices are put in place.

OPTIMAL FORAGING IN LESLIE-GOWER PREDATOR-PREY MODELS WITH ALTERNATIVE FOOD

M. Teixeira-Alves[a,b], **L. Mailleret**[a] and **F. Grognard**[b]

[a]UR880 - URIH / INRA Sophia Antipolis
400 route des Chappes, 06903 Sophia Antipolis Cedex
{mteixeira,ludovic.mailleret}@sophia.inra.fr

[b]COMORE / INRIA Sophia Antipolis
2004 route des Lucioles, 06902 Sophia Antipolis Cedex
frederic.grognard@inria.fr

Keywords: predator-prey model, optimal foraging, switching, alternative food.

In generalized Lotka-Volterra prey-predator models, many studies have been lead to analyze the influence of an alternative food source for the predator, both when this source has a fixed density [1] or is an alternative dynamic prey ([2] and subsequent works). Here, we will lead such a study with fixed density preys for two Leslie-Gower like models, where the prey population determines the carrying capacity of the predator population [3]:

$$\begin{cases} \dot{N} = r_N N \left(1 - \dfrac{N}{K}\right) - q\lambda NP \\ \dot{P} = r_P P \left(1 - \dfrac{P}{q\alpha_1 N + (1-q)\alpha_2 A}\right) \end{cases} \tag{17}$$

$$\begin{cases} \dot{N} = r_N N - q\lambda \left(\dfrac{N}{1 + T_N N} P\right) \\ \dot{P} = r_P P \left(1 - \dfrac{P}{q\alpha_1 N + (1-q)\alpha_2 A}\right) \end{cases} \tag{18}$$

where N and P are the dynamic prey and predator population densities and A is a constant alternative food source. The optimally foraging predator population will have to make a choice by taking some $q \in [0,1]$ between foraging the dynamic prey ($q = 1$), the other one ($q = 0$) or to adopt an intermediate strategy, depending on the respective prey densities. Its objective will be to maximize its per capita growth rate $\frac{\dot{P}}{P}$. Predators adopt pure strategies except when $N = \frac{\alpha_2 A}{\alpha_1}$ (both food sources produce the same carrying capacity), where a Filippov definition of the solutions can give rise to sliding modes.
The behavior depends on whether the positive equilibrium with $q = 1$ is (i) stable or (ii) unstable or (iii) does not exist in models (17) and (18). Case (i) results in the maximization of the predator density at equilibrium. In case (ii), the alternative food source stabilizes the system which was unstable without it; it keeps the oscillations under control and results in a limit cycle that goes through a sliding mode. In case (iii) however, the other food source cannot prevent the populations to grow unbounded.
In conclusion, the predator makes the most of the presence of an alternative food source by obtaining a maximal value for its equilibrium from it (case (i)) or gaining some stability (case (ii)). The effect on the dynamic prey N is not as straightforward; in particular, we show that no apparent competition principle [4] needs to hold.

References

[1] V. Krivan, “Optimal foraging and predator-prey dynamics”, *Theor. Pop. Biol.*, v. 49, p. 265-290, 1996.
[2] M. Van Baalen, V. Krivan, P. Van Rijn, and M. Sabelis, ”Alternative food, switching predators, and the persistence of predator-prey systems”, *American Naturalist*, v. 157, p. 512-524, 2001.
[3] P.H. Leslie, “Some further notes on the use of matrices in population mathematics”, *Biometrika* , v. 35, p. 213-245, 1948.
[4] R.D. Holt, "Predation, apparent competition, and the structure of prey communities”, *Theor. Pop. Biol.*, v. 12, p. 197-229, 1977.

INVESTIGATING THE IMPACT OF ORGANIZATIONAL FACTORS ON THE NOSOCOMIAL RISK THROUGH AGENT-BASED MODELING

L. Temime[a], **P.Y. Boëlle**[b], **C. Brun-Buisson**[c], **L. Kardas**[a],**L. Opatowski**[c] and **D. Guillemot**[c]

[a]Chaire Hygiène & Sécurité,
Conservatoire national des Arts et Métiers,
292 rue St Martin - 75141 Paris Cedex 3
laura.temime@cnam.fr

[b]Inserm UMR-S707,
27 rue Chaligny - 75571 Paris Cedex 12
pierre-yves.boelle@u707.jussieu.fr

[c]Inserm U657,
146 rue Léo Saignat - 33076 Bordeaux Cedex
didier.guillemot@pasteur.fr

Over recent years, nosocomial pathogens have become widespread in hospitals worldwide. The underlying mechanisms driving their transmission involve a complex combination of host, pathogen and environmental effects. In particular, nosocomial outbreaks have been correlated with host activities and behavior, such as compliance with control measures (including hygiene practices) and frequency of bodily contacts. Hence, it is to be expected that organizational factors at the hospital level may play a major part in the control of nosocomial risk.

We have developed a spatially explicit agent-based modeling platform of pathogen transmission in a hospital ward, called NosoSim. Our model includes a wide array of organizational parameters, from health-care workers daily schedules and affectations to the geographical disposition of patient rooms.
In this presentation, we show how NosoSim can be used to investigate the effect of several of these organizational factors on the nosocomial risk. First, we assess the potential of health-care workers, based on their work profile in terms of patient affectations and daily schedules, to become superspreaders of a bacterial pathogen. Second, we study how shift work organization at the hospital level may impact the risk of pathogen transmission within the hospital.

Both presented studies suggest that organizational factors play a major part in the nosocomial risk. They also demonstrate that flexible models such as NosoSim may help investigate this impact and could in time become decisional tools to be used directly by clinicians or hygiene specialists inside hospitals.

MODELLING CD8 T-CELL IMMUNE RESPONSE

Emmanuelle Terry[a], **Fabien Crauste**[a] and **Olivier Gandrillon**[b]

[a]Institut Camille Jordan, UMR CNRS 5208
Universit Claude Bernard Lyon 1
21 avenue Claude Bernard, 69622 Villeurbanne Cedex, France
terry@math.univ-lyon1.fr, crauste@math.univ-lyon1.fr

[b]Centre de Gntique Molculaire et Cellulaire, UMR CNRS 5534
Universit Claude Bernard Lyon 1
16 rue Raphal Dubois, 69622 Villeurbanne Cedex, France
gandrillon@cgmc.univ-lyon1.fr

Keywords: immune response, CD8 T-cell, ordinary differential equations, delay equations

The primary CD8 T-cell response, due to a first encounter with a pathogen, happens in two phases: an expansion phase, with a fast increase of T-cell count, followed by a contraction phase. This contraction phase is followed by the generation of memory cells. These latter are specific of the antigen and will allow a faster and stronger response when encountering the antigen for the second time. Several works recently proposed models of the CD8 immune response [1,2,3]. Some of these works do not consider any regulation of the immune response [1,3], whereas others propose very detailed and complex models [2].

We will discuss two models of the primary response, in which nonlinearities account for molecular regulation of cell dynamics. The first one is based on ordinary differential equations, the second one on partial delay differential equations, and the delay takes into account the time cells take to differentiate from one state to the other one. We will discuss in particular the roles and relevance of feedback controls that could regulate the response. Finally, we will show some simulations we can get from the models and confront them to experimental data.

References

[1] Antia R., Ganusov V.V. and Ahmed R., "The role of models in understanding CD8+ T-cell memory", *Nature Reviews*, v.5, p.101-111, 2005.
[2] Kim P.S., Lee P.P. and Levy D., "Modeling regulation mechanisms in the immune system", *J. Theor. Biol.*, v.246, p.33-69, 2007.
[3] Rouzine I.M., Murali-Krishna K. and Ahmed R., "Generals die in friendly fire, or modeling immune response to HIV", *J. Computational and Appl. Math.*, v.184, p.258-274, 2005.

Analysis of a two-patch model for the dynamical transmission of tuberculosis

Jean Jules Tewa[a,1], **Samuel Bowong**[b,1], **Boulchard Mewoli**[c] and **Jurgen Kurths**[d]

[a,]Department of Mathematics and Physics
National Advanced School of Engineering
P.O. Box 8390 Yaounde, Cameroon
tewajules@gmail.com

[b]Department of Mathematics and Computer Science
Faculty of Science, University of Douala
P.O. Box 24157 Douala, Cameroon
sbowong@gmail.com

[c]Department of Mathematics
Faculty of Science, University of Yaounde I
PO Box 82 Yaounde, Cameroon
bmewoli@yahoo.fr

[d]Postdam Institute for Climate Impact Research (PIK)
Telegraphenberg A 31, 14412 Potsdam, Gemany
juergen.kurths@pik-potsdam.de

Abstract

This paper deals with the analysis of a two-patch model for the dynamical transmission of tuberculosis. We compute the basic reproduction ratio $\mathcal{R}_0$, the disease-free equilibrium and prove that when the basic reproduction ratio is less than unity, the disease-free equilibrium is globally asymptotically stable. Conditions for the existence of endemic equilibria (when the disease persists in the two sub-populations) are derived. Numerical results are provided to illustrate theoretical results. We find that i) the increased progression rate from latent to active TB for individuals in one sub-population may play a significant role in the rising prevalence of TB in the other sub-population; and ii) the increased migration rates from the first sub-population to the second sub-population have increased the prevalence level of TB in the second sub-population while decreasing TB prevalence in the first sub-population.

Key Words: Epidemiological models, Tuberculosis, Patches, Stability, Lyapunov functions.

1 Introduction

Infectious diseases have had a profound effect on human populations, including their evolution and cultural development. Despite significant advances in medical science, infectious diseases continue to impact human populations in many parts of the world. Tuberculosis (abbreviated as TB for tubercle bacillus) is a common

[1]UMI 209 IRD/UPMC UMMISCO, Bondy, Projet MASAIE INRIA Grand Est, France and Projet Grimcape, LIRIMA, Cameroun

POPULATION DYNAMICS OF CELLULAR ADHESION SITES: THE SELF-ORGANISATION OF PODOSOMES

Ph.Tracqui

Lab. TIMC-IMAG, Equipe DynaCell
CNRS
In3S, La Tronche Cedex France
Philippe.Tracqui@imag.fr

Podosomes are self-organized and dynamic actin-containing structures that move around inside the cytoplasm within minutes. Furthermore, they either exist as individual podosomes or aggregate to form rings of podosomes, known as rosettes [1]. In this latter case, this large-scale pattern emerges from the lower-scale organisation of podosomes actin cores.

Furthermore, as podosome rings changed size and shape, tractions stresses underneath the podosomes were exerted onto the substrate [2], suggesting that interactions of mechanical tension and actin dynamics monitor mechanochemical instabilities [3] that are crucial for regulating podosomes self-organization in living cells.

In order to decipher the roots of this self-organizing process, we propose a theoretical model that couples the dynamics of F-actin polymerization, including nucleation and branching mediated by the Arp2/3 protein complex, to the contractile properties of the cell cytoskeleton and transmission of forces to the substrate [4]. Correlations of spatio-temporal modifications of F-actin arrays with the control of cell adhesion by mechanical forces are analyzed. Special attention is paid to the regulation processes monitored by the stiffness of extracellular substrates, with resulting pattern formations discussed in the light of experimental results.

References

[1] O. Collin, P. Tracqui, A. Stephanou, Y. Usson, J. Clement-Lacroix and E. Planus, "Spatio-temporal dynamics of actin-rich adhesion microdomains: influence of substrate flexibility", *J. Cell Sci.* 119(9), p. 1914-1925, 2006.
[2] O. Collin, S. Na, F. Chowdhury, M. Hong, M.E. Shin, F. Wand and N. Wang, "Self-organized podosomes are dynamic mechanosensors", *Curr. Biol.* 18(17), p. 1288-1294, 2008.
[3] P. Tracqui,"Mechanical instabilities as a central issue for *in silico* analysis of cell dynamics", *Proceedings IEEE* 94(4), p. 710-724, 2006.
[4] T. Boudou, J. Ohayon, C. Picart, R. Pettigrew and P. Tracqui. "Nonlinear elastic properties of polyacrylamide gels: Implications for quantification of cellular forces", *Biorheology* 46, p. 191-205, 2009.

Insensitizing controls for a linear population dynamics model

O. Traore [a] and **J. Velin**[b]

[a]Department of Mathematics
University of Ouagadougou
03 BP 7021, Burkina Faso
traore.oumar@univ-ouaga.bf

[b]Department of Mathematics
University of Antilles-Guyane
Campus Fouillole
97159, Pointe-à-Pitre, Guadeloupe (France)
jean.velin@univ-ag.fr

We consider a system which is a linear population dynamics model simulating the distribution of individuals during the time, depending on age and spatial position. This distribution is defined by the state, solution of the system. In the situation studied here, the born term is not local and describes a recruitment process of this population. Furthermore, the initial distribution of the newborn is not known exactly contrary to the standard case presented by several authors in the literature. So, being given the norm of the solution of the system, as in [2], [3], we look for a control which makes it locally insensible to the lack of information on the distribution at the initial time. We show that the insensitivity condition can be interpreted as the local null controllability (at time and in age) for the state of an auxiliary problem. The existence result of the control function as solution of the local null controllability, derives from an explicit construction combining a variational method (see [4], [5]) and some Carleman inequalities adapted to the studied model (see [1], [3], [6]).

References

1. B. Ainseba, "Exact and Approximate Controllability of the Age and Space Population Structured Problem", *J. Math. Anal.App*, v. 275, p. 562-574, 2002.
2. O. Bodart and C. Fabre, "Controls insensitizing the norm of the solution of a semilinear heat equation", *Journal of Mathematical Analysis and Applications* v. 195, n°3, p. 658-683, 1995.
3. L. de Teresa, "Controls insensitizing the norm of the solution of a semilinear heat equation in unbounded domains", *ESAIM: COCV,* v. 2, p. 125-149, 1997.
4. O. Nakoulima, "Contrôlabilité à zèro avec contraintes sur le contrôle", *C.R. Acad. Sci. Paris* Ser. I, v. 339, p. 405-410, 2004.
5. O. Nakoulima, "Optimal control for distributed systems subject to null-controllability. Application to discriminating sentinels", *ESAIM: COCV,* v. 13, n° 4, p. 623-638, 2007.
6. O. Traoré,, "Null Controllability for a nonlinear population dynamics model", *Int. J. Math. Mathematical Sc.* v. 2006 ID 49279, p. 1-20, 2006.

Sur la modèle de Kirshner-Panetta en immunothérapie du cancer

Alexei Tsygvintsev

Unité de mathématiques pures et appliquées
École normale supérieure de Lyon, CNRS UMR 5669

Mots clés: immunothérapie de cancer, modélisation

En 1998 D. Kirschner a proposé les équations différentielles pour étudier l'interaction entre les cellules cancéreuses et le le système immunitaire. Nous faisons l'analyse qualitative de ces équations. Nous montrons également comment l'introduction du délai permet de diminuer et parfois éradiquer les effets secondaires de IL-2 thérapie. C'est un travaille commun avec Denis Kirschner and Sandip Banerjee.

References

[1] D. Kirschner and J. Panetta, Modeling immunotherapy of the tumor-immune interaction, J. Math Biol, 37 (1998), 235-252.
[2] Kirschner, D and Tsygvintsev, A. On the global dynamics of a model for tumor immunotherapy. Journal of Mathematical Biosciences and Engineering Volume 6(3) 2009. pp 573-583.
[3] Banerjee, S, Immunotherapy with Interleukin-2: A Study Based on Mathematical Modeling, International Journal of Applied Mathematics and Computer Science, Volume 18 , Issue 3 (Number 3 / September 2008)

MONTEBELLO:

A METAPOPULATION BASED MODEL OF CARCINOGENESIS

David Tuck[a] , **Willard Miranker**[b,] **and Jose Costa**[a]

[a]Department of Pathlogy
Yale University School of Medicine,
New Haven, Connecticut, USA
david.tuck@yale.edu
jose.costa@yale.edu

[b]Department of Computer Science
Yale University
New Haven, CT, USA
willard.miranker@cs.yale.edu

The dynamics of the lengthy process by which tumors arise from normal tissues is not well understood [1]. Within spatially constrained tissues at high risk for cancer, a diverse set of clones (metapopulations) appears, competes, dies out, survives or proliferates, and is subjected to exogenous and endogenous perturbations leading to complex dynamics [2,3]. We have refined a computational model of carcinogenesis, the Montebello Model (MM), that uses evolutionary and metapopulation dynamics to explore the interaction of mutation, gene function, disturbance and chance in the genesis of tumors. The operation of the model shows how different agents acting on tissues can cause tumors by modifying the dynamics among metapopulations of cells in the context of continuous physiological cell proliferation and death. Disturbance, with no or little effect on mutation, effectively drives tumor formation. The simulations demonstrate that changes in the distribution of mutational load in the tissue as a whole can foreshadow the emergence of a tumor and thus provide a measure of risk. They also show that modifying the regimen of disturbance prevents the emergence of tumors. The efficacy of these interventions can be monitored by following the patterns of genetic alterations through time. The model and simulations provide new insights into how mutation rates and disturbance (e.g. inflammation [4]) interact in the causation of cancer and illustrate how variational biometrics can measure risk and perhaps serve as surrogate end points for preventive intervention.

References

[1] Beerenwinkel, N. et al. Genetic progression and the waiting time to cancer. PLoS Comput Biol 3, e225(2007).
[2] González-García, I., Solé, R.V. & Costa, J. Metapopulation dynamics and spatial heterogeneity in cancer. Proc Natl Acad Sci U S A 99, 13085-9(2002).
[3] Tarafa, G. et al. Mutational load distribution analysis yields metrics reflecting genetic instability during pancreatic carcinogenesis. Proc Natl Acad Sci U S A 105, 4306-11(2008).
[4] Mantovani, A. et al. Cancer-related inflammation. Nature 454, 436-444(2008).

TWO-HOST, TWO-VECTOR R_0 FOR BLUETONGUE

J. Turner[a], **R.G. Bowers**[b] and **M. Baylis**[a]

[a]Department of Animal & Population Health
School of Veterinary Science, University of Liverpool
Leahurst, Neston, South Wirral, CH64 7TE, UK
j.turner@liverpool.ac.uk
baylism@liverpool.ac.uk

[b]Department of Mathematical Sciences
University of Liverpool
Mathematical Sciences Building, Liverpool, L69 7ZL, UK
sx04@liverpool.ac.uk

Bluetongue is a vector-borne infection of ruminants that can have serious economic consequences. The infection is endemic in parts of Africa [1] and the southern Mediterranean [2], where it is commonly transmitted by the biting midge *Culicoides imicola*. In recent years, the range of this vector has expanded, seemingly as a result of climate change [2], and the disease has occurred in new areas (e.g. Northern Europe) revealing that other previously unsuspected vector species are capable of transmitting this infection. Although *C. imicola* is still generally considered to be the main vector, other vector species (e.g. *C. obsoletus* in Europe and *C. bolitinos* in South Africa) are now being recognized as important. In particular, it has been suggested [1] that *C. bolitinos* could be the primary vector, even in areas where it occurs in much lower numbers than *C. imicola*, because it has a significantly higher vector competence. Although the presence of two competent vectors does not necessarily mean that they are both transmitting the infection during the same outbreak, there is evidence that this was the case in Sicily [3].

Vector species differ not only in their ability to transmit infection, but also in their susceptibility to environmental conditions. These factors can be included in the formula for the basic reproduction ratio (R_0), which indicates the infection's ability to spread. Here we present an expression for R_0 for a two-host, two-vector system and illustrate it using data relating to *C. imicola* and *C. bolitinos* [1], by considering differences in vector abundance and vector competence between the vector species. We also present several approximations based on a previously published two-host, one-vector formula [4] and compare and contrast the results.

References

[1] J.T. Paweska, G.J. Venter and P.S. Mellor, "Vector competence of South African *Culicoides* species for bluetongue virus serotype 1 (BTV-1) with special reference to the effect of temperature on the rate of virus replication in *C. imicola* and *C. bolitinos*", Med. Vet. Entomol., v. 16, p. 10-21, 2002.
[2] B.V. Purse, P.S. Mellor, D.J. Rogers, A.R. Samuel, P.P.C. Mertens and M. Baylis, "Climate change and the recent emergence of bluetongue in Europe", Nat. Rev. Microbiol., v. 3, p. 171–181, 2005.
[3] A. Torina, S. Caracappa, P.S. Mellor, M. Baylis and B.V. Purse, "Spatial distribution of bluetongue virus and its *Culicoides* vectors in Sicily", Med. Vet. Entomol., v. 18, p. 81–89, 2004.
[4] S. Gubbins, S. Carpenter, M. Baylis, J.L.N. Wood and P.S. Mellor, "Assessing the risk of bluetongue to UK livestock: uncertainty and sensitivity analyses of a temperature-dependent model for the basic reproduction number", J. R. Soc. Interface, v. 5, p. 363–371, 2008.

Keywords
Mathematical model, vector-borne infection, bluetongue, basic reproduction ratio, R_0.

TIME-LAPSE LIVE CELL IMAGING AND FLOW CYTOMETRY AS DATA SOURCES FOR MODELLING ANTICANCER TREATMENT

P. Ubezio[a], **M. Lupi**[a], **V. Colombo**[a], **F. Falcetta**[a]

[a]Biophysics Unit, Department of Oncology
Istituto di Ricerche Farmacologiche *Mario Negri*
Via La Masa, 19 – 20156 Milano – Italy
paolo.ubezio@marionegri.it

Cancer cell proliferation and response to a treatment comprises a complex sequence of events that results in highly heterogeneous behaviors, some cells dying, some not proliferating at all, and some dividing once or more during the observation period. In anticancer research, cell proliferation has been studied in vitro for a long time by flow cytometry (FC) or time-lapse live cell imaging (TL). FC and TL platforms, considered singularly, produce data that convey a piece of the information, FC focusing on distributions of cells in G1, S, G2M cell cycle phases, TL on lineage trees following cells in subsequent generations, but are unable to render unequivocally the dynamics of the underlying cell cycle progression. Exploiting our previous modeling experience [1-3] we used a cell cycle model including subsequent cell generations to achieve a full reconstruction in silico of the cell cycle progression fully consistent with time courses data obtained with both platforms. The model is based on an age- and phase-structure (G1, S, G2M phases) with drug effects superimposed. The inputs are parameters that describe the cell cycle during unperturbed growth (mean and coefficient of variation of phase durations) and the antiproliferative response induced in each phase by the treatment (probabilities of arrest or killing in each generation). The simulation gives the temporal evolution of cell flow into the different cell cycle phases and subsequent generations from the start of the treatment, from which cell cycle percentages and relative cell numbers in each generation were calculated. These data were fitted to the results of experiments where an ovarian cancer cell line was exposed to X-rays during its exponential growth. Replicate wells were collected at different times for FC analyses or placed onto a time-lapse instrument designed to capture phase contrast images over 72h, every 20 minutes. With this method we disclosed the heterogeneity of the response of cancer cells to X ray exposure, demonstrating that some cells were intercepted by G1, S, G2M checkpoints before dividing (generation 0), others after one or even two mitoses (generation 1 and 2 respectively). Some cells experienced repeated delays in different phases and generations. The fate of the cells was also heterogeneous, even within the same lineage, some descendant remained definitively arrested (particularly in G1 in generation 1 and 2), some refused originating polyploid cells and others died. In conclusion we demonstrated that modeling can be successfully applied to integrate flow cytometry and time-lapse live cell imaging in studies of cell cycle dynamics, reaching a generation-wise picture of G1, S, G2M perturbations induced by anticancer treatment.

References

[1] F. Montalenti, G. Sena, P. Cappella, and P. Ubezio "Simulating cancer-cell kinetics after drug treatment: Application to cisplatin on ovarian carcinoma", *Phys. Rev. E*, 57:5877-5887, 1998.
[2] B. Basse and P. Ubezio "A generalised age and phase structured model of human tumour cell populations both umperturbed and exposed to a range of cancer therapies", *Bull. Math. Biol.* 69:1673-90, 2007.
[3] P. Ubezio, M. Lupi, D. Branduardi, P. Cappella, E. Cavallini, V. Colombo, G. Matera, C. Natoli, D. Tomasoni, M. D'Incalci "Quantitative assessment of the complex dynamics of G1, S and G2M checkpoint activities", *Cancer Res.* 69: 5234-5240, 2009.

EVOLUTION OF BODY CONDITION DEPENDENT DISPERSAL

Margarete Utz*, **Eva Kisdi** and **Mats Gyllenberg**

Department of Mathematics
University of Helsinki
Gustaf Hällströmin katu 2b, 00014 University of Helsinki, Finland
*margarete.utz@helsinki.fi

Dispersers often differ in body condition from non-dispersers. Empirical studies give evidence that body condition can be relevant for an individual's decision whether to stay in its natal habitat patch or to leave and seek for new territory. Differences in body condition between dispersers and non-dispersers are observed among different species. In some of the studied examples, dispersers are heavier, larger, fatter or faster-running than philopatrics, whereas in other examples dispersers are observed to be lighter, smaller, slower, less aggressive or have less reproductive success than their conspecifics. However, there is little comprehension of mechanisms that lead to dispersal of either strong or weak individuals, and, despite the large amount of empirical studies on body condition dependent dispersal, very little emphasis has been given to actually model body condition dependent dispersal mathematically.

We investigate the evolutionarily stable dispersal strategy in a population where body condition ("strength", e.g. body size) varies between and within families. The offspring compete for living sites such that kin competition selects for dispersal. Strong individuals are better competitors in a weighted lottery. The evolving strategy is the probability of dispersal as a function of the individual's and its siblings' body condition, i.e., we ask whether stronger or weaker individuals of a given family will disperse, and which families disperse or retain more offspring.

If body condition does not influence survival during dispersal, then there is no unique ESS. Condition-dependent dispersal strategies with equal competitive weight retained in the natal site form equivalence classes such that strategies within the same class are selectively neutral, and the evolutionarily stable class contains strategies with wildly different relationships between body condition and dispersal probability. This result may explain why there is no consistent pattern between body condition and dispersal found in empirical studies. If body condition also influences the probability of survival during dispersal, then neutrality is removed and individuals with higher survival probability disperse (these may also be the competitively weaker individuals if e.g. smaller body size helps to avoid dispersal risks).

keywords: adaptive dynamics – body condition dependent dispersal – evolution – ESS – function-valued trait – kin competition – spatially structured population

A MULTINOMIAL MODEL OF TUMOR GROWTH TREATED BY RADIOTHERAPY

P. Vallois[a] and **T. Bastogne**[b] and **R. Keinj**[c]

[a] Institut Élie Cartan
Projet BIGS, INRIA
Université Henri Poincaré, UMR CNRS 7502
B.P. 239, F54506 Vandoeuvre les Nancy Cedex
vallois@iecn.u-nancy.fr

[b] Centre de Recherche en Automatique de Nancy
Projet BIGS, INRIA
Université Henri Poincaré, UMR CNRS 7039
B.P. 239, F54506 Vandoeuvre les Nancy Cedex
thierry.bastogne@cran.uhp-nancy.fr

[c]Institut Élie Cartan
Projet BIGS, INRIA
Université Henri Poincaré, UMR CNRS 7502
B.P. 239, F54506 Vandoeuvre les Nancy Cedex
keinj@iecn.u-nancy.fr

A main challenge in radiotherapy is to personalize the treatment by adapting the dose fractionation scheme to the patient. One way is to model the treatment effect on the tumor growth. In this study, we propose a new multinomial model based on a discrete-time Markov chain able to take into account both cell repair and cell damage heterogeneity. The proposed model relies on the 'Hit' theory in radiobiology and assumes that a cancer cell contains m targets which must be all deactivated to produce cell death. The malignant cells population is then split up into m categories to incorporate the variation of cancer cell radio-sensitivity according to their states. This work gives also a new formulation of the tumor control probability (TCP) suited to the perspective of dynamic fractionation schedules in radiotherapy.

Keywords: Markov chains, multinomial model, tumor growth, radiotherapy.

MODELLING THE ECOLOGY AND EVOLUTION OF INFLUENZA VIRUSES

S.P. van Noort[a], **R. guas**[a], **A. Parisi**[b], **A. Nunes**[b] and **M.G.M. Gomes**[a,*]

[a]Instituto Gulbenkian de Ciência
P-2781-901 Oeiras
Portugal
[*]ggomes@igc.gulbenkian.pt

[b]Departamento de Fisica
Universidade de Lisboa
P-1649-003 Lisbon
Portugal

We present a mathematical model for influenza transmission assuming a periodic template for seasonality. The model is parameterized on influenza-like illness data collected by Influenzanet (an internet-based monitoring system) and EISN (European Influenza Surveillance Network). Sequential epidemics, in the Netherlands, Belgium and Portugal, are well adjusted by the model given that two parameters are season-specific, while the remaining are constrant throughout all influenza seasons. Season-specific parameters are the proportion of the population that is susceptible at the beginning of the influenza season, and the probability that infection progresses to influenza-like illness. The results associate early epidemics with large susceptibility pools and low symptom rates. Furthermore, comparison with weather data is consistent with low temperature and absolute humidity as risk factors for influenza symptoms, while comparison with molecular typing of the virus associates large susceptibility pools with large evolutionary jumps.

We proceed with a mathematical model that specifies antigenic properties of the virus and their evolution under immune selection by the host population. We find that the antigenic drift characteristic of influenza A viruses occurs most naturally when hosts are differentiated by the maturity of their immune system.

A microscopic approach for bacterial chemotaxis

N. Vauchelet[a] and **F. James**[b]

[a]UPMC, Univ Paris 06, UMR 7598 LJLL
INRIA Rocquencourt, Project BANG
UPMC, Boite courrier 187, Paris F-75005, France
vauchelet@ann.jussieu.fr

[b]Univ Orléans, UMR CNRS 6628,
BP 6759 - 45067 Orléans Cedex 2
Francois.James@math.cnrs.fr

Keywords. Chemotaxis; Kinetic equations; semi-Lagrangian method; hydrodynamical limit.

Chemotaxis is the process in which bacteria change their state of movement according to a chemical substance in their environment, called chemoattractant in the case of positive chemotaxis. In this work we will focus on the case of *E. Coli* whose movement is composed of two different phases : the 'run' phase and the 'tumble' phase. The 'run' phase consists of a swim in a straight line, while the 'tumble' phase is the reorientation of bacteria. In order to take into account the detailed individual movement of cells, we propose in this work to consider a kinetic model introduce in [2]. This model governs the evolution of the distribution function of cells $f = f(t, x, v)$ and of the chemoattractant concentration $S(t, x)$, where t, x and v denote respectively time, position and velocity :

$$\partial_t f + v \cdot \nabla_x f = \int_V (T[S](v' \to v) f(v') - T[S](v \to v') f(v))\, dv',$$
$$-\Delta S + S = \int_V f(v)\, dv.$$

The turning kernel $T[S](v' \to v)$ corresponds to the rate of bateria changing their velocity from v' to v.

This work is devoted to the case where bacteria are assumed to be able to respond to temporal gradients along their paths. The resulting model has been proposed in [1] where the turning kernel is given by $T[S](v' \to v) = \phi(\partial_t S + v' \cdot \partial_x S)$ for a given nonincreasing positive real-valued function ϕ. Existence and uniqueness result for this model will be presented. Numerical simulations (cf [3]) allows to compare the dynamics of this model to a simplified version, obtained by taking $T[S](v' \to v) = \phi(v' \cdot \partial_x S)$. We will then adimensionalize the system to derive thanks to a hydrodynamical limit a macroscopic system of conservation law.

References

[1] Y. Dolak, C. Schmeiser, *Kinetic models for chemotaxis : Hydrodynamic limits and spatio-temporal mechanisms*, J. Math. Biol. **51**, 595–615 (2005).
[2] H.G. Othmer, S.R. Dunbar, W. Alt, *Models of dispersal in biological systems*, J. Math. Biol. **26**, 263–298 (1988).
[3] N. Vauchelet, *Numerical simulation of a kinetic model for chemotaxis*, to appear in Kin. Relat. Methods.

FUZZY MODELING FOR PREDICTING THE RISK OF RECURRENCE AND PROGRESSION OF SUPERFICIAL BLADDER TUMORS

L. L. Vendite[a], **K. D. Savergnini**[a] and **W.E. Matheus**[b]

[a]Department of Applied Mathematics
University of Campinas
13.083-859, Campinas(SP) Brazil
vendite@ime.unicamp.br, kenia@ime.unicamp.br

[b] Department of Urology
University of Campinas
13.083-859, Campinas(SP) Brazil
wematheus@uol.com.br

Nowadays, the bladder cancer is the fourth most common cancer in adults and the second most frequent urogenital tumor. Predicting recurrence and progression of superficial bladder tumors, with available clinical information to decide the therapy to be used is hard work. In this work, two mathematical models were developed to help specialist on the decision process. The tool used to developed these models was the fuzzy sets theory, by it capacity in dealing with uncertainties inherent in medical concepts. In the first model, Stage, Grade and Size of tumor were also considered input variables and Risk of Recurrence of a superficial bladder tumor as output variable of the first Fuzzy Rule-Based Systems (FRBS). In the second model, in addition to the Stage, Grade and Size of the tumor, also was considered as input variable of a second FRBS Carcinoma in situ and as a output variable, the Risk of Progression of superficial tumors. For each model, simulations were made with data originated from of patients of the Clinics Hospital/UNICAMP and A. C. Camargo Hospital of So Paulo, with the aim to verify the reliability of results generated by the two systems. From a database and possibility found by FRBS, after the possibility-probability transformation, we can generate the real probability of each fuzzy output set.

Keywords: Bladder Tumors, Fuzzy Modelling

References

[1] Castanho, M. J. ; Vendite, L. L. ; Barros, L. C. ; Yamakami,A. Fuzzy Receiver Operating Characteristic Curve: An Option to Evaluate Diagnostic Tests. IEEE Transactions on Information Technology in Biomedicine, v. 11, p. 244-250, 2007..

STOCHASTIC METAPOPULATION MODELING OF INFLUENZA DYNAMIC: CONDITIONS OF SUCCESFULL INVASION OF THE NOVEL A/H1N1 SUBTYPE

E. Vergu[a], **S. Ballesteros**[b], **A. Camacho**[c] and **B. Cazelles**[c,d]

[a]MIA UR 341
INRA
Jouy-en-Josas, France
elisabeta.vergu@jouy.inra.fr

[b]Centro de Matematica e Aplicacoes Fundamentais
Universidade de Lisboa
Lisboa, Portugal
balleste@biologie.ens.fr

[c]UMR 7625
UPMC-ENS-CNRS
Paris, France
camacho@biologie.ens.fr

[d]UMMISCO UMI 209
IRD-UPMC
Bondy, France
cazelles@biologie.ens.fr

During influenza pandemics occurred in the last century, the new strain replaced the previous circulating strains of the same type (except in 1977). The scenario seems to recur for the current pandemic: besides an apparently increasing circulation of type B strains, the great majority of worldwide subtyped A influenza viruses is now pandemic A/H1N1 [1]. To better understand the successful dominance of the new virus, it is very important to provide a general framework capturing the dynamics of coexisting subtypes and to characterize replacement conditions.

Here, we propose a time-continuous metapopulation stochastic model for three co-circulating subtypes (e.g. A/H3N2, A/H1N1 and pandemic A/H1N1), incorporating three key processes of influenza dynamics: antigenic drift [2], seasonal forcing in transmission and temporary full cross-immunity [3]. The model realism is enhanced by considering three age-classes with specific contact rates based on data [4]. The worldwide network underlying the metapopulation structure comprises 52 major cities coupled through real passengers flows [5]. The complete model including all these heterogeneities is of high dimension (more than 10000 state variables) and complexity (thousands simultaneous stochastic transitions).

The impact of key determinants (basic reproductive ratio, R_0, antigenic drift rate, δ, and seasonality, ϵ) was explored through computer-intensive simulations based on parameters consistent with recent estimations. For realistic mean $1/\delta = 2.5$ years for all viruses, resident subtypes can be replaced even by a new virus with a smaller R_0. When $R_0 = 1.5$ for all subtypes and assuming a strong seasonality, the probability of replacement tends to 1 for a rapidly evolving new subtype ($1/\delta$ of the new subtype <1 year) whatever the value of δ for the resident subtype. Other realistic scenarios are identified in the case of a moderate ϵ: the replacement is highly probable for $1/\delta$ of the new subtype between 2 and 4 years.

Besides the interpretation of the replacement dynamics of the new influenza virus in terms of R_0 and antigenic drift rate, our approach highlights two important points. First, the rapid replacement of the previously circulating resident viruses of the same type suggests that maintaining vaccination against these subtypes is not relevant. Second, our model provides a general and appropriate framework to study the spatial variability of initial conditions leading to different local pandemic scenarios all over the world.

References

[1] http://gamapserver.who.int/GlobalAtlas/home.asp.

[2] S. Ballesteros, E. Vergu, and B. Cazelles, "Influenza A Gradual and Epochal Evolution: Insights from Simple Models?", *PLoS ONE*, v. 4(10):e7426, 2009.

[3] N.M. Ferguson, A.P Galvani, and R.M. Bush, "Ecological and immunological determinants of influenza evolution?", *Nature*, v. 422, p. 428-433, 2003.

[4] J. Wallinga, P. Teunis, and M. Kretzschmar, "Using data on social contacts to estimate age-specific transmission parameters for respiratory-spread infectious agents?", *Am J Epidemiol*, v. 164, p. 936-944, 2006.

[5] A. Flahault, E. Vergu, and P-Y. Bolle, "Potential for a global dynamic of influenza A (H1N1)?", *BMC Inf Dis*, v. 9:129, 2009.

Spreading speeds and traveling waves for non-cooperative integro-difference systems

Haiyan Wang[a] and **Carlos Castillo-Chavez**[b]

[a]Division of Mathematical & Natural Sciences
Arizona State University
Phoenix, AZ85069-7100, USA
wangh@asu.edu

[b]Mathematics, Computational and Modeling Sciences Center
Arizona State University
PO Box 871904, Tempe, AZ, 85287, USA
ccchavez@asu.edu

The development of macroscopic descriptions for the joint dynamics and behavior of large heterogeneous ensembles subject to ecological forces like dispersal remains a central challenge for mathematicians and biological scientists alike. Over the past century, specific attention has been directed to the role played by dispersal in shaping plant communities, or on the dynamics of marine open-ocean and intertidal systems, or on biological invasions, or on the spread of disease, to name a few. Mathematicians and theoreticians have set the foundation of a fertile area of research at the interface of ecology, mathematics, population biology and evolutionary biology. Integrodifference systems, the subject of this talk, arise naturally in the study of the spatial dispersal of organisms whose local population dynamics are prescribed by models with discrete generations. Much of the mathematical research has focused on the study of existence of traveling wave solutions and characterizations of the spreading speed particularly, in the context of cooperative systems.

In this talk, we report our recent results in [3]. We shall characterize the spreading speed for a large class of non-cooperative systems, all formulated in terms of integrodifference equations, by the convergence of initial data to wave solutions. In this setting, the spreading speed is characterized as the slowest speed of a family of non constant traveling wave solutions. Our results are applied to a spatial non-cooperative competition system and a set of sufficient conditions for linear determinacy in the two-species competition models is derived. These conditions depend on the dispersals of the invader and out-competed resident and the interactions between the resident and the invader.

References

[1] H. Weinberger, M. Lewis and B. Li, Analysis of linear determinacy for spread in cooperative models, J. Math. Biol. 45(2002) 183-218.
[2] M. Hassell and H. Comins, Discrete time models for two-species competition. Theoretical Population Biology, 9(1976),202-221.
[3]. H. Wang and C. Castillo-Chavez, spreading speeds and traveling waves for non-cooperative integro-difference systems, http://arxiv.org/abs/1003.1600, 2010.

Analysis of an Epidemic-Like Model for the Spread of Religion

Curtis L. Wesley

Louisiana State University in Shreveport
Department of Mathematics, Shreveport, LA 71115, USA

In many ways, the spread of religion behaves similar to the spread of a contagious disease through a population. Therefore, I describe a epidemic-like model for the spread of a religion through a nonconstant population. The model is analyzed and a threshold value for the growth of religion is calculated. Numerical simulations using hypothetical data are also created.

THE ROLE OF SHARED DISEASE IN ECOLOGICAL INVASIONS

A. White[a] , **S. Bell**[b], **J. A. Sherratt**[c], and **M. Boots**[d]

[a]Department of Mathematics and the Maxwell Institute for Mathematical Sciences
Heriot-Watt University
Edinburgh, EH14 4AS
A.R.White@hw.ac.uk

[b]Department of Mathematics and the Maxwell Institute for Mathematical Sciences
Heriot-Watt University
S.Bell@ma.hw.ac.uk

[c]Department of Mathematics and the Maxwell Institute for Mathematical Sciences
Heriot-Watt University
J.A.Sherratt@hw.ac.uk

[d]Department of Animal and Plant Sciences
University of Sheffield
M.Boots@shef.ac.uk

Theory has been developed that examines the role of infectious disease in ecological invasions for particular natural systems. For example, a squirrelpox virus carried by grey squirrels and lethal to red squirrels has been shown to be a key factor in the rapid replacement of red squirrels and spread of greys throughout much of the UK [1]. However, a general understanding of the role that shared disease may play in invasions is lacking. Here, we develop a strategic theoretical framework to determine the role of disease, in addition to competition, in ecological invasions and the expansion of species' spatial range. We investigate the effect of different disease parameters on the replacement time of a native species by an alien invader. The outcome is critically dependent on the relative effects that the disease has on the two species and less dependent on the basic epidemiological characteristics of the interaction. This framework is also used to investigate the effect of disease on the spatial spread of the invader. Our results show an interesting phenomenon where a wave of disease spreads through the landscape ahead of the wave of replacement [2].

Keywords: ecological invasion, disease, spread of infection, travelling waves

References

[1] D.M. Tompkins, A. White and M. Boots, "Ecological replacement of native red squirrels by invasive greys driven by disease", *Ecology Letters*, v. 6, p. 189-196, 2003.

[2] S. S. Bell, A. White, J.A. Sherratt and M. Boots, "Invading with biological weapons: the effect of shared disease on ecological invasions", *Theoretical Ecology*, v. 2, p. 53-66, 2009.

THE EVOLUTION OF HOST-PARASITE RANGE

A. White[a] , **A. Best**[b], **E. Kisdi**[c], **J. Antonovics**[d], **M. A. Brockhurst**[e] and **M. Boots**[f]

[a]Department of Mathematics and the Maxwell Institute for Mathematical Sciences
Heriot-Watt University
Edinburgh, EH14 4AS
A.R.White@hw.ac.uk

[b]Department of Animal and Plant Sciences, University of Sheffield, A.Best@shef.ac.uk

[c]Department of Mathematics and Statistics, University of Helsinki, eva.kisdi@helsinki.fi

[d]Department of Biology, University of Virginia, ja8n@virginia.edu

[e]School of Biological Sciences, University of Liverpool, michael.brockhurst@liverpool.ac.uk

[f]Department of Animal and Plant Sciences, University of Sheffield, M.Boots@shef.ac.uk

Understanding the coevolution of hosts and parasites is one of the key challenges for evolutionary biology. Adaptive dynamics techniques have examined coevolutionary outcomes in classical infectious disease model frameworks in which infection depends on absolute rates of transmission and defence [1]. These models typically predict either that one strain dominates or that there is evolutionary branching, where disruptive selection around a fitness minimum causes the emergence of two distinct strains. This may therefore provide insight into the onset of diversity but does not fully explain the generation and maintenance of the wide range of variation in host and parasite strains observed in natural systems. Here we present a fully coevolutionary host-parasite model using the assumptions of adaptive dynamics, but rather than assuming that transmissibility and defence are absolute we approximate an 'all or nothing' infection process where the success of infection depends upon the relative 'range' of host resistance and parasite infectivity. A parasite that can infect a wide range of host strains will pay a cost in terms of disease transmission compared to parasites that infect a narrower range of hosts. A similar trade-off exists in terms of the range of parasite strains a host can resist and the host reproductive rate. Infection success therefore depends on the specific characteristics of both the parasite and the host. We show that considerable diversity can be generated and maintained due to epidemiological feedbacks, with strains differing in the range of host and parasite types they can respectively infect or resist. The generation of diversity in both hosts and parasites is dependent on the shape of the trade-off relationships for host resistance and parasite transmission, but is more likely in long-lived hosts and chronic disease with long infectious periods. Overall our model shows that significant diversity in infectivity and resistance range can evolve and be maintained from initially monomorphic populations [2].

Keywords: adaptive dynamics, coevolution, host-parasite models, infectious disease

References

[1] A. Best, A. White and M. Boots, "The implications of coevolutionary dynamics to host-parasite interactions", *American Naturalist*, v. 173, p. 779-91, 2009.

[2] A. Best, A. White, E. Kisdi, J. Antonovics, M. A. Brockhurst and M. Boots, "The evolution of host-parasite range", *American Naturalist*, In Press.

ASSESSING THE ROLE OF IMMUNE RESPONSE IN THE DIRECT PROGRESSION AND ENDOGENOUS REACTIVATION OF TUBERCULOSIS

H. Yang

We develop and analyze a mathematical model that describes the interaction between *Mycobacterium tuberculosis* and the immune response. In general, cellular immune response results in the suppression of mycobacterial infection, but does not completely eradicate it. This is the reason why the majority of cases (95%) limits proliferation of the bacilli and produces a long-lasting partial immunity, and 5% of infected individuals develop early progressive disease that occurs within 2-5 years of infection. One of the characteristics of *Mycobacterium* infection is the replication of the bacteria inside of alveolar macrophages. For this reason 5% of asymptomatic individuals have late disease, which is caused by endogenous reactivation as long as several decades after infection. From the model we analyze the interplay between immune response and granuloma formation and further release of bacteria, which characterizes the endogenous reactivation of tuberculosis. In order to control the relapse of tuberculosis by controlling the release of bacteria by tuberculoids, we analyze the effects of drug treatment and gene therapy.

A MINIMAL DISCRETE-TIME MODEL FOR AGE-STAGE-STRUCTURED POPULATIONS OF COMPETING PLANTS

Nikolay N. Zavalishin

Laboratory of Mathematical Ecology,
A.M. Obukhov Institute of Atmospheric Physics RAS,
3, Pyzhevsky Lane, 119017, Moscow, Russia,
nickolos@ifaran.ru

Competition between plant species is considered as a major mechanism of secondary succession following clear-cutting. Dynamics of competing raspberry (Rubus idaeus) and aspen (Populus tremula) populations are described by means of a special kind of matrix population model, which takes into account both the chronological age and the ontogenetic stage of individual plants. Modelling relies upon data sets from clear-cut areas of the Central Forest Reserve (Tver Region, European Russia). Observations on the permanent sample plots for 8 years provided data [1] which were used for design of a nonlinear model of two competing species dynamics during succession. Virtually large numbers of age-stage groups in the both populations are aggregated into a few macro-groups caused by their functional role in the competition processes: for raspberry they are: juveniles of population size $\boldsymbol{I_r}(t)$, reproductives of size $\boldsymbol{R_r}(t)$, and post-reproductives of $\boldsymbol{P_r}(t)$; for aspen: shrubs of population size $\boldsymbol{S_a}(t)$, and trees of $\boldsymbol{T_a}(t)$. Competitive relations between and within these macro-groups are reflected in the discrete-time model equations. The competition effect on a macro-group is mathematically expressed by multiplying the linear terms that provide for the group dynamics in the absence of the competitor with the nonlinear factors that depend on the population of the competitor group as a decreasing function. Self-competition effects are also present within certain macro-groups. All the nonlinear factors are assumed to have exponential form except for the following effects: $\boldsymbol{T_a \to P_r}$ and $\boldsymbol{T_a \to T_a}$; for the latter two a milder rational functional type is used. Estimation of the coefficients is the task of a calibration procedure, which has been developed for the model and observation data. It uses the numbers of marked individuals monitored with regard to transitions specified by the life cycle graphs of each species [1]. The number of calibrating equations is typically greater than the number of unknown coefficients to be calibrated, hence the solution becomes a matter of optimization on the least-squares basis.

The aggregated, low-dimensional model demonstrates biologically interpretable outcomes of competition expressed in both interior and boundary steady states, the latter indicating a possibility of competitive exclusion. The competition outcome in each particular situation is determined by the sizes of the juvenile raspberry group and of the aspen shrubs. Nevertheless, variation of calibrated coefficients also affects the outcome, changing markedly the geometry of stability domains for the interior and boundary steady states. In order to study an approximate form of stability domains in the phase space, two system parameters are expressed through the initial sizes of two raspberry and aspen macro-groups, and the problem is solved by methods of bifurcation theory for discrete-time systems.

References

[1] N.G. Ulanova, N.N. Zavalishin, and D.O. Logofet, “Competition between and within aspen (*Populus tremula*) and raspberry (*Rubus idaeus*) after clear-cutting: matrix models of structured populations dynamics”, *Forest Science and Technology*, Vol. 3, No. 1, p. 68-77, 2007.

MODELLING SPATIAL AND SPATIALLY AVERAGED EXPLOITED FOOD CHAINS OF A RESOURCE-CONSUMER TYPE

Nikolay N. Zavalishin[a]

Laboratory of mathematical ecology,
A.M. Obukhov Institute of atmospheric physics RAS,
3, Pyzhevsky Lane, 119017, Moscow, Russia,
nickolos@ifaran.ru

Dynamic models of food chains demonstrate both regular – equilibria and periodic orbits - and irregular structures under variation of their parameters. In fact, there are two methods for modelling of food chain dynamics: the most popular approach is based on equations of interacting populations connected by predator-prey relations where the first species is usually self-regulated, while another - less widespread techniques - considers a chain in units of the entering resource with its explicit description [1]. For the last approach a collection of interesting dynamic features on bifurcations of equilibria under the nutrient inflow increase and strange attractor formation was established [2]. In this work, the method is used to study dynamics of a spatially distributed exploited partially closed trophic chain with explicit nutrient influx in comparison to its spatially aggregated analogue. Problems of travelling wave solutions existence and formation of spatial patterns under harvesting efforts and varying of input nutrient or energy fluxes are of special interest. Perturbations of two parameters – external resource input flux and harvesting mortality coefficient for the last species in the chain of even ($n = 2$) and odd ($n = 3$) length for the corresponding local wave system initiate different types of travelling waves like a regular wave and wave front. Increasing the resource flux can lead to Hopf bifurcation of non-trivial equilibrium in a wave system of ODE corresponding to appearance of wave trains – periodic wave solutions in the initial PDE model. Rising a harvesting mortality coefficient can result in a wave front leading the last species in the chain to rapid decrease to zero and the chain itself to be shortened. For spatially averaged trophic chains of the same structure and functional responses stability boundaries for equilibria in the same parameter space can be obtained. Another subject of interest in spatially distributed chains of a resource-consumer type is a relative role of such spatial effects as diffusion, migration and chemotaxis in pattern formation and instability of uniform steady states. Numerical analysis with elements of analytical instability conditions allow one to estimate approximately domains of existing dissipative and irregular structures in particular case studies. They include exploited spatially non-uniform chains of a “resource-consumer” type two- and three-level trophic structures of Okhotsk and Bering Seas at the Russian Far East provided in one of them the omnivory effect.

References

1. Svirezhev Y.M., Logofet D.O., Stability of biological communities. Moscow, Mir Publishers, 1983, 320 pp.

2. Jorgensen S.-E., Svirezhev Y.M., Towards a thermodynamic theory for ecological systems, Elsevier, Amsterdam, 2004, 366 pp.

A spatio-temporal model to describe the spread of *Salmonella* within a laying flock

P. Zongo[a], **A. Viet**[b], **P. Magal**[c] and **C. Beaumont**[d]

[a]INRA UR 083 Recherche Avicoles
37380 Nouzilly, France
Pascal.Zongo@tours.inra.fr

[b]INRA ONIRIS UMR 1300 Bio-agression Epidémiologie et Analyse de Risques
44307 Nantes, France
aviet@info.unicaen.fr

[c]UMR CNRS 5251 IMB-INRIA Sud-Ouest Anubis
Université Victor Segalen Bordeaux 2, 33000 Bordeaux, France
Pierre.Magal@math.u-bordeaux1.fr

[d]INRA UR 083 Recherche Avicoles
37380 Nouzilly, France
Catherine.Beaumont@tours.inra.fr

Salmonella is one of the major sources of toxi-infection in humans, particularly in France, most often as a consequence of consumption of contaminated eggs or poultry products. The main reason for this association is the presence in hens flocks of silent carriers, i.e. animals harbouring S. enteritidis without expressing any visible symptoms. Many prophylactic means have been developed to reduce the prevalence of *Salmonella* carriers-state. While none allows a total reduction of the risk, synergy could result in a drastic reduction of it. Evaluating the risk ex-ante by modelling approach would be very useful to estimate such gain in food safety. Previously, deterministic models for Salmonella transmission in a hen house were derived in the absence of individual variations in response to infection. Here, we propose an individual-based model to describe the spatio-temporal spread of *Salmonella* within a laying flock taking into account this variability. For each animal, the model includes the individual bacterial load and ability to reduce it thanks to the immune response, influencing the diffusion of the contaminant within the hen house. It allows studying the effects, on the spatio-temporal repartition of the bacteria, of mean level and variability of different resistance traits, such as the rate of excretion (and in particular the effect of super excretors) or the level of immune response.

Keyword: *Salmonella*; Individual-based model; within host; bacterial transmission; individual heterogeneity

Author Index